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Surgical treatment of an oesophageal achalasia in a small breed dog

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Abstract: A 6-year-old, male Yorkshire Terrier dog presented with persistent regurgitation and severe weight loss. Based on the clinical signs, physical and other various diagnostic examinations, including fluoroscopy, were performed. Myasthenia gravis was ruled out through a serum acetylcholine receptor antibody titre measurement and a negative response to neostigmine bromide (0.02 mg/kg) within 4 hours of injection. The dog was diagnosed with idiopathic oesophageal achalasia. As a treatment, a modified Heller's oesophagomyotomy was performed, and the dog recovered well with no signs of recurrence for 18 months.

Keywords: canine oesophageal achalasia; oesophagomyotomy; modified Heller's myotomy; myenteric plexus

Oesophageal achalasia is a rare motility disorder of the oesophagus with unknown aetiology and the occurrence has been reported as a hereditary or an acquired neuromuscular disease in dogs (Clifford 1974). It can be characterised by a lack of normal oesophageal peristalsis and an uncoordinated opening of the lower oesophageal sphincter (LES), leading to impaired oesophageal emptying and gradual oesophageal dilation (Birgisson and Richter 1997; Woltman et al. 2005). Subsequent dysphagia, accompanied by varying degrees of regurgitation, severe weight loss, and pain, are the identifying features of achalasia (Woltman et al. 2005). Although it has been reported that idiopathic achalasia is associated with a loss of inhibitory innervation of the oesophageal myenteric plexus, the initiating cause is still unidentified so far (Park and Vaezi 2005). In dogs, it can be a congenital condition in the young or an acquired condition in the old with no sex predisposition (Lawther 1970; Clifford 1974). Here, we report a case of a dog with oesophageal achalasia that was diagnosed by means of various clinical examinations and treated with an oesophagomyotomy.

Case description

A 6-year-old, castrated, male Yorkshire Terrier dog presented with persistent regurgitation. The dog primarily regurgitated foods, occasionally with white foamy vomitus, over 3 months, and this was accompanied by severe weight loss during this period. A physical examination revealed a wasted dog with a 1 out of 5 body condition score. The dog was bright, alert, and responsive with a normal capillary refill time, body temperature, heart rate, and respiratory rate. A blood analysis with a complete blood count and serum chemistry profile showed leukocytosis and mild elevations in the glucose and blood urea nitrogen (BUN) concentrations, and alanine amino transferase (ALT) and gamma

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glutamyl transferase (GGT) activities. On a plain thoracic radiograph, a moderate to severe abnormally air-distended area was found at the thoracic oesophagus level (Figure 1). An oesophagography was performed to evaluate the megaoesophagus and oesophageal motility. The administration of liquid barium sulfate followed by a fluoroscopy observation revealed a failure of the lower oesophageal sphincter (LES) to open properly. A neostigmine injection (0.02 mg/kg, *i.v.* JE-IL PHARM, Seoul, Republic of Korea) test was performed for a differential diagnosis of myasthenia gravis, but there was no distinct difference in the size of the lower oesophagus before or after the injection (Figure 2). Furthermore, to rule out myasthenia gravis, an immunology test was performed through a serum acetylcholine receptor antibody titre measurement, which showed a normal value (0.03 nmol/l; canine reference value < 0.6 nmol/l, IDEXX Reference Laboratories). Based on the two diagnostic tests, myasthenia gravis was ruled out as the cause of the lower oesophageal dilation. To investigate a hypothyroidism associated-peripheral neuropathy, the serum total thyroxine (TT4), the free T4, and the canine thyroid stimulating hormone (cTSH) were measured after the TSH stimulation test

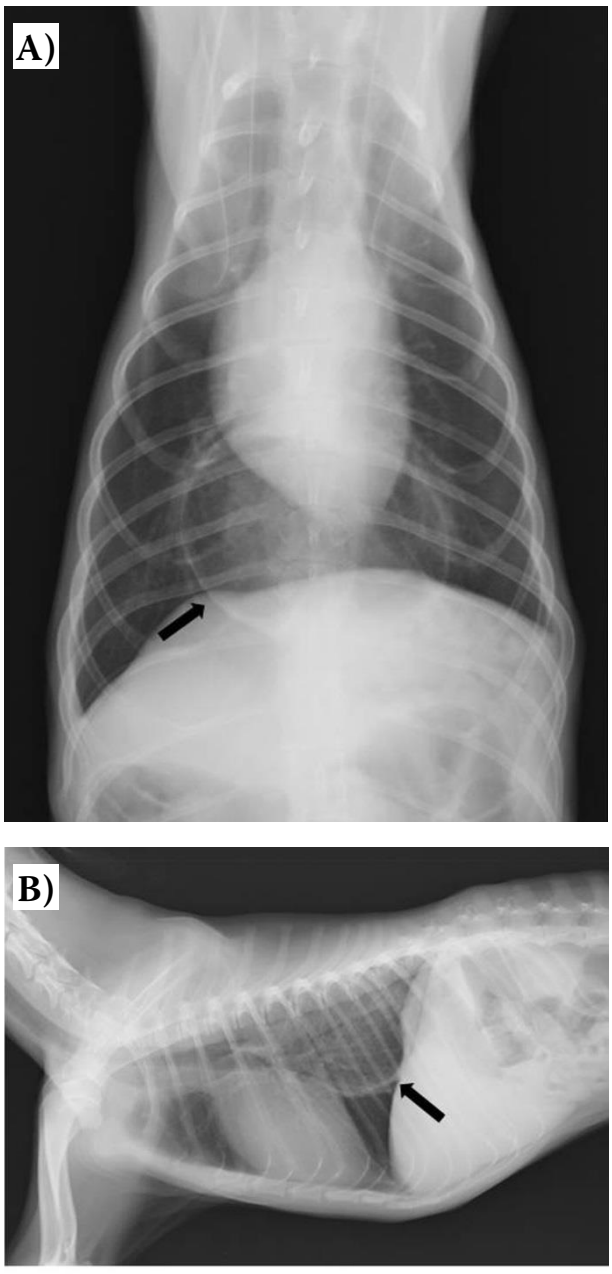

Figure 1. (**A**) The ventrodorsal and (**B**) right lateral positioned plain thoracic radiographs show abnormal gas distended in the area at the level of the lower oesophagus (black arrows)

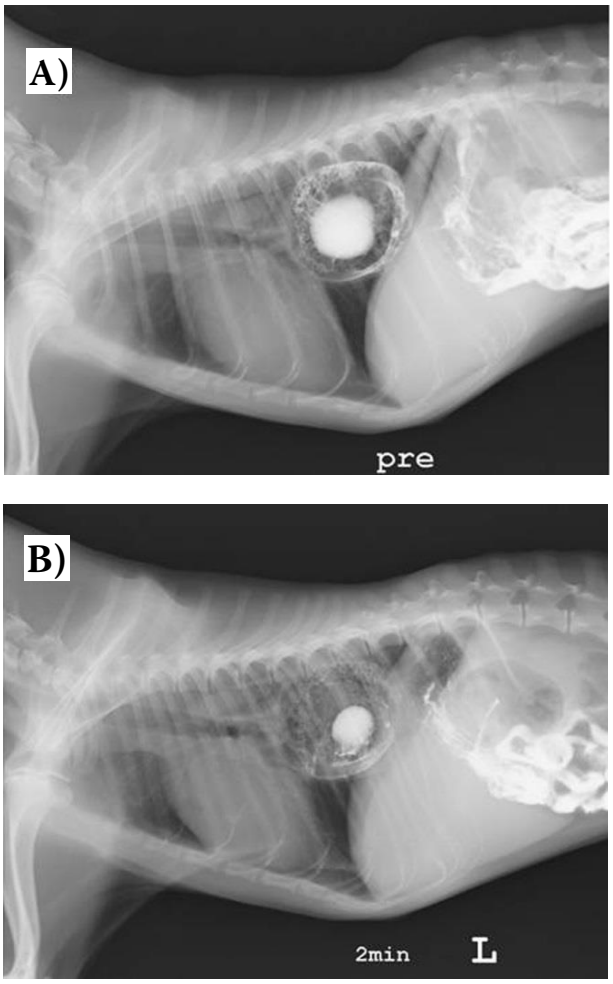

Figure 2. A neostigmine injection (0.02 mg/kg, *i.v.*) test differential diagnosis for myasthenia gravis. No change was observed in the size of the lower oesophagus before (**A**) or after (**B**) the injection

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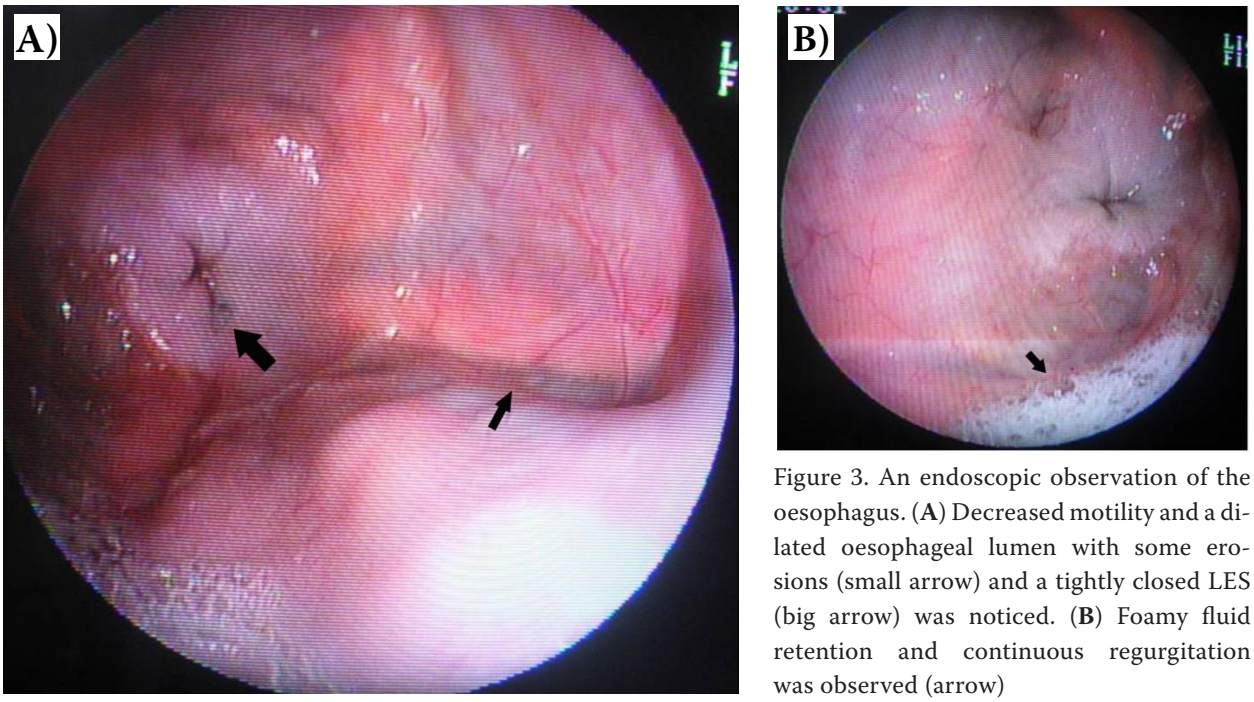

Figure 3. An endoscopic observation of the oesophagus. (**A**) Decreased motility and a dilated oesophageal lumen with some erosions (small arrow) and a tightly closed LES (big arrow) was noticed. (**B**) Foamy fluid retention and continuous regurgitation was observed (arrow)

(IDEXX Reference Laboratories). Hypothyroidism was ruled out as the results showed a 9 (reference value 10–40 µg/l) in the TT4, an 8 (reference value 6–37 ng/l) in the free T4, and a 0.10 (reference value 0.05–0.42 ng/ml) in the cTSH. Through an endoscopic observation, the oesophageal body showed poor motility with a dilated lumen and a foamy fluid retention, some erosion in places, and that the LES was tightly closed (Figure 3). Accounting for all examination results and clinical signs, we tentatively diagnosed the dog with oesophageal achalasia. As a palliative treatment to relieve the regurgitation and promote oesophageal emptying, a distal oesophagomyotomy with a modified Heller's procedure was selected as a surgical option. The surgical procedure was performed as previously described (Boria et al. 2003). Under general anaesthesia, the dog was positioned in the

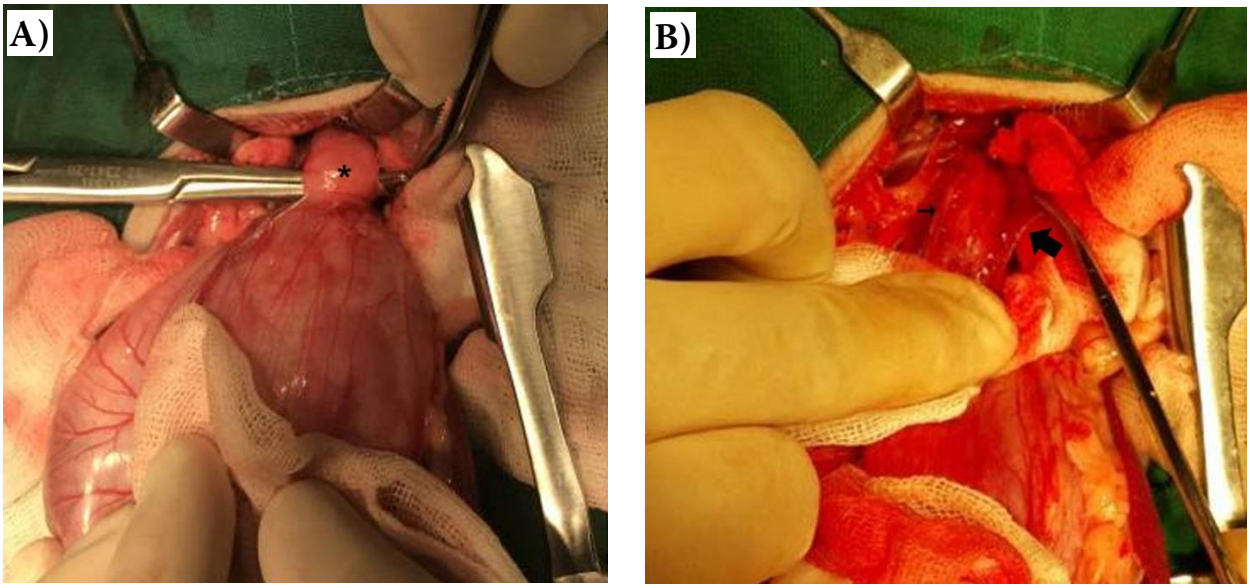

Figure 4. A modified Heller's myotomy procedure. (**A**) Identification of the gastro-oesophageal junction (asterisk). (**B**) After the myotomy, the incised serosa layer is grasped with forceps (big arrow) and the submucosal layer underneath is bulged (small arrow)

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dorsal recumbency, and the surgical area was prepared properly. An abdominal midline skin incision was made from the xiphoid process to the umbilicus. After opening the peritoneum, the stomach was gently retracted to approach to the gastro-oesophageal junction, where the LES is located. Subsequently, the distal part of the oesophagus and the cardia of the stomach were identified, and a longitudinal incision over the serosa and the muscular layer was made on the ventral part of the gastro-oesophageal junction, extending about 1 cm from the distal oesophagus to 1 cm from the proximal stomach (Figure 4). The incision at the gastro-oesophageal junction was left unsutured, an 8-Fr balloon catheter was adopted as a gastrostomy tube, and the abdomen was closed in a routine manner. The postsurgical fluoroscopies, performed at 1, 2, and 6 weeks after the surgery to evaluate the LES function, showed improvement in the passage of the contrast medium through the LES over time (Figure 5). Furthermore, a gradual increase in the body weight was observed after the surgery (followed up for six months), and the clinical signs improved as well.

DISCUSSION AND CONCLUSION

In 1929, Hurst and Rake named the condition of achalasia, which means the "failure to relax," as the disease is due to the failure of the oesophageal sphincter to relax (Birgisson and Richter 1997). Achalasia of the oesophagus is characterised by a lack of peristalsis of the distal oesophagus and an abnormal LES relaxation (Kempf et al. 2014).

Acquired oesophageal dilation may result from many disorders, including myasthenia gravis and polymyopathy, which cause a neuromuscular malfunction, and an endocrine disease such as hypothyroidism as well (Kook 2013). In this case report, as previously described, several differential diagnostic exams were performed to identify the cause of the dilated oesophagus and LES dysfunction (Boria et al. 2003). A test for myasthenia gravis should be performed in dogs with decreased oesophageal motility, because myasthenia gravis can imitate idiopathic megaoesophagus (Kook 2013). A recent case report by Kempf et al. demonstrated a myasthenic dog with moderate oesophageal dilation, showed a dramatic positive response to

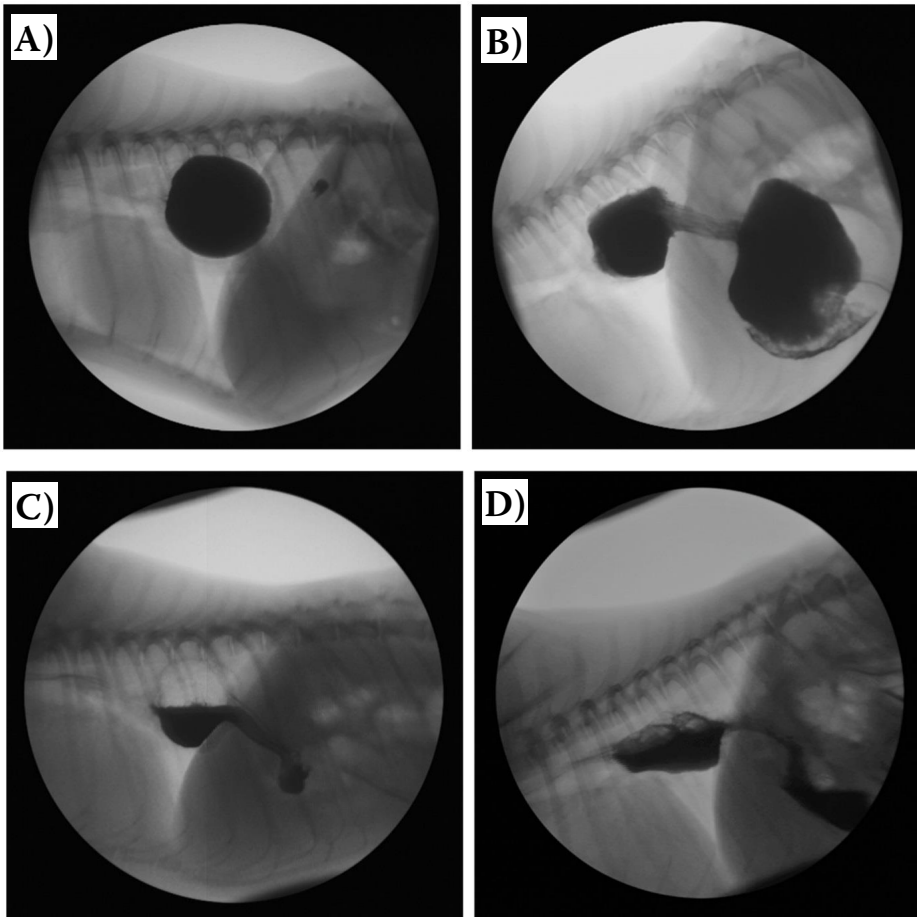

Figure 5. Fluoroscopic findings. Before the oesophagomyotomy (**A**), the contrast medium could hardly pass through the LES, but the patient showed gradual improvement after the surgery over time: (**B**) 1 week postoperatively, (**C**) 2 weeks postoperatively, (**D**) 6 weeks postoperatively. Each photo was captured from fluoroscopic videos that were approximately 1–3 min long

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a cholinesterase inhibitor (Kempf et al. 2014). Hypothyroidism is somewhat controversial, yet a case of reversible megaoesophagus associated with primary hypothyroidism in a dog has been reported (Fracassi and Tamborini 2011). Among the possibilities, myasthenia gravis was ruled out by a neostigmine stimulation test and a serum acetylcholine receptor antibody titre measurement, hypothyroidism was ruled out by a thyroid hormonal test, and oesophageal diverticulum was ruled out by an oesophageal endoscopic observation.

This uncommon disease of unknown aetiology is thought to be related to the loss of the inhibitory ganglion cells in the myenteric (Auerbach's) plexus, and characterised by the lack of distal oesophageal peristalsis and insufficient LES relaxation (Earlam et al. 1967, Kempf et al. 2014). The LES of a canine oesophagus is composed of an outer longitudinal striated muscle layer and an inner smooth muscle layer (Boria et al. 2003). The striated muscle of the proximal oesophagus is innervated directly by the somatic efferent cholinergic fibres of the vagus nerve. On the other hand, the smooth muscle of the distal oesophagus is innervated by the preganglionic vagus nerve fibres, which first innervate the myenteric plexus. Subsequently, the oesophageal wall and the LES are innervated by the postganglionic neurons, consisting of excitatory and inhibitory neurons that are associated with the oesophageal and LES contraction and relaxation. Loss of the inhibitory innervation in the myenteric plexus results in a loss of oesophageal peristalsis as well as the failure of the LES to relax (Park and Vaezi 2005). Because the canine oesophagus does not consist of a specialised large mass of muscle, the LES functions as a physiologic rather than an anatomic sphincter, and the normal function of the LES is to relax and open during deglutition (Boria et al. 2003).

In a case report by Earlam et al. (1967), the authors presented a case of a 3-month-old German Shepherd with congenital oesophageal achalasia, who suffered from a lower oesophageal obstruction and underwent surgery with a modified Heller's method. Unfortunately, the puppy died on the third day after the surgery due to a sudden respiratory infection, secondary to aspiration pneumonia. In a histological examination, the authors demonstrated that there were no ganglion cells detected in the myenteric plexus between the muscle layers of the oesophagus (Earlam et al. 1967). Another experimental study also described how a severe destructive change in the myenteric plexus could be causative of oesophageal achalasia. Okamoto et al. (1967) experimentally produced selective damage to the ganglion cells of the oesophageal myenteric plexus in dogs by perfusion of the lower oesophagus via the left gastric artery. In their experiment, the authors used Tyrode's solution containing 0.002% of mercuric chloride, which is well known to have a specific affinity for nerve cells. Through this experimental research, the authors demonstrated that the destruction of the myenteric plexus seemed to induce a gradual stenosis at the lower oesophagus and a failure of the LES to open in a coordinated fashion (Okamoto et al. 1967).

As of now, the treatment options for oesophageal achalasia are focused on the palliation of the symptoms because the neuromuscular defect is not curable in terms of recovering the underlying pathology (Woltman et al. 2005). In human medicine, the use of drugs to treat oesophageal achalasia can be attractive because it is a non-invasive therapy, but there are some limitations, including an unpredictable efficacy, which is secondary to poor oesophageal emptying and other side effects. Botulinum toxin (Botox) can be less invasively injected into the LES by a flexible endoscope without immediate complications. According to the literature (Vaezi and Richter 1998), however, although Botox seemed to be effective initially in 60–85% of patients, 50% were recurrent within six months after injection. Furthermore, the efficacy decreases with repeated injections and can cause intense inflammation of the gastro-oesophageal junction (Woltman et al. 2005). Another less invasive treatment option for oesophageal achalasia is pneumatic dilation, which is the forceful dilation of the LES with graded, polyethylene balloons. After evaluating the LES diameter and perforation by an oesophagram with a water-soluble contrast medium, the balloon is kept inflated for 1–3 min and then deflated (Woltman et al. 2005). With the pneumatic dilation, approximately 70% of patients gain considerable ease of the dysphagia after one year (Csendes et al. 1989), but the risk of incidental perforation during the dilation attempt is as high as 6% (Reynolds and Parkman 1989).

In this case report, a modified Heller's myotomy was performed as previously reported (Boria et al. 2003), and the dog showed a favourable prognosis. A surgical therapy for oesophageal achalasia

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was first described by Ernest Heller in 1914, originally with double myotomies along the gastro-oesophageal junction, with one in the anterior and another in the posterior. These days, a modified single myotomy is usually performed in the anterior only with excellent results in up to 95% of the patients (Woltman et al. 2005). In 1967, Ellis et al. compared three types of myotomies in an experimental study using dogs. They evaluated a classic Heller myotomy, which consisted of a double extramucosal myotomy extending for 3 cm on either side of the gastro-oesophageal junction; a long myotomy, which consisted of a single anterior extramucosal myotomy extending for 3 cm on either side of the gastro-oesophageal junction; and a short myotomy, which consisted of a single anterior extramucosal oesophagomyotomy of 3 cm and which extended only a few millimetres onto the stomach. The authors demonstrated that a short myotomy limited to the oesophagus is recommended because longer and more drastic myotomies have no benefit for the sphincteric pressure decreases and may lead to gastro-oesophageal reflux (Ellis et al. 1967). In previous reports in human medicine, the development of minimally invasive techniques allowed a modified Heller's myotomy to be performed by either a thoracoscopic or a laparoscopic procedure (Shimi et al. 1991; Pellegrini et al. 1992).

To the author's knowledge, a lower oesophageal achalasia is extremely rare in the veterinary literature, while an upper oesophageal sphincter achalasia (cricopharyngeal achalasia) has been well described as a congenital disorder in dogs. Although a manometry evaluation of the LES is considered to be the gold standard in confirming the diagnosis (Woltman et al. 2005), a fluoroscopic evaluation with a contrast medium can also be a crucial tool for the diagnosis of oesophageal achalasia. In conclusion, a surgical therapy with a modified Heller's myotomy is a simple and effective treatment option to relieve the regurgitation associated with oesophageal achalasia.

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