Aerodigestive Disease in Dogs

Megan Grobman, DVM, MS, PhD

KEYWORDS

• Aspiration • Dysphagia • Motility • Pneumonia • Reflux • Megaesophagus • Respiratory

KEY POINTS

- Aerodigestive disorders span a wide range of important clinical syndromes affecting the upper and lower airways, bronchioles, and pulmonary parenchyma (ie, alveoli and interstitium).
- Aerodigestive diseases frequently reflect defects in swallowing and/or airway protection.
- Aerodigestive diseases can occur in the absence of digestive signs and in the face of normal radiographs.
- The role of bacterial infection in cases of aspiration pneumonia is unclear.
- Videofluoroscopic swallow studies represent the criterion standard for diagnosis of dysphagia in veterinary medicine.

Video content accompanies this article at http://www.vetsmall.theclinics.com.

INTRODUCTION

Although aspiration pneumonia is the most well-recognized aerodigestive disorder (AeroD) in veterinary clinical practice, AeroDs span a range of common and clinically important conditions (**Table 1**). AeroDs reflect failures in airway protection, abnormal swallowing, or a combination of these that result in, or contribute to, respiratory disease. In people, AeroDs have been implicated in the pathogenesis and progression of several acute and chronic respiratory diseases. Reflux, for example, has been implicated in chronic cough, asthma, pulmonary fibrosis, and chronic obstructive pulmonary disease.[1,2] Importantly, treatment of reflux was found to reduce the frequency of disease exacerbations and slow the rate of decline in lung function.[3]

A similar association has been documented in dogs. A study evaluating dogs presenting exclusively for cough, in the absence of clinical signs of digestive disease, found swallowing abnormalities in 80.6%, including 9/11 dogs with normal thoracic radiographs.[4] Furthermore, there is a demonstrated correlation between gastrointestinal (GI) signs and the severity of respiratory disease in brachycephalic dogs, with 88% of

Auburn University, Department of Clinical Sciences, 1220 Wire Road, Auburn, AL 36849, USA
E-mail address: Meg0098@auburn.edu

Vet Clin Small Anim 51 (2021) 17–32
https://doi.org/10.1016/j.cvsm.2020.09.003
vetsmall.theclinics.com

Table 1
Aerodigestive syndromes reported in the veterinary literature by anatomic location

Condition	
Upper airway	Laryngitis,[35,74] laryngeal paralysis/dysfunction,[19,33,35,74] larygopharyngeal reflux,[4] nasopharyngeal reflux,[4] otitis,[21] upper airway obstruction,[23] rhinitis,[4] sleep disordered breathing (apnea)[23]
Bronchi, bronchioles, and pulmonary parenchyma	Acute respiratory distress syndrome,[35] aspiration pneumonia/pneumonitis,[35] bronchiectasis,[65] bronchomalacia,[4] diffuse aspiration bronchiolitis,[35] exogenous lipid pneumonia,[75] interstitial lung disease,[35,43] large airway obstruction[76]
Oral preparatory defects	Cranial nerve defects,[53] periodontal disease,[4] myopathy/neuropathy[53,77]
Pharyngeal swallow defects	Cricopharyngeal achalasia,[56] cricopharyngeal dyssynchrony,[56] myopathy/neuropathy,[53] pharyngeal hypomotility,[4] pharyngeal spasticity,[4] pharyngeal mucocele,[78] reflux (laryngopharyngeal)[4]
Esophageal swallow defects	Esophageal hypomotility,[4] LES-AS,[4] megaesophagus,[4] myopathy/neuropathy[53] hiatal hernia,[4,79] reflux (gastroesophageal)[4]
Gastric	Vomiting (acute or chronic)[53]

Otitis is included the upper airway disorders due to the connection of the Eustachian tube to the nasopharynx. This list is not exhaustive but is intended to reflect the more common diseases encountered in clinical veterinary practice.
Data from Refs. [4,19,21,23,33,35,43,53,56,65,74,76–79]

brachycephalic dogs treated medically for GI disease demonstrating clear and sustained improvement after brachycephalic airway surgery.[5] Additionally, investigators found a decreased complication rate and improved prognosis in brachycephalic dogs treated for reflux prior to brachycephalic airway surgery.[5] This suggests that a subpopulation dogs with respiratory signs have AeroDs and, like humans, may respond to treatment targeting the digestive tract. Unfortunately, AeroDs may present with occult digestive signs, representing a significant clinical challenge for the practitioner.

PHYSIOLOGY OF SWALLOWING

Swallowing is a complex act involving several muscles of the upper respiratory and digestive tracts and can be considered both a feeding and an airway protective behavior. The cooperation between swallowing and breathing is not limited to the removal of oral and respiratory secretions and prevention of aspiration during swallowing. Safe swallowing also requires centrally mediated coordination to ensure breathing and swallowing do not occur at the same time, that there is time to clear secretions after coughing to prevent reaspiration, and to reset the respiratory cycle to ensure a fairly consistent respiratory rate and rhythm.[6]

The act of swallowing can be separated into voluntary and involuntary phases. The voluntary phase involves the prehension and mastication of food and propulsion to the oropharynx. This phase in entirely voluntary and absent in reflexive swallows, such as may be seen when swallowing accumulated oral secretions[7–10]

The presence of a bolus in the rostral oropharynx initiates the pharyngeal phase of the swallow. This and all subsequent phases are involuntary. The pharyngeal phase is mediated by sensory afferents of cranial nerves IX and X and involves passage of the bolus through the upper esophageal sphincter into the esophagus.[6,11] This phase of swallowing also is characterized by the initiation of airway protective behaviors, including the blockage of the nasopharynx and larynx, which are absent during the oral preparatory and esophageal phases.[12,13]

Passage of the bolus into the esophagus marks the beginning of the esophageal phase of swallowing. Contraction of the upper esophageal sphincter and accompanying peristaltic contraction is the primary wave. Luminal distention triggers progressive contractions (secondary waves). The esophageal phase ends with the opening of the lower esophageal sphincter (LES) to allow the passage of food into the stomach and closure to prevent reflux.[14]

DYSPHAGIA, REGURGITATION, AND VOMITING

The term, *dysphagia*, can be used to describe a defect in any of the phases of swallowing. Dysphagia may be classified based on location/phase (oral preparatory, pharyngeal, or esophageal) and/or mechanism (mechanical vs functional). Dysphagia may be secondary to functional defects in swallowing (eg, myasthenia gravis, pharyngeal hypomotility, and cricopharyngeal achalasia) or secondary to structural abnormalities (eg, severe dental disease, trauma, foreign bodies, strictures, and neoplasia). Key clinical features associated with dysphagia include difficulty with prehension, gagging, repetitive swallowing while eating, and regurgitation. Additional clinical features of aerodigestive disease are listed in **Table 2**. Although these features may help a practitioner localize the problem to a specific location/swallowing phase, treatment depends on the specific underlying etiology. Although some conditions can be managed medically (esophagitis and myasthenia gravis), disorders of motility (cricopharyngeal achalasia and LES–achalasia-line syndrome [AS]) require targeted intervention. Incorrect localization may lead to significant worsening of clinical signs, making correct identification critically important.[14–16]

The clinical feature most commonly associated with defects in the esophageal phase of swallowing is regurgitation. Regurgitation refers to the retrograde passive expulsion of esophageal contents. Importantly, in regurgitation the airway protective behaviors that occur during vomiting (ie, laryngeal adduction and soft palate elevation) are absent. Obstructive disorders causing regurgitation include but are not limited to esophageal foreign bodies, vascular ring anomalies, strictures, and esophageal/paraesophageal tumors. Functional disorders include defects of the LES (eg, LES-AS), muscle (eg, esophageal hypomotility), inflammatory disease (eg, esophagitis), neuromuscular dysfunction (eg, botulism, myasthenia gravis, and polyneuropathy/polymyopathy), and toxins (eg, lead, thallium, and organophosphates).[15–17]

In contrast to regurgitation, vomiting is a centrally mediated process, resulting in the active expulsion of GI contents from the stomach and proximal duodenum. Vomiting also involves coordinated efforts to protect the airways. Despite this, vomiting, like regurgitation, is considered a major risk factor for respiratory disease by overwhelming these protective mechanisms.[15–18]

REFLUX AND REFLUX DISEASE

A less well-known, but important, link between respiratory and digestive disease is gastroesophageal reflux (GER). GER refers to the back flow of stomach contents into the esophagus. An extraesophageal manifestation of GER is extraesophageal

Table 2
Aerodigestive signs and related clinical mechanisms in dogs

Aerodigestive Signs	Related Clinical Mechanism
Cough	Airway protective mechanism, nonspecific marker of respiratory disease. The severity of cough does not reflect disease severity because chronic aspiration may diminish the cough reflex.[80]
Dysphonia	Laryngeal dysfunction
Gagging while eating	Pharyngeal swallow dysfunction
Increased effort while swallowing	Pharyngeal swallow dysfunction
Lip licking	Reflux
Neck extension	Esophageal pain, reflux, pharyngeal swallow dysfunction (when observed while eating)
Night restlessness, flank biting, prayer position	Epigastric pain frequently encountered with reflux and sliding hiatal hernia
Regurgitation	Reflux, esophageal dysphagia, esophagitis
Repetitive dry swallowing (not while eating)	Reflux
Repetitive swallowing attempts per bolus (while eating)	Pharyngeal swallow dysfunction
Respiratory distress	Acute upper airway obstruction, severe aspiration event
Reverse sneezing	Nasopharyngeal reflux, pharyngeal collapse, pharyngitis
Stridor	Laryngeal dysfunction
Stertor	Nasal or pharyngeal disease
Throat clear	Airway protective mechanism suggestive of poor pharyngeal clearance, nasopharyngeal reflux, postnasal drip

reflux (EER). The broader category of EER includes laryngopharyngeal reflux, where the refluxate contacts the larynx, and pharynx and nasopharyngeal reflux, where the refluxate extends into the nasopharynx. Reflux often is an occult process in veterinary species, but it has been implicated in laryngeal dysfunction, increased mortality after brachycephalic airway surgery, otitis media via the eustachian tube, and chronic rhinitis.[5,19–22] In pediatric animals, reflux can cause apnea, bradycardia, laryngeal closure, and pediatric death.[23] GER occurs commonly in healthy, asymptomatic humans and has been documented in up to 41% of asymptomatic dogs.[24] Pathologic and physiologic reflux differ, however, in terms of volume, timing, and location within the esophagus. Discrimination is possible by several imaging modalities, including videofluoroscopic swallow studies (VFSSs). Reflux often remains occult until patients develop complications: esophagitis, laryngeal dysfunction, regurgitation, and a wide spectrum of respiratory diseases.

The combinations of GER, clinical signs, and gross pathology (dependent on site) are termed, *GER disease* (*GERD), EER disease* (*EERD*), *laryngopharyngeal reflux* disease, and *nasopharyngeal reflux disease*. Clinical signs are dependent on frequency, volume, and duration of contact between the refluxate and the esophagus, larynx, and pharynx. Causes of reflux include spontaneous transient relaxations of the LES (most common), diminished basal LES pressure, straining (coughing, vomiting, or increased intra-abdominal pressure), and hiatal hernia.[1,2,5,19,20,25] Pathology associated with

GERD can occur by direct contact of low pH refluxate and digestive enzymes, leading to tissue damage and stimulation of regional nerve terminals, and by macroaspiration/microaspiration. The pathophysiology of tissue damage secondary to reflux and aspiration is discussed in more detail later. VFSS clips depicting EER and pathologic GER with a sliding hiatal hernia are provided in the supplemental materials.

AERODIGESTIVE DISORDERS

Because AeroDs can reflect dysfunction in any phase of swallowing as well as failures in airway protection, the number of potential conditions is substantial. Failures in airway protection include both mechanical and functional defects (eg, laryngeal mass and laryngeal paralysis, respectively) as well as clinical scenarios where the normal airway protective mechanisms are overwhelmed (eg, vomiting and regurgitation).

Additional complications are multifocal disease and comorbid conditions contributing to clinical signs. A study using VFSSs to evaluate patients for occult AeroDs identified 72% of dogs with swallowing abnormalities had defects in more than one location.[4] Additionally 55% of those with swallow abnormalities had respiratory disease contributing to their clinical signs. This relationship is well established in people where the act of coughing induces reflux events in patients with known reflux induced chronic cough.[26] Similarly, in dogs with reflux and laryngeal paralysis, the degree of negative pressure associated with upper airway obstruction is sufficient to induce additional reflux events.[27] These studies suggest that in many patients both digestive and respiratory disease contribute to disease progression, likely in a self-perpetuating cycle. In such cases, case management is dependent on identifying and addressing both the digestive and respiratory components of disease.

Aspiration-related respiratory disorders reflect a subpopulation of AeroDs where gastric and/or oropharyngeal contents are aspirated into the respiratory tract. Detection of aspiration events presents an additional challenge because both macroaspiration and microaspiration events (ie, aspiration of microscopic particulates) can cause respiratory disease.[28] The ultimate prevalence of aspiration syndromes in dogs currently is unknown; however, they represent a significant source of morbidity and mortality in human patients and are associated with disease progression, exacerbations of clinical signs, and treatment costs.[29] Numerous conditions recognized in people also have been reported in dogs through individual case reports and case series.[19,30–33] A review of aspiration-related respiratory disorders in dogs has been published previously[30] and aspiration-associated respiratory syndromes reported in dogs are listed in **Table 1**.

Tissue Damage Secondary to Reflux and Aspiration

Reflux of GI secretions has been linked to diseases of the larynx, pharynx, and middle ear (via the eustachian tube) through the action of acid, digestive enzymes (ie, pepsin), and bile acids.[34,35] Although each substance is capable of causing damage independently, increased tissue damage is documented when they occur in combination, suggesting that management should address both the acidic and nonacidic components of reflux.[19,27,34]

Damage to the airways, and pulmonary parenchyma occurs through the aspiration of acid, digestive enzymes, and foreign material.[17,30,36–38] Aspiration events may be oropharyngeal or gastroesophageal in origin and may or may not be clinically observed.[39] Because 50% of healthy people aspirate during sleep without apparent clinical significance,[40] it is likely that the development of respiratory disease depends on the composition and volume of the aspirated material, and the presence of

functional airway protective mechanisms (ie, mucociliary clearance, and innate immunity).[41]

Inflammation in acidic aspiration is characterized by a biphasic response.[42] After aspiration, direct damage to the respiratory epithelium occurs immediately, followed hours later by neutrophilic inflammation. As in the upper airway, digestive enzymes also play a role in inflammation. Pepsin is directly cytotoxic, and bile acids have been documented in bronchoalveolar lavage fluid in human patients with reflux-associated respiratory disease and in dogs with pulmonary fibrosis.[43–47] In addition to acid and digestive enzymes, the aspiration of small particles also contributes to respiratory inflammation. In small animal models, tracheal instillation of small (<10 um) particles leads to neutrophilic inflammation.[48] Importantly, the combination of acidic aspiration and particulate aspiration appears to have a synergistic effect, where the combination results in more severe inflammation than either alone.[39,48]

Aspiration and Antibiotics

In people, the inhalation of low pH gastric fluid, digestive enzymes, and/or particulate material frequently leads to sterile inflammation (ie, aspiration pneumonitis).[49] This may or may not lead to a secondary bacterial complication (ie, aspiration pneumonia).[39,50] The distinction between aspiration pneumonitis and aspiration pneumonia is important, because treatment differs between these 2 conditions.[51] Aspiration pneumonitis is treated supportively, whereas aspiration pneumonia is treated with antimicrobials.[49,51] This distinction rarely is made in veterinary medicine and antibiotics frequently are initiated without objective evidence of bacterial infection. In dogs, the prevalence of secondary bacterial infection in aspiration pneumonia is unknown. Although prospective studies are needed, it is possible that antimicrobials are not always necessary and, as in people, inappropriate antibiotic use in dogs with aspiration pneumonitis may contribute to the development of resistant bacterial pathogens.[51]

Risk Factors

Increased risk for AeroDs have been reported for human patients with decreased consciousness, deficits in airway protection, dysphagia, and conditions where barriers to aspiration are overwhelmed (eg, vomiting and regurgitation).[48] Similar risk factors are seen in dogs. Diminished consciousness, body position during anesthetic recovery, duration of anesthesia, vomiting and regurgitation, seizures, cranial nerve deficits, and the presence of megaesophagus are independent risk factors for aspiration.[52,53] More than 1 risk factor may be present: 32% of dogs with aspiration pneumonia had greater than or equal to 2 risk factors.[53]

Common disorders associated with aspiration are esophageal disease (39.8%), vomiting (38.6%), neurologic disease (27.3%), laryngeal disease (18.2%), and anesthesia (13.6%).[53] Of those with esophageal disease, megaesophagus was identified in 71.4% of dogs. The remaining dogs were diagnosed with non-ME esophageal dysmotility, hiatal hernia, and an unknown disorder in 17.1%, 2.8%, and 8.6%, of dogs respectively.[53] Although focusing specifically on aspiration pneumonia, these studies highlight the role of digestive disease, in particular esophageal dysfunction, in the development of respiratory disease.

CLINICAL APPROACH

The range of conditions contributing to aerodigestive disease is broad and may be present in the absence of digestive signs. As such, conscious clinical recognition of these syndromes on the part of the veterinary practitioner is required.

Signalment can be used to help identify specific disease states. For example, congenital abnormalities usually are diagnosed at a young age and include persistent right aortic arch, cleft palate, and cricopharyngeal achalasia. Likewise, certain breeds are associated with a high incidence of dysphagia, including German shepherd dogs (vascular ring anomaly),[54] large breed dogs (masticatory muscle disorders),[55] golden retrievers (cricopharyngeal achalasia), and French bulldogs with sliding hiatal hernia.[56,57] Ultimately, however, AeroDs can occur in any breed regardless of age. Evaluation for AeroDs requires a thorough history. Identification may be aided by specific lines of questioning. These include identifying patients with recurrent disease (eg, recurrent aspiration pneumonia), identification and localization of dysphagia (ie, oral preparatory, pharyngeal, or esophageal), and inciting events (ie, eating and drinking or time of day) as well as historical vomiting/regurgitation events or recent anesthesia.[4] A thorough physical examination, including evaluation for comorbidities, and observation of swallowing are critically important components of evaluation for AeroDs. Specific clinical features suggestive of AeroDs are provided in **Table 2** and supplemental Video 1.

Initial Evaluation

Observation

Clinical episodes may be observed either at home or in the clinic. Observing a patient during feeding may allow a clinician to identify and localize dysphagia. Recording episodes is extremely helpful in patients with intermittent clinical signs (eg, reflux, sliding hiatal hernia).

Oral examination

A through oral examination is recommended in patients with evidence of AeroDs (**Fig. 1**). For example, patients with EER frequently have evidence of significant pharyngeal erythema. This may be performed in combination with a laryngeal function examination.

Minimum database

A minimum database may include, complete blood count, serum biochemical profile, and urinalysis. Unfortunately, findings may be non-specific. Changes in CBC, chemistry panel and urinalysis may be nonspecific.

Thoracic radiographs

Thoracic radiographs (minumum 3 views) are recommended. At least 1 lateral view should include the cervical trachea. Radiographs provide critical information on both primary diseases and comorbid conditions (eg, megaesophagus and/or aspiration pneumonia). When investigating reflux, fluid in the distal esophagus on thoracic radiographs occasionally may be observed in normal patients. Clinical significance is difficult to determine based on this finding alone. Unfortunately, radiographs lack the sensitivity to detect dynamic functional disorders or subtle pulmonary pathology. Furthermore, patients with AeroDs may present with normal thoracic radiographs.[4] AeroDs cannot be ruled out in patients with normal radiographs in the presence of supporting clinical signs.

Abdominal imaging

Abdominal imaging (radiographs/abdominal ultrasound) may be considered in patients with clinical signs suggestive of abdominal disorders (eg, vomiting and abdominal pain).

Treatment trials with client reporting

Treatment trials with client reporting are best used where reflux is suspected. Treatment trials are used in people with suspected reflux disease due to their simplicity,

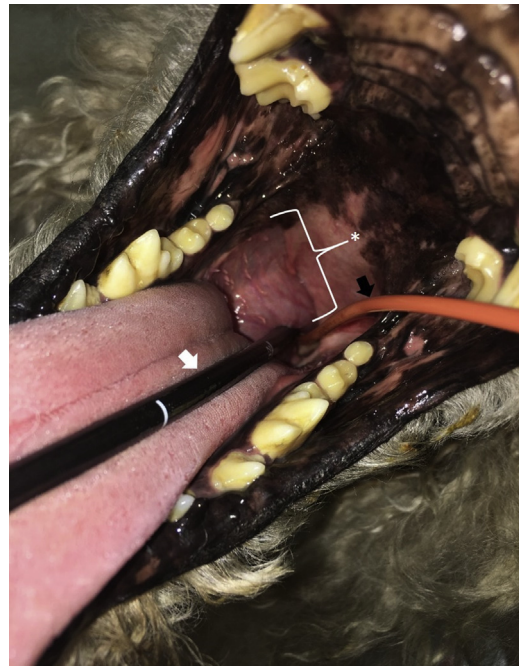

Fig. 1. A 13-year-old female spayed standard poodle was presented for aspiration pneumonia, a 4-week history of repetitive swallowing while eating, and increased upper respiratory noise (stertor). Oral examination prior to bronchoscopy (*white arrow*) revealed a large pharyngeal mucocele (*white brackets* and *asterisk*). Red rubber catheter is used to provide supplemental oxygen (*black arrow*).

noninvasiveness, and availability.[58] Although classically performed with proton pump inhibitors (eg, omeprazole, 1 mg/kg orally, every 12 hours, 30 minutes before feeding), a treatment trial also may include prokinetics (eg, metoclopramide, erythromycin or cisapride), especially where delayed gastric emptying is suspected. In human medicine, it is recommended that treatment trials for suspected reflux be performed for at least 8 weeks to 12 weeks in order to allow adequate time for a treatment response.[58]

Unfortunately, treatment trials are dependent on client reporting. Client reporting is inherently prone to bias due to variable client vigilance and a failure to recognize episodic, subtle clinical signs. Pretreatment and post-treatment surveys and visual analog scale (VAS) scores can help mitigate bias in client reporting.[59] Those patients who fail to respond to treatment trials should have such treatments discontinued appropriately to reduce risk of complications.[60] Additional diagnostics then are needed.

Advanced Testing

Laryngeal function examination

Laryngeal function examination is an important part of evaluation in patients with AeroDs. This is true particularly for patients with pharyngeal or esophageal dysphagia due to shared innervation between the pharynx, larynx, and the proximal esophagus.[4,33] Laryngeal dysfunction has been documented in dogs with reflux and in dogs lacking overt evidence of laryngeal disease (eg, stridor).[4,19] Because of this

relationship, swallow evaluation may be indicated prior to surgical correction of laryngeal paralysis to identify occult swallow dysfunction that may further increase the risk of aspiration events.[61] Laryngeal function examination may be performed in general practice but requires careful attention to technique.[61]

Videofluoroscopic swallow study

VFSSs have the benefit of allowing real time imaging (**Fig. 2**). They are considered the criterion standard for the evaluation of dysphagia in dogs.[24] Historically, these have been performed with dogs held in lateral recumbency and force-fed. This has limited use of VFFS in several disorders due to unacceptable risks of aspiration. Recent

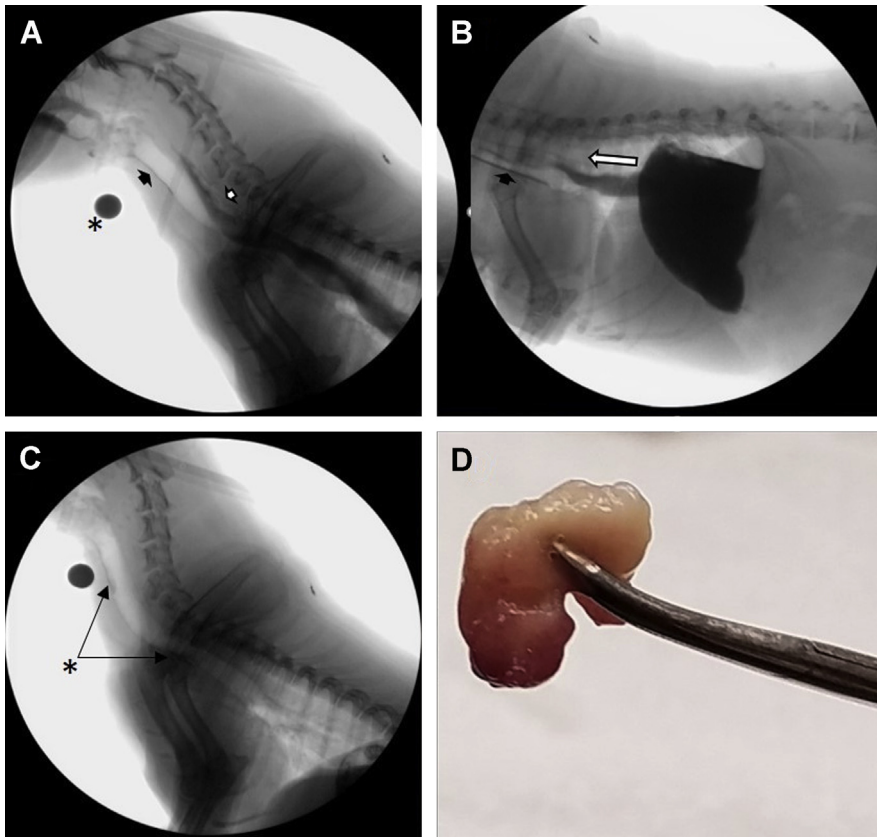

Fig. 2. This 12-year-old male castrated terrier cross was presented for a 4-month history of coughing while eating and drinking and nighttime restlessness. (*A*, *B*, *C*) Still images from a VFSS. (*A*) The dog was consuming a meal of liquid consistency. Liquid bolus is denoted by the white arrow. The degree of liquid accumulation in the esophagus was considered a normal variant due to multiple rapid swallow inhibition. Aspirated material is denoted by the black arrow; 1-cm size marker is denoted by the asterisk. (*B*) Aspirated contrast material is visible distal to the thoracic inlet (*black arrow*). Spontaneous reflux is denoted by the white arrow. (*C*) The arrows denote residual contrast material in the proximal and distal esophagus. Attempts at clearing this material (ie, cough) was conspicuously absent during image collection; 1-cm size marker is denoted by the asterisk. (*D*) On oral examination, a mass was found to obstruct normal adduction of the arytenoids. This was identified as an inflammatory polyp on histopathology.

studies have advocated an alternative which, by allowing free feeding, reduces risk of aspiration to what would be expected from feeding at home. Natural feeding position and bolus sizes further increase the physiologic relevance of VFSSs in dogs.[24,62] Objective swallowing metrics established using standardized recipes with rheological properties objectively consistent with commercially available products have been recently published.[24] Numerous swallowing abnormalities, including esophageal hypomotility, LES-AS, moderate–large volume reflux and macroaspiration, are detectable using this method. To date, LES-AS has been detected only by VFSSs.[63,64]

Respiratory fluoroscopy may be performed alone or in combination with VFSSs. If combined, it is recommended that respiratory fluoroscopy be performed first so that that airways and associated structures are not masked by contrast in the esophagus. Respiratory fluoroscopy is recommended particularly where static or dynamic architectural changes to larynx, trachea, and mainstem bronchi are suspected.

Unfortunately, the availability of fluoroscopy is currently limited to specialty practice and veterinary teaching hospitals. VFSS clips demonstrating EER and a sliding hiatal hernia are available in supplemental materials (Video 2 and Video 3, respectively).

Computed tomography

Computed tomography (CT) is useful for evaluating thoracic masses, extraluminal/intraluminal esophageal lesions, pharyngeal/retropharyngeal masses, and aspirated foreign material and for thorough evaluation of pulmonary diseases secondary to chronic aspiration (aspiration bronchiolitis and bronchiectasis) (**Fig. 3**). CT is considered the most sensitive diagnostic modality for the identification of bronchiectasis.[65] Survey radiography and a single breathing phase CT are both limited, however, in that they can only reliably detect static abnormalities changes. Because swallow disorders are dynamic, a more sensitive imaging modality is one that can evaluate swallowing in real time (ie, VFSSs).

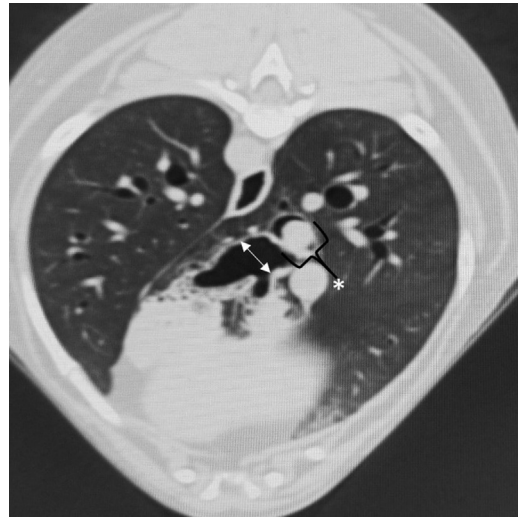

Fig. 3. This 8-year-old female spayed cocker spaniel was presented for a 9-month history of cough. Transverse CT image showed marked bronchiectasis of the accessory lobar bronchus (*double-headed arrow*) with soft tissue attenuating foreign material in the bronchial lumen of the accessory lung lobe (*brackets* and *asterisk*). Subsequent bronchoscopy identified complete occlusion of the accessory lobar bronchus with secretions and kibble foreign material.

Endoscopy

Endoscopy (**Fig. 4**) is routinely available in most specialty practices. Esophageal mucosal lesions may strongly support reflux esophagitis in dogs.[66] In human studies, however, endoscopy identified abnormalities in less than 50% of patients with known GERD.[67] A study in dogs demonstrated similar results, and supports this interpretation that esophagoscopy is a specific but poorly sensitive test for reflux disease in dogs.[66] Endoscopic evaluation of the nasopharynx and bronchoscopy with bronchoalveolar lavage fluid analysis (cytology and culture) remain an important mainstay for evaluation of airway and pulmonary parenchymal disease in dogs.

pH monitoring

Tests evaluating esophageal and gastric pH are rare outside of tertiary care centers or veterinary teaching hospitals. pH monitoring has proved invaluable in optimizing antacid therapy in dogs.[60,68] Diagnostic tests relying on esophageal or pharyngeal pH, however, fail to recognize reflux in human patients treated with proton pump inhibitors or those with nonacidic reflux (ie, secondary to digestive enzymes), which is increasingly implicated in disease representing up to 90% cases in some human studies.[47,69–71] The importance of nonacidic reflux in dogs currently is unknown.

Manometry

High-resolution manometry is considered a mainstay in evaluation of dysphagia in people. In veterinary medicine, however, it is significantly limited by availability, cost, and the need for substantial operator training.[72,73]

MANAGEMENT

Management is dependent on the underlying etiology. The key to management is appropriate patient identification and disease localization.

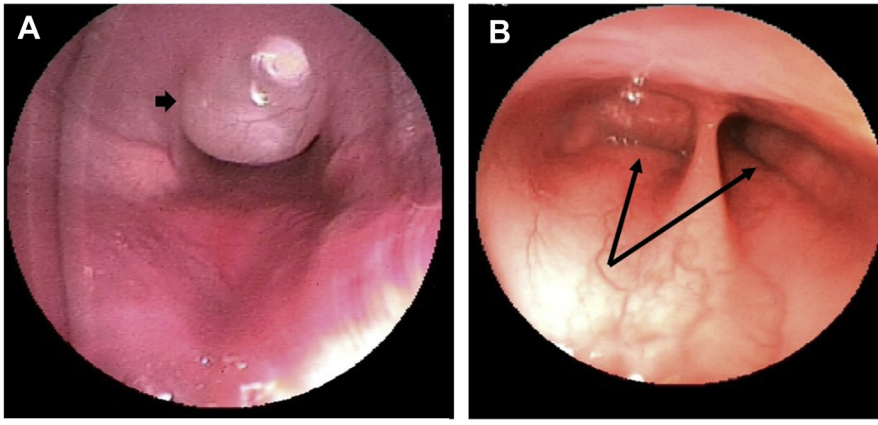

Fig. 4. This 9-year-old male castrated shih tzu was presented for a chronic history of cough, nasal discharge, suspected obstructive sleep apnea, and otitis media. Body condition score was 9/9. (*A*) Retroflexed choanal examination showed an obstructive fluctuant cyst (*arrow*) as well as diffuse nasopharyngeal erythema. (*B*) Rostral to the cyst, obstructive edematous nasopharyngeal turbinates (*arrows*) were noted. In addition, bilateral laryngeal paresis was noted on laryngeal function examination. A treatment-trial for EER was initiated. Weight loss also was recommended. Treatment response was noted after 4 weeks with decreased nasal discharge, nocturnal apneic episodes, and cough.

SUMMARY

The understanding of AeroDs in veterinary medicine is still in its infancy. Current evidence suggests, however, that dogs with AeroDs may represent a large and underrecognized patient population. Identification requires conscious awareness of the relationship between the respiratory and digestive tracts and AeroDs should be considered even in the absence of clinical signs of digestive disease and in dogs with normal thoracic radiographs.

DISCLOSURE

The author does not have any financial or personal relationships that could inappropriately influence or bias the content of this article. United States Patent No. 9,107,385 for the free-feeding kennels is held by the Curators of the University of Missouri, listing as inventors: Teresa Lever, Joan Coates, Mitchell Allen, and Laila Al-Khashti.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cvsm.2020.09.003.

REFERENCES

1. Groome M, Cotton JP, Borland M, et al. Prevalence of laryngopharyngeal reflux in a population with gastroesophageal reflux. Laryngoscope 2007;117(8):1424–8.
2. Koufman JA. Laryngopharyngeal reflux is different from classic gastroesophageal reflux disease. Ear Nose Throat J 2002;81(9 Suppl 2):7–9.
3. Celli BR, Thomas NE, Anderson JA, et al. Effect of pharmacotherapy on rate of decline of lung function in chronic obstructive pulmonary disease: results from the TORCH study. Am J Respir Crit Care Med 2008;178(4):332–8.
4. Grobman ME, Masseau I, Reinero CR. Aerodigestive disorders in dogs evaluated for cough using respiratory fluoroscopy and videofluoroscopic swallow studies. Vet J 2019;251:105344.
5. Poncet CM, Dupre 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(3):137–42.
6. Logemann J. Swallowing physiology and pathophysiology. Otolaryngol Clin North Am 1988;21(4):613–23.
7. Nishino T. Swallowing as a protective reflex for the upper respiratory tract. Anesthesiology 1993;79(3):588–601.
8. Paterson WG, Hynna-Liepert TT, Selucky M. Comparison of primary and secondary esophageal peristalsis in humans: effect of atropine. Am J Physiol 1991;260(1 Pt 1):G52–7.
9. Matsuo K, Palmer JB. Anatomy and physiology of feeding and swallowing: normal and abnormal. Phys Med Rehabil Clin N Am 2008;19(4):691–707,vii.
10. Bautista TG, Sun Q-J, Pilowsky PM. The generation of pharyngeal phase of swallow and its coordination with breathing: interaction between the swallow and respiratory central pattern generators. Progress in brain research, vol. 212. Elsevier; 2014. p. 253–75.
11. Hollshwandner CH, Brenman HS, Friedman MH. Role of afferent sensors in the initiation of swallowing in man. J Dent Res 1975;54(1):83–8.
12. Shaker R, Dodds WJ, Ren J, et al. Esophagoglottal closure reflex: a mechanism of airway protection. Gastroenterology 1992;102(3):857–61.

13. Shaker R, Ren J, Medda B, et al. Identification and characterization of the esophagoglottal closure reflex in a feline model. Am J Physiol 1994;266(1 Pt 1): G147–53.
14. Pollard RE. Imaging evaluation of dogs and cats with dysphagia. ISRN Vet Sci 2012;2012:238505.
15. Ettinger SJ, Feldman EC. Textbook of veterinary internal medicine: diseases of the dog and the cat, vol. 7. St. Louis (MO): Elsevier Saunders; 2010.
16. Smith JA, Houghton LA. The oesophagus and cough: laryngo-pharyngeal reflux, microaspiration and vagal reflexes. Cough 2013;9:12.
17. Houghton LA, Lee AS, Badri H, et al. Respiratory disease and the oesophagus: reflux, reflexes and microaspiration. Nat Rev Gastroenterol Hepatol 2016;13: 445–60.
18. Praud JP. Upper airway reflexes in response to gastric reflux. Paediatr Respir Rev 2010;11(4):208–12.
19. Lux CN, Archer TM, Lunsford KV. Gastroesophageal reflux and laryngeal dysfunction in a dog. J Am Vet Med Assoc 2012;240(9):1100–3.
20. Lecoindre P, Richard S. Digestive disorders associated with the chronic obstructive respiratory syndrome of brachycephalic dogs: 30 cases (1999-2001). Rev Med Vet (Toulouse) 2004;155(3):141–6.
21. Salguero R, Herrtage M, Holmes M, et al. Comparison between computed tomographic characteristics of the middle ear in nonbrachycephalic and brachycephalic dogs with obstructive airway syndrome. Vet Radiol Ultrasound 2016; 57(2):137–43.
22. Flouraki E, Kazakos G, Savvas I, et al. Rhinitis following intraoperative gastro-oesophageal reflux in a dog. Vet Rec Case Rep 2019;7(2):e000792.
23. Boesch RP, Shah P, Vaynblat M, et al. Relationship between upper airway obstruction and gastroesophageal reflux in a dog model. J Invest Surg 2005; 18(5):241–5.
24. Harris R, Grobman M, Allen M, et al. Standardization of a videofluoroscopic swallow study protocol to investigate dysphagia in dogs. J Vet Intern Med 2017;31(2): 383–93.
25. Gaude GS. Pulmonary manifestations of gastroesophageal reflux disease. Ann Thorac Med 2009;4(3):115–23.
26. Kahrilas PJ, Smith JA, Dicpinigaitis PV. A causal relationship between cough and gastroesophageal reflux disease (GERD) has been established: a pro/con debate. Lung 2014;192(1):39–46.
27. Tarvin KM, Twedt DC, Monnet E. Prospective controlled study of gastroesophageal reflux in dogs with naturally occurring laryngeal paralysis. Vet Surg 2016; 45(7):916–21.
28. Cardasis JJ, MacMahon H, Husain AN. The spectrum of lung disease due to chronic occult aspiration. Ann Am Thorac Soc 2014;11(6):865–73.
29. Dal Negro RW, Turco P, Micheletto C, et al. Cost analysis of GER-induced asthma: a controlled study vs. atopic asthma of comparable severity. Respir Med 2007; 101(8):1814–20.
30. Nafe L, Grobman M, Masseau I, et al. Aspiration-related respiratory disorders in dogs. J Am Vet Med Assoc 2018;253(3):293–300.
31. Hawkins EC, Basseches J, Berry CR, et al. Demographic, clinical, and radiographic features of bronchiectasis in dogs: 316 cases (1988-2000). J Am Vet Med Assoc 2003;223(11):1628–35.
32. Johnson LR. Laryngeal structure and function in dogs with cough. J Am Vet Med Assoc 2016;249(2):195–201.

33. Stanley BJ, Hauptman JG, Fritz MC, et al. Esophageal dysfunction in dogs with idiopathic laryngeal paralysis: a controlled cohort study. Vet Surg 2010;39(2): 139–49.
34. Adhami T, Goldblum JR, Richter JE, et al. The role of gastric and duodenal agents in laryngeal injury: an experimental canine model. Am J Gastroenterol 2004; 99(11):2098–106.
35. Nafe LA, Grobman ME, Masseau I, et al. Aspiration-related respiratory disorders in dogs. J Am Vet Med Assoc 2018;253(3):292–300.
36. Hu X, Yi ES, Ryu JH. Diffuse aspiration bronchiolitis: analysis of 20 consecutive patients. J Bras Pneumol 2015;41:161–6.
37. Lee AS, Ryu JH. Aspiration pneumonia and related syndromes. Mayo Clin Proc 2018;93(6):752–62.
38. Marik PE. Pulmonary aspiration syndromes. Curr Opin Pulm Med 2011;17(3): 148–54.
39. Hunt EB, Sullivan A, Galvin J, et al. Gastric aspiration and Its role in airway inflammation. Open Respir Med J 2018;12:1–10.
40. Gleeson K, Eggli DF, Maxwell SL. Quantitative aspiration during sleep in normal subjects. Chest 1997;111(5):1266–72.
41. Troche MS, Brandimore AE, Godoy J, et al. A framework for understanding shared substrates of airway protection. J Appl Oral Sci 2014;22(4):251–60.
42. Kennedy TP, Johnson KJ, Kunkel RG, et al. Acute acid aspiration lung injury in the rat: biphasic pathogenesis. Anesth Analg 1989;69(1):87–92.
43. Määttä OM, Laurila HP, Holopainen S, et al. Reflux aspiration in lungs of dogs with respiratory disease and in healthy West Highland White Terriers. J Vet Intern Med 2018;32(6):2074–81.
44. Pauwels A, Decraene A, Blondeau K, et al. Bile acids in sputum and increased airway inflammation in patients with cystic fibrosis. Chest 2012;141(6):1568–74.
45. Grabowski M, Kasran A, Seys S, et al. Pepsin and bile acids in induced sputum of chronic cough patients. Respir Med 2011;105(8):1257–61.
46. Tobey NA, Hosseini SS, Caymaz-Bor C, et al. The role of pepsin in acid injury to esophageal epithelium. Am J Gastroenterol 2001;96(11):3062–70.
47. Kahrilas PJ, Kia L. Pepsin: a silent biomarker for reflux aspiration or an active player in extra-esophageal mucosal injury? Chest 2015;148(2):300–1.
48. Raghavendran K, Nemzek J, Napolitano LM, et al. Aspiration-Induced lung injury. Crit Care Med 2011;39(4):818–26.
49. Prather AD, Smith TR, Poletto DM, et al. Aspiration-related lung diseases. J Thorac Imaging 2014;29(5):304–9.
50. Dent J, El-Serag H, Wallander MA, et al. Epidemiology of gastro-oesophageal reflux disease: a systematic review. Gut 2005;54(5):710–7.
51. DiBardino DM, Wunderink RG. Aspiration pneumonia: a review of modern trends. J Crit Care 2015;30(1):40–8.
52. Ovbey DH, Wilson DV, Bednarski RM, et al. Prevalence and risk factors for canine post-anesthetic aspiration pneumonia (1999–2009): a multicenter study. Vet Anaesth Analg 2014;41(2):127–36.
53. Kogan DA, Johnson LR, Sturges BK, et al. Etiology and clinical outcome in dogs with aspiration pneumonia: 88 cases (2004–2006). J Am Vet Med Assoc 2008; 233(11):1748–55.
54. Krebs IA, Lindsley S, Shaver S, et al. Short- and long-term outcome of dogs following surgical correction of a persistent right aortic arch. J Am Anim Hosp Assoc 2014;50(3):181–6.

55. Ryckman LR, Krahwinkel DJ, Sims MH, et al. Dysphagia as the primary clinical abnormality in two dogs with inflammatory myopathy. J Am Vet Med Assoc 2005;226(9):1519–1523, 1501.
56. Elliott RC. An anatomical and clinical review of cricopharyngeal achalasia in the dog. J S Afr Vet Assoc 2010;81(2):75–9.
57. Reeve E, Sutton D, Friend E, et al. Documenting the prevalence of hiatal hernia and oesophageal abnormalities in brachycephalic dogs using fluoroscopy. J Small Anim Pract 2017;58(12):703–8.
58. Gasiorowska A, Fass R. The proton pump inhibitor (PPI) test in GERD: does it still have a role? J Clin Gastroenterol 2008;42(8):867–74.
59. Paul-Dauphin A, Guillemin F, Virion J-M, et al. Bias and precision in visual analogue scales: a randomized controlled trial. Am J Epidemiol 1999;150(10): 1117–27.
60. Marks SL, Kook PH, Papich MG, et al. ACVIM consensus statement: support for rational administration of gastrointestinal protectants to dogs and cats. J Vet Intern Med 2018;32(6):1823–40.
61. MacPhail CM. Laryngeal disease in dogs and cats: an update. Vet Clin North Am Small Anim Pract 2020;50(2):295–310.
62. Bonadio C, Pollard RE, Dayton P, et al. Effects of body positioning on swallowing and esophageal transit in healthy dogs. J Vet Intern Med 2009;23(4):801–5.
63. Grobman ME, Schachtel J, Gyawali CP, et al. Videofluoroscopic swallow study features of lower esophageal sphincter achalasia-like syndrome in dogs. J Vet Intern Med 2019;33(5):1954–63.
64. Grobman M, Hutcheson K, Lever T, et al. Mechanical dilation, botulinum toxin A injection, and surgical myotomy with fundoplication for treatment of lower esophageal sphincter achalasia-like syndrome in dogs. J Vet Intern Med 2019;33(3): 1423–33.
65. Johnson LR, Johnson EG, Vernau W, et al. Bronchoscopy, imaging, and concurrent diseases in dogs with bronchiectasis: (2003-2014). J Vet Intern Med 2016; 30(1):247–54.
66. Munster M, Horauf A, Lubke-Becker A, et al. Idiopathic esophagopathies resembling gastroesophageal reflux disease in dogs. Tierarztl Prax Ausg K Kleintiere Heimtiere 2013;41(3):173–9.
67. Vaezi MF. Diagnosing gastroesophageal reflux disease with endoscopic-guided mucosal impedance. Gastroenterol Hepatol 2016;12(4):266.
68. Tolbert K, Bissett S, King A, et al. Efficacy of oral famotidine and 2 omeprazole formulations for the control of intragastric pH in dogs. J Vet Intern Med 2011; 25(1):47–54.
69. Bardhan KD, Strugala V, Dettmar PW. Reflux revisited: advancing the role of pepsin. Int J Otolaryngol 2012;2012:646901.
70. Johnston N, Dettmar PW, Bishwokarma B, et al. Activity/stability of human pepsin: implications for reflux attributed laryngeal disease. Laryngoscope 2007;117(6): 1036–9.
71. Sifrim D, Holloway R, Silny J, et al. Acid, nonacid, and gas reflux in patients with gastroesophageal reflux disease during ambulatory 24-hour pH-impedance recordings. Gastroenterology 2001;120(7):1588–98.
72. Kook P, Kempf J, Ruetten M, et al. Wireless ambulatory esophageal pH monitoring in dogs with clinical signs interpreted as gastroesophageal reflux. J Vet Intern Med 2014;28(6):1716–23.
73. Kempf J, Heinrich H, Reusch CE, et al. Evaluation of esophageal high-resolution manometry in awake and sedated dogs. Am J Vet Res 2013;74(6):895–900.

74. Gaynor EB. Gastroesophageal reflux as an etiologic factor in laryngeal complications of intubation. Laryngoscope 1988;98(9):972–9.
75. Carminato A, Vascellari M, Zotti A, et al. Imaging of exogenous lipoid pneumonia simulating lung malignancy in a dog. Can Vet J 2011;52(3):310–2.
76. Stadler K, Hartman S, Matheson J, et al. Computed tomographic imaging of dogs with primary laryngeal or tracheal airway obstruction. Vet Radiol Ultrasound 2011; 52(4):377–84.
77. Strøm P, Marks SL, Rivera J, et al. Dysphagia secondary to focal inflammatory myopathy and consequent dorsiflexion of the tongue in a dog. J Small Anim Pract 2018;59(11):714–8.
78. Smith MM. Surgery for cervical, sublingual, and pharyngeal mucocele. J Vet Dent 2010;27(4):268–73.
79. Pollard RE, Marks SL, Cheney DM, et al. Diagnostic outcome of contrast videofluoroscopic swallowing studies in 216 dysphagic dogs. Vet Radiol Ultrasound 2017;58(4):373–80.
80. Niimi A, Matsumoto H, Ueda T, et al. Impaired cough reflex in patients with recurrent pneumonia. Thorax 2003;58(2):152–3.