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Gastrointestinal Dysmotility and the Implications for Respiratory Disease

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Abstract

Purpose of the review: Gastroesophageal reflux disease (GERD) is frequently implicated as a cause for respiratory disease. However, there is growing evidence that upper gastrointestinal dysmotility may play a significantly larger role in genesis of respiratory symptoms and development of underlying pulmonary pathology. This paper will discuss the differential diagnosis for esophageal and gastric dysmotility in aerodigestive patients and will review the key diagnostic and therapeutic interventions for this dysmotility.

Recent findings: Previous studies have shown an association between GERD and pulmonary pathology in children with aerodigestive disorders. Recent publications have demonstrated the presence of esophageal and gastric dysfunction, using fluoroscopic and nuclear medicine studies, in aerodigestive patients who commonly present to pulmonary and otolaryngology clinics. High-resolution impedance manometry (HRIM) has revolutionized our understanding of esophageal dysmotility and its role in pathogenesis of aspiration and esophageal dysfunction and subsequent respiratory compromise.

Summary: Esophageal and gastric dysmotility have a profound effect on development of respiratory symptoms and pulmonary sequelae in aerodigestive patients. However, our understanding of the pathophysiology is in its infancy. Prospective studies are needed to address key clinical questions such as: What degree of dysmotility initiates respiratory compromise? What diagnostic tests and therapeutic options best predict aerodigestive outcomes?

Keywords

Dysmotility; fundoplication; high resolution esophageal manometry; aerodigestive; gastroesophageal reflux; impedance

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Compliance with Ethics Guidelines

Conflict of Interest

Lusine Ambartsumyan, Samuel Nurko, and Rachel Rosen declare no conflict of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

Introduction:

Gastroesophageal reflux disease (GERD) has long been implicated as a cause for (or associated with) respiratory symptoms and lung disease in children. Recent advances in technology have revealed that esophageal and gastric dysmotility may be more significant drivers of respiratory disease than gastroesophageal reflux alone. Gastrointestinal (GI) dysmotility and/or anatomic abnormalities (e.g. strictures, vascular compression) should be included in the differential diagnosis of any patient who presents with persistent respiratory symptoms before the symptoms are attributed to GERD. The treatment will vary depending on the underlying pathophysiology of GI dysmotility (Table 1). While even subtle motility disorders may cause symptoms, there are several key motility diagnoses to consider in the differential including disorders of the upper and lower esophageal sphincters, the esophageal body, and the stomach.

Cricopharyngeal Achalasia/Dysfunction

Cricopharyngeal dysfunction, in its most extreme form called cricopharyngeal achalasia, is characterized by either incomplete or dis-coordinated relaxation of the upper esophageal sphincter (UES) during swallowing (1–3). The lack of coordination between contraction of the pharynx and the relaxation of the UES results in a functional obstruction at the level of the cricopharyngeus muscle (2–4). Neonates and children may present with choking/gagging, coughing, excessive salivation, nasal reflux, regurgitation, failure to thrive, and recurrent respiratory tract infections from aspiration (2, 3, 5). It is considered to be primarily idiopathic in origin; however, it has been associated with neurological and mitochondrial disorders (3). Cricopharyngeal muscle hypertrophy and fibrosis have been reported on histopathology (6).(7). Videofluoroscopic swallow study or barium swallow study is used in children to evaluate the mechanics of deglutition, specifically signs of aspiration or laryngeal penetration. It classically shows pooling of contrast in the posterior pharynx, also known as the cricopharyngeal “bar”, and inadequate passage of contrast into the esophagus (2, 3) (Figure 1). High resolution esophageal manometry (HRM) is the gold standard test to diagnose UES dysfunction including elevated UES pressures and/or lack of coordination between pharyngeal contraction and UES relaxation (2, 3) (Figure 2). Pairing impedance with HRM is critical to determine the intrabolus pressures which predict risk for aspiration. Rommel et al used automated impedance manometry (AIM) to characterize the relationship between the UES pressure and bolus flow dynamics in children with aspiration, defining a swallow risk index to predict aspiration risk. Children with aspiration were found to have elevated post swallow pharyngeal residue and increased UES pressures during the maximum bolus flow (8). This technology offers the opportunity to follow patients longitudinally without the need for radiation, predict which patients will respond to therapies such as UES dilation, and understand the pathophysiology behind swallowing dysfunction. Currently, HRM is recommended for any patient with suspected UES dysfunction and/or aspiration (9).

Treatment in infants is often supportive and includes maintenance of nutrition with oral and enteral feeding as functional improvement has been reported over time (10). Depending on other comorbidities, some centers consider open and endoscopic cricopharyngeal myotomy (3, 4), cricopharyngeal botulinum toxin injection (2, 11), or endoscopic balloon dilations

(12, 13). The risks associated with the procedures, such as vocal cord paralysis, permanent disruption of the UES, and impairment of airway protective mechanisms, need to be carefully considered. The role of HRM with impedance to predict which candidates will respond to each therapy will be a critical next step in the management of these patients.

Primary motility disorders

Primary motility disorders of the esophagus typically occur in older children and teenagers. The most well characterized is esophageal achalasia which is defined as absent peristalsis of the esophageal body and abnormal relaxation of the lower esophageal sphincter (LES) (14, 15). It is rare in children with a reported incidence of 0.1 – 0.18 per 100, 000 cases annually (16, 17) and rare cases in infancy (18, 19). There is a higher incidence in adolescents and a mean age of diagnosis of 10.9 years (16). Associated genetic conditions include Allgrove syndrome or Triple “A” syndrome (achalasia, adrenal insufficiency, alacrima), Alport syndrome, and Down syndrome (20–22).

As with adults, children commonly present with dysphagia (56% - 94%), regurgitation of undigested foods (76% - 83%), and vomiting (59%–85%) (17, 22–26). Chest pain may be the presenting symptom in 23–38% of patients (22, 24, 26). Greater than fifty percent may develop weight loss and failure to thrive (22, 23). Younger children are more likely to present with respiratory symptoms (27, 28) with 24–41% having chronic cough, choking, or recurrent respiratory infections and aspiration pneumonias (22, 23, 26). Because of these high rates of respiratory symptoms, patients may present to aerodigestive, pulmonary, and otolaryngology centers with sole symptoms of nocturnal cough, regurgitation, or difficulty handling secretions.

During initial evaluation of chronic symptoms children may undergo various diagnostic studies including esophagram and an upper endoscopy, the findings of which may support the diagnosis of esophageal achalasia. Esophagram findings include a dilated esophagus, poor emptying of contrast, and a narrow esophagogastric junction (EGJ) with a classic bird-beak” appearance (Figure 3) (14, 15, 29, 30). The upper endoscopy may reveal a dilated esophagus that has retained saliva and food products, lack of stricture, candida overgrowth from chronic stasis, or even normal mucosa (14, 15, 29, 30). Notably, a normal appearing and open LES during endoscopy does not rule out achalasia. As part of the work up for chronic lung disease, a fluid filled esophagus on chest x-ray and/or a dilated esophagus on high resolution lung CT (HRCT) should trigger an evaluation of achalasia.

Respiratory sequela of achalasia are common but not always present. In a prospective study comparing adults with achalasia to controls, Makharia et al demonstrated that at time of presentation greater than 53% of patients had abnormalities of either pulmonary function or structure (31). Pulmonary function testing (PFTs) demonstrated obstructive and/or restrictive patterns and HRCT demonstrated parenchymal lesions ranging from ground glass opacities and fibrosis to bronchiectasis (31). Lung involvement is likely secondary to extrinsic compression, recurrent microaspiration of gastric contents, or microaspiration during swallowing from UES dysfunction which is a newly reported phenomenon (31, 32). Clearly

based on the degree of abnormalities seen on pulmonary testing, multidisciplinary care is important (31).

While all of the above tests are suggestive of achalasia, the gold standard diagnostic test for esophageal achalasia is HRM which shows classic findings of absent esophageal peristalsis +/- abnormal LES relaxation (Figure 4) (7, 14, 15, 29, 30). HRM allows for classification of esophageal achalasia into three subtypes (1,2 and 3) (29, 33), though which type is more associated with respiratory symptoms is not known. The diagnosis of achalasia in children can be challenging given the lack of standardized normative values in pediatrics. When performing HRM in children with aerodigestive symptoms including in those who aspirate, it is critical to give liquids (and ideally solids) during manometry in order to avoid false positive testing as dry swallows may not trigger peristaltic waves. Giving liquids is essential as it allows for assessment of esophageal stasis. From an aerodigestive perspective, the degree of esophageal stasis is crucial as it exposes the airway to food particles and to subsequent risk of micro-aspiration. The addition of impedance to HRM allows for clear visualization of the degree of esophageal emptying and stasis, and the extent of residual stasis in patients who have undergone treatment for achalasia. While HRM may be difficult to interpret without pediatrics normal values (34), when performed at specialized motility centers it has been shown to have excellent intra-rater variability and moderate inter-rater variability in differentiating achalasia from non-achalasia (35).

The goal of treatment is to alleviate the functional obstruction at the LES in order to facilitate effective esophageal emptying (14, 15, 29, 30). Treatment options include esophageal pneumatic balloon dilation (PD) and surgical myotomy. In pediatrics, response to pneumatic balloon dilation (PD) has been reported in 10–90% of patients with the majority of studies showing favorable responses with 1–2 dilations (17, 36, 37). Surgical myotomy is also an option with significant improvement in symptoms (38–40) and low complication and recurrence rates (26, 41). In a systematic review of 21 studies of 331 children, Pacilili and Davenport reported that laparoscopic Heller myotomy (LHM) was effective in 85% of children without differences in incidence of reflux or dysphagia between those with and without fundoplication (42). This latter point is critical as reflux control is not needed to improve symptoms. It is important to remember that if a fundoplication is performed during the myotomy, a secondary obstruction is created, which in concert with lack of esophageal motility could once again predispose to esophageal fluid retention and secondary aspiration. Finally, over the past decade a new minimally invasive endoscopic myotomy, known as Perioral Endoscopic Myotomy (POEM), has been shown to be effective in adults (43, 44) and recently in small pediatric case series (45–47).

There are no pediatric studies that address the role of achalasia therapies in the treatment of extraesophageal complications. Studies in adults show that pneumatic dilation or surgical myotomy are universally effective in treating not only dysphagia but especially respiratory symptoms in aerodigestive patients (48–51). Improvement in both pulmonary function on PFTs (48, 51) and parenchymal changes on HRCT (48) have been demonstrated in adults. The resolution of respiratory symptoms after improving esophageal drainage is a key concept in aerodigestive medicine. Of note, improvement in pulmonary function and symptoms occur despite increased gastroesophageal reflux which are associated with both

treatment modalities. This highlights that stasis and not reflux is the biggest driver of extraesophageal symptoms in children and adults. Interestingly, in addition to the effects of therapy on the LES, there is evidence that there may be upstream effects on the UES function such as a reduction in intrabolus pressures which may have implications for swallowing including a reduction in aspiration risk (32). Pulmonary outcome studies for children with achalasia are clearly needed.

Secondary Motility Disorders

Esophageal atresia (EA), with or without tracheo-esophageal fistula (TEF), is a congenital anomaly of the foregut with a worldwide incidence of 1:2500 – 1:4500 births (52–54). Approximately fifty percent of cases have an associated anomaly including VACTERL association, CHARGE syndrome, and trisomies (13, 18, 21) (52, 55). The Gross and the Vogt classification systems are used to classify EA/TEF into 5 types where distal TEF with proximal EA (Gross type C, Vogt type 3B) accounts for 88.5% of cases (54, 56). EA/TEF is commonly diagnosed shortly after birth and surgically repaired early in life. Advances in diagnostic modalities and surgical techniques have led to low rates of mortality and survival rates of greater than 90% (56, 57). However, children develop long-term respiratory and gastrointestinal complications associated with significant morbidity and impact on quality of life (54, 56, 58–60). Patients have gastrointestinal and pulmonary symptoms that persist from infancy to adulthood necessitating multidisciplinary care (61). Symptoms range from coughing, choking, wheezing, and respiratory infections to gagging, vomiting, feeding difficulties, weight loss, and cyanotic spells (62).

Pulmonary symptoms are prevalent across all age groups (61). Common respiratory symptoms include cough (8–75%), wheezing (14–40%), choking and dyspnea (37%). Presenting pulmonary diagnoses are bronchitis (14–74%), bronchiectasis (17%), recurrent respiratory tract infections (10–53%), tracheomalacia (14–29%), and pneumonia (5–50%) (59, 63–69). Restrictive (11–69%) and obstructive (38–50%) lung disease have been reported on pulmonary function testing (PFT) (58, 59, 64, 65, 70–72). Chetucti et al demonstrated poor lung function regardless of acute and ongoing presence of gastrointestinal and pulmonary symptoms, suggesting that close monitoring is important (64).

From a diagnostic perspective, it is important to investigate the potential gastrointestinal contributors to respiratory symptoms. These include esophageal stricture with subsequent stasis, missed or recurrent TEF, esophageal dysmotility causing stasis of food or acid refluxate, esophageal stasis secondary to an obstructive fundoplication, and aspiration during swallowing due to vocal cord paralysis or laryngeal cleft. A critical first step is to rule out anatomical abnormalities. An esophagram should be used to evaluate for strictures, hernias, status of a fundoplication if present, or fistulae. Following the esophagram, endoscopic evaluation of the esophagus is important to assess the anastomosis and the size of the proximal esophageal pouch, to investigate for hernias, the presence of fistulae, and to treat strictures that may be present. Endoscopy is also important to assess for mucosal abnormalities including infectious, peptic, or eosinophilic esophagitis as all may contribute to dysmotility or other morbidity. Reflux esophagitis is often the focus in children with

EA/TEF because of their poor clearance of acid refluxate and high rates of peptic esophagitis. However, recently eosinophilic esophagitis (EoE) has been recognized as a growing concern in this populations, in large part because of the impact on dysmotility. Dhaliwal et al reported that 17% of their pediatric cohort met diagnostic criteria for EoE, were more likely to present with symptoms of vomiting, dysphagia, and cyanotic spells, and had a 1.9 times relative risk for stricture formation (73). Patients with EoE are also at greater risk for respiratory symptoms including asthma and stridor as well as infiltration of their airway with eosinophils (74–78). In their pediatric cohort, Chat et al reported a significant reduction of symptoms of reflux and dysphagia, a decrease in prevalence of strictures and dilations, and improved feeding tolerance after effective treatment of EoE (79). Given the similarities of presenting symptoms between GER and EoE, current ESPGHAN/ASPGHAN consensus guidelines recommend evaluation for EoE in all symptomatic children who are refractory to PPI and are being considered for anti-reflux surgery (62).

Following endoscopy, an assessment of esophageal motility is indicated to assess for the presence or absence of peristalsis and/or esophageal stasis. Van Wijk et al reported that patients with intact esophageal motility completed bolus clearance 85.5% of the time, which was significantly higher when compared to those with abnormal motility who completed bolus clearance 29.7% of the time (80). These latter patients who have poor bolus clearance are at risk for aspiration of esophageal contents with resultant respiratory disease. Other studies support this degree of dysmotility and describe commonly seen motor patterns (81–84). In their pediatric cohort who underwent HRM, Lemoine et al characterized the esophageal dysmotility into three patterns: aperistalsis (38%), pressurization (15%), and distal contractions (47%) (Figure 5) (83). Outcome studies are clearly needed to compare the degree of dysmotility with pulmonary morbidity.

Finally, one of the most common diagnostic tests performed in patients with EA/TEF is reflux testing with pH-metry or pH-impedance, despite a lack of clear association between reflux test results and pulmonary outcomes (85). Furthermore, symptoms of reflux mimic symptoms of esophageal stasis such as nocturnal cough and feeling of food coming up into the mouth. Based on cross sectional studies, 33–63% of patients have reflux symptoms, and 20–55% have abnormal pH probe testing (62). The question is whether the abnormal testing is responsible for the symptoms. Furthermore, reflux testing needs to be interpreted with caution. Patients with EA/TEF commonly have esophageal dysmotility or esophagitis and consequently have low esophageal impedance baselines, which may make the number of reflux episode by impedance falsely low (81). Similarly, because of the dysmotility, any reflux that occurs may be poorly cleared, therefore while the number of reflux episodes appears low the esophageal contact time is prolonged. While acid suppression is recommended to prevent complications of long term acid damage, the decision to pursue antireflux surgery (fundoplication) is complicated from a motility and airway perspective. While effective in the short-term in preventing gastric contents from entering the esophagus, failure of the wrap may range between 20–45% within the first 1.5–2.5 years (86). Moreover, in a patient with esophageal dysmotility the fundoplication can create a functional outlet obstruction resulting in worsening symptoms including dysphagia, respiratory symptoms, and difficulty handling secretions. This obstruction creates esophageal stasis and

pooling of saliva, food products, and refluxate that once again predisposes the patient to aspiration and respiratory compromise (Figure 5).

The differential diagnosis for respiratory symptoms in children with EA/TEF should also include oropharyngeal dysphagia with aspiration. Videofluoroscopic swallow study (VFSS) and/or fiberoptic endoscopic swallow evaluation are used for diagnosis (62, 87). Symptoms of oropharyngeal dysphagia overlap entirely with symptoms of gastroesophageal reflux, therefore clinicians need to have a low threshold for evaluation (88). Children commonly present with feeding difficulties characterized by food refusal, cyanotic spells, and recurrent respiratory infections (87). If aspiration is diagnosed then the differential diagnosis should expand from oropharyngeal dysfunction to also include vocal cord paralysis (3–17%) (62, 89) and laryngeal cleft (26%) (90).

Lastly, the implications of gastric dysmotility should be considered in the evaluation of gastric and pulmonary symptoms in patients with EA/TEF. Gastric abnormalities have been reported in patient with EA/TEF and include delay in gastric emptying and dumping with or without fundoplication (54, 62, 80). Van Wijk et al reported a delay in gastric emptying in 71.4% of infants and 55.5 % of adults in their cohort who underwent ¹³C-octanoate breath test (80). The role of gastric dysmotility in development or progression of pulmonary disease in patients with EA/TEF remains to be elucidated.

Motility Considerations in Lung Transplant

Bronchiolitis obliterans syndrome (BOS) is the most common cause of chronic lung allograft dysfunction (CLAD) and affects up to 80% of adult and greater than 50% of pediatric lung transplant recipients at 5 years post-transplant (91, 92). Gastroesophageal reflux disease (GERD) and silent aspiration of gastric contents has been associated with BOS and allograft failure in adults (91, 93). To date, there are only two publications that address the role of GERD in pediatric lung transplant patients. Benden et al reported that 9 out of 10 patients had abnormal esophageal acid exposure using pH-metry where all three patients with acute rejection had pathological reflux (94). Dy et al reported that of their cohort 50% had pathological reflux on pH-metry or pH-MII (92). Interestingly, in the latter study, neither the reflux burden nor fundoplication were associated with allograft rejection but delayed gastric emptying (DGE) was linked to CLAD independent of GER on multivariate analysis (92). A delay in gastric emptying has been reported in adults pre and post lung transplantation (95, 96). Raviv et al demonstrated an association between DGE at pre and 3 months post-transplant and BOS (96). Esophageal dysmotility, in addition to gastric dysmotility may also play a role in rejection. Abnormal esophageal peristalsis and hypercontractile esophagus have been reported with increased frequency in post lung transplant patients (97) and these motility changes may be associated with increased risk of rejection. Tagaroonsanti et al used HRIM to demonstrate that esophagogastric junction outflow obstruction (EGJOO), incomplete bolus clearance (IBC), and proximal reflux were risk factors for CLAD (98). They also reported that hypo-contractility on HRIM (defined by the Chicago classification v2.0) was associated with IBC and abnormal number of reflux events, both previously implicated in CLAD (99). Outflow obstruction in conjunction with abnormal bolus clearance perpetuate esophageal stasis and delayed esophageal acid

clearance, therefore increasing the risk of aspiration. These findings highlight the multifaceted role that alterations in gastrointestinal motility play in the development of CLAD.

In children who have undergone transplantation, the goal of management is to treat the esophageal and/or gastric dysmotility in order to facilitate transit and reduce stasis, thereby minimizing the risk of aspiration. While there are no pediatric studies to date that assess specific therapeutic interventions, azithromycin (100) and pyloric botulinum toxin-A injections (95) have been reported to decrease reflux burden and improve gastric emptying respectively in adult lung transplant recipients. Moreover, post-pyloric feeds may need to be initiated in children who fail therapeutic modification of esophageal and/or gastric dysmotility.

Systemic Scleroderma

Juvenile systemic scleroderma (jSSc) is a progressive connective tissue disease characterized by multisystem organ involvement including cardiac, renal, respiratory and gastrointestinal systems (101). The hallmark findings of esophageal dysmotility in systemic scleroderma (SSc) are absent esophageal peristalsis and a hypotensive LES. The absent peristalsis predisposes patients to esophageal stasis and the hypotensive LES predisposes patients to gastroesophageal reflux episodes. As with other motility disorders, the gold standard test to diagnose esophageal dysmotility is HRM. In the only pediatric study to date, using esophageal manometry, Guariso et al reported that 35% of their localized scleroderma pediatric cohort had abnormalities of esophageal peristalsis and LES tone (102). Other testing that may provide clues to the diagnosis include findings of patulous, dilated, or fluid filled esophagus on HRCT, that may have been performed for the evaluation of pulmonary symptoms.

Interstitial lung disease (ILD) has been associated with GI dysmotility. ILD is a common pulmonary manifestation of juvenile systemic scleroderma (jSSc) with significant morbidity and mortality (101, 103). GERD has been associated with pulmonary abnormalities in adults with SSc (104–106). Patients with ILD have been shown to have increased reflux burden compared to those without ILD using pH-impedance (105). However, abnormal findings on esophageal manometry have been associated with ILD on HRCT and with changes in pulmonary function on PFTs (107, 108). To date there are no pediatric studies evaluating the role of esophageal dysmotility in the development or progression of ILD. Poor LES pressures, esophageal dysmotility, and abnormal bolus clearance create the perfect setting for esophageal stasis of food and acidic refluxate subsequently increasing the risk of aspiration and progressive lung disease. This is especially concerning in patients who have severe disease with oropharyngeal dysphagia and are known to have risk of aspiration or penetration on VFSS. Early recognition of esophageal dysmotility is paramount as it may indicate visceral involvement and disease progression consequently necessitating escalation of systemic treatment by the Rheumatologist.

As with jSSc, esophageal dysmotility should also be suspected in children with polymyositis, dermatomyositis, Raynaud's phenomenon, and mixed connective tissue diseases who present with respiratory symptoms.

Post-Fundoplication Esophageal Obstruction

Respiratory symptoms in aerodigestive patients with history of fundoplication should prompt interrogation of the fundoplication integrity and its impact on esophageal function. The fundoplication may be too tight and cause an esophageal outlet obstruction. It may also slip and result in either a herniation into the chest which can compress and obstruct the lower esophagus, or a bisection of the stomach which can affect stomach capacity. Symptoms may include weight loss, dysphagia, nausea, emesis, feeding difficulties/refusal, retching, gagging, coughing, choking, and difficulty handling secretions. In children with aerodigestive symptoms, the fundoplication needs to be assessed from above by giving contrast orally or by a nasoesophageal tube, and when possible from below by instilling contrast into the gastrostomy tube. This will assess for esophageal air-fluid levels and identify the location of the fundoplication imprint relative to the chest and stomach. An important next step in the evaluation may be HRM to assess for esophageal outflow obstruction, stasis, and elevations in intrabolar pressure that suggest a functional obstruction (Figure 6) (109–111). There is no data on the relationship between respiratory symptoms and post-fundoplication complications. Patients with post-operative dysphagia have been shown to have elevated intrabolar pressures and bolus flow dynamics represented by an elevated dysphagia risk index pre and postoperatively using automated impedance manometry (109, 110). Due to risk of esophageal obstruction and the need for repeat surgeries in those who undergo early fundoplication, whenever possible, post-pyloric feeding is considered a preferred alternative to fundoplication in children with respiratory symptoms. The rates of reflux are identical between fundoplication and post-pyloric feeding with the advantage that post-pyloric feeds do not result in esophageal obstruction (112, 113). When esophageal obstruction with stasis is diagnosed in children with aerodigestive disorders, it is absolutely crucial to relieve the distal obstruction by botulinum toxin-A injection of the LES, balloon dilation of the fundoplication, or even surgically taking down the fundoplication.

Conclusion:

Understanding role of dysmotility in aerodigestive patients is in its infancy. Prospective studies are needed to answer key clinical questions such as: What degree of dysmotility triggers respiratory symptoms? What therapeutic options for relieving esophageal obstruction or for treating gastroparesis will result in improved aerodigestive outcomes? What diagnostic tests of dysmotility best predict aerodigestive outcomes? Multidisciplinary research is needed to move the field of aerodigestive medicine beyond the diagnosis and treatment of GERD to embody esophageal and gastric motility disorders.

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Figure 1. Esophageal filling defect consistent with cricopharyngeal dysfunction. *Arrow* points to the filling defect.

Figure 2. Absent relaxation of the upper esophageal sphincter (*circle*). White impedance waves superimposed on the pressure tracings show persistent bolus presence in the oropharynx.

Figure 3. Esophagram of a patient with achalasia. (A) An air-fluid level in the proximal esophagus (arrow). (B) The classic bird's beak appearance to the lower esophageal sphincter.

Figure 4.

High resolution esophageal manometry with impedance in a patient with Type 2 Achalasia.

(A) Absent peristalsis, absent relaxation of the lower esophageal sphincter, and pan-pressurization of the esophagus. (B) Stasis of fluid throughout the esophagus, with low impedance values, indicating liquid in the esophagus, shown in *pink and circled*. The addition of impedance to the tracing shows clear stasis, putting the patient at risk for aspiration that could not be seen with standard high resolution manometry alone.

Figure 5. High resolution esophageal manometry with impedance in a patient with esophageal atresia. The upper esophageal sphincter and lower esophageal sphincter relax appropriately but there is absent peristalsis and stasis in the esophagus, shown by impedance in *pink (circle)*.

Figure 6. High resolution esophageal manometry in a patient who underwent a fundoplication. **(A)** There is normal peristalsis with incomplete relaxation of the lower esophageal sphincter (LES). **(B)** Evidence of stasis shown by impedance in pink throughout the esophagus including immediately above the area of the fundoplication/LES (*circles*).

Table 1:

The Pathophysiology of GI dysmotility associated with respiratory complications.

1. Gastroesophageal Reflux
2. Esophageal Stasis   a. Lack of peristalsis   a. Esophageal achalasia   b. Scleroderma   c. Esophageal atresia   b. Esophageal stricture with proximal stasis   c. Vascular rings   d. Distal obstruction   a. Esophageal achalasia   b. Fundoplication
3. Oropharyngeal Dysphagia   a. Anatomic abnormalities   a. Laryngeal cleft   b. Vocal cord paralysis   c. Palatal malformations   b. Dysmotility   a. Cricopharyngeal dysfunction   b. Pharyngeal dysfunction
4. Gastric Dysmotility
5. Anatomic Malformations   a. Tracheoesophageal fistula   a. Primary or recurrent
6. Correctable Causes of Esophageal Dysmotility   a. Eosinophilic esophagitis   b. Hypothyroidism   c. Medications