

TITLE: Esophagology and Esophagoscopy

**SOURCE: Grand Rounds Presentation, The University of Texas Medical Branch
(UTMB Health), Department of Otolaryngology**

DATE: October 27, 2011

RESIDENT PHYSICIAN: Benjamin Walton, MD

FACULTY PHYSICIAN: Michael Underbrink, MD

DISCUSSANT: Michael Underbrink, MD

SERIES EDITOR: Francis B. Quinn, Jr., MD

ARCHIVIST: Melinda Stoner Quinn, MSICS

"This material was prepared by resident physicians in partial fulfillment of educational requirements established for the Postgraduate Training Program of the UTMB Department of Otolaryngology/Head and Neck Surgery and was not intended for clinical use in its present form. It was prepared for the purpose of stimulating group discussion in a conference setting. No warranties, either express or implied, are made with respect to its accuracy, completeness, or timeliness. The material does not necessarily reflect the current or past opinions of members of the UTMB faculty and should not be used for purposes of diagnosis or treatment without consulting appropriate literature sources and informed professional opinion."

Introduction

The Otolaryngologist needs to be familiar with the esophagus, its anatomy, disease process, diagnostic evaluation, and treatment. The esophagus is somewhat unique in that its location is not confined to the head and neck. It is an organ system that traverses several specialty divisions. The Otolaryngologist is an important member in the treatment team for esophageal abnormalities which also includes the Gastroenterologist and Cardiothoracic surgeon. New advancements in endoscopic technology have further expanded the role of the Otolaryngologist in the field of esophagology. In-office esophagoscopy has become a safe and reliable tool in diagnosis and treatment of various esophageal abnormalities.

Anatomy

The esophagus is a vertical muscular tube that extends from the hypopharynx to the stomach. It is generally 23-25 cm in length and passes through the neck, superior mediastinum and posterior mediastinum anterior to the cervical and thoracic vertebrae. The esophagus generally has a slight left curve in its descent before returning to the midline. In the superior mediastinum, the esophagus runs posterior to the trachea in contact with the common carotid arteries. Importantly, the recurrent laryngeal nerves run along the esophagus in the tracheoesophageal groove. Some esophageal pathology may compress or infiltrate these nerves causing hoarseness. Generally, the thoracic duct lies to the left of the esophagus. The esophagus passes posterior and to the right of the descending aorta on its way down to the stomach until the inferior mediastinum where the esophagus passes anterior and slightly to the left of the aorta. The left main bronchus crosses anterior and indents the esophagus below the arch of the aorta. The Aorta, left main stem bronchus and diaphragm are three sites of external compression. These three anatomical locations are sites of esophageal compression and can be seen on routine esophagoscopy. In the thorax, the right vagus nerve descends posterior to the esophagus and the left vagus nerve descends anteriorly. The muscles of the esophagus consist of an inner circular layer of muscle continuous with the inferior constrictor muscles of the pharynx and an outer longitudinal layer. Between these two layers, the Auerbach's myenteric plexus sits. This nervous plexus is responsible for peristalsis. The outer longitudinal layer of muscle is arranged in fascicles at its proximal site attaching to the cricoid. More distally, the fascicles blend to form a uniform layer surrounding the esophagus. The outer longitudinal muscle layer is composed of striated muscle in the upper third, mixed striated and

smooth muscle in the middle third, and smooth muscle in the lower third of the esophagus. Unlike the other organs composing the digestive tract the outer coat of the esophagus is a loose fibroelastic tissue rather than a strong serosa.

The esophagus is lined by a non-keratinized stratified squamous epithelium which covers a thin lamina propria. The muscularis mucosae is a smooth muscle arranged longitudinally deep to the lamina propria. This muscle generally thickens down in the lower third of the esophagus. A submucosal layer consists of thick collagenous and coarse elastic fibers. This layer contains mucus glands and Meissner's plexus.

Access into and out of the esophagus is controlled by two sphincters. The upper esophageal sphincter (UES) is a high pressure zone. It consists of an inner circular layer of muscle that is thicker at the cricoids blending with the cricopharyngeal muscle. The lower esophageal sphincter is no one distinct muscle. It is generally 3 cm in length and has a resting tone of 15-45 mm Hg. This resting tone is important to keep in mind as pathology such as achalasia distorts its resting tone. The lower esophageal sphincter (LES) relaxes in response to swallowing, secondary peristalsis, and occasionally without peristalsis. It may descend into the abdomen 1 to 3 cm with normal respiration. The LES may also relax transiently secondary to a vagally-mediated reflex. This is a normal part of digestion which is triggered by gastric distention. This is termed Transient Lower Esophageal Sphincter Relaxation and is the primary mechanism for gastroesophageal reflux in normal individuals and for those with mild gastroesophageal reflux disease.

Swallowing and Dysphagia

Swallowing, also known as primary peristalsis is the mechanism in which a bolus of food is passed from the oral cavity into the stomach. A tight coordination of multiple muscle factors works to push the bolus down. Any problem with a muscle group in this process can lead to dysphagia. The UES, esophageal body, and LES work in a coordinated behavior to transfer food through the esophagus. There are three main phases in swallowing known as the oral phase, oropharyngeal phase and esophageal phase. The oral phase of swallowing is voluntary; however, the esophageal phase is not. Bolus passage through the mouth into the pharynx is known as the oropharyngeal phase. This phase predicated on very precise timing of the opening and closure of various portions of the pharynx for proper swallowing and airway protection. The larynx elevates in this phase allowing the epiglottis to seal the airway. The food bolus then passes through the relaxed UES. The UES then closes and peristalsis begins as progressive circular contractions propel the bolus down the esophagus through a relaxed LES. Normal esophageal pressures during peristalsis can range from 30 to 180 mm Hg. There is a secondary peristalsis that occurs in the esophagus. Secondary peristalsis is progressive contraction in the esophageal body stimulated by sensory receptors rather than by swallowing. This mechanism clears food that has been poorly cleared by primary peristalsis.

Through precise movement and coordinated muscular activity, the food bolus travels into the stomach. However, any process that disrupts this coordinated activity can cause dysphagia. Dysphagia is defined as a sensation of food being delayed in its normal passage from the mouth to the stomach. As it is defined, there are multiple reasons a patient can have dysphagia. One way to further define dysphagia is to understand the differences in sensation. Patients who generally complain of difficulty with initiating a swallow have what is termed oropharyngeal dysphagia. Patients who complain that food "sticks" after swallowing have what is termed esophageal dysphagia. Important in the evaluation of dysphagia is a good, thorough clinical history. Distinguishing the type of dysphagia can be helpful in

creating a specific diagnosis. Patients complaining of solid food dysphagia often have a structural lesion. Intermittent solid food dysphagia may represent an esophageal ring. Dysphagia to solids and liquids likely represents a motility disorder as the cause of dysphagia.

Oropharyngeal dysphagia is sometimes termed transfer dysphagia. The oropharyngeal phase of swallowing is where precise movements of the oropharynx, hypopharynx, and esophagus allow both transfer of food and airway protection. This type of dysphagia generally arises from diseases of the upper esophagus, pharynx, or UES. Patient generally will present with complaints of difficulty initiating a swallow with immediate cough, gag, or nasal regurgitation. Patients with complaints of dysphagia to the throat or cervical area are less likely to have primary esophageal dysphagia as studies have shown 30% of patients who localize their difficulty to the cervical or throat area have esophageal dysphagia. Oropharyngeal dysphagia is most commonly caused by neuromuscular dysfunction. There are many causes of oropharyngeal dysphagia. Neuromuscular causes include CVA, amyotrophic lateral sclerosis, Parkinson's disease, Myasthenia gravis, and Tardive dyskinesia. Structural causes include cervical osteophytes, zenker's diverticulum, tumors, and post-cricoid webs.

Esophageal dysphagia encompasses a large number of causes. It is often best to group these causes into four main categories. Causes of esophageal dysphagia are grouped into motility abnormalities, strictures, rings and webs, and eosinophilic esophagitis.

Esophageal motility disorders have undergone several classification systems. The latest system consists of 4 major patterns: inadequate relaxation of the LES, atypical disorders of LES relaxation, hypercontraction disorders, and hypocontraction disorders. Disorders of inadequate relaxation of the LES include classical Achalasia and atypical disorders of LES relaxation. Classical achalasia is a primary esophageal motility disorder. It generally is of unknown etiology; however, Chagas disease caused by infection with the *Trypanosoma cruzi* protozoa. Histological examination shows patchy inflammatory infiltrates of T cells, eosinophils, and mast cells into the Auerbach's myenteric plexus. These patients present with dysphagia to solids and liquids. Most patients try to accommodate this dysphagia with several maneuvers. 75% of these patients present with regurgitation and 60% of these patients complain of weight loss. However, in most patients, this loss is generally minimal. Diagnosis of achalasia is done with a barium esophagram with fluoroscopy. The esophagram reveals the classic "bird's beak" tapering of the distal esophagus. Esophageal manometry reveals aperistalsis in the body of the esophagus with baseline elevation of LES pressure. There is currently no cure for Achalasia. Treatments generally include pneumatic dilation and surgical myotomy. Importantly, all patients considered for dilation must be surgical candidates as dilation incurs a 2-5% risk of perforation. Surgical treatment includes the Heller's myotomy which is an anterior myotomy across the LES. This procedure is often done with an anti-reflux procedure. Alternatively, non-surgical candidates can be treated with botulinum toxin injections into the LES. This treatment is effective in 85% of the patients. However, its long term efficacy is not good as 90% patients have symptom return in 6 months.

The spectrum of esophageal dysphagia disorders of uncoordinated contraction includes diffuse esophageal spasm. Diffuse esophageal spasm is the presence of simultaneous and repetitive contractions in the esophageal body. Unlike achalasia, patients with diffuse esophageal spasm have some normal peristalsis. Often, these patients are diagnosed after substantial work-up for cardiac-related disorders is negative. The barium esophagram shows the classic "corkscrew esophagus." Treatment of this disorder includes nitrates and calcium channel blockers.

Hypercontraction esophageal disorders include nutcracker esophagus and isolated hypertensive LES. Nutcracker esophagus is more commonly seen of the two hypercontractile disorders. Generally, these patients present with noncardiac chest pain. Esophageal manometry reveals high-amplitude peristalsis. Many experts believe that nutcracker esophagus is not a true primary motility disorder.

Disorders of hypocontraction include ineffective esophageal motility. Ineffective esophageal motility is diagnosed when esophageal manometry reveals contraction amplitudes of less than 30 mm Hg in 30% or more of wet swallows. There is a higher incidence in patients with GERD.

It is important to remember that there are also other factors that can cause motility disorders. There are many systemic conditions that can cause secondary motility disorders. Most commonly, scleroderma or progressive systemic sclerosis can cause esophageal dysmotility. Also, hypothyroidism, diabetes mellitus, and amyloidosis can cause dysmotility.

Strictures can occur along the entire length of the esophagus and cause dysphagia in patients. A stricture is defined as a loss of lumen area within the esophagus. The normal diameter of the esophagus is 20 mm, and generally symptoms of dysphagia occur when the diameter of the lumen is less than 15 mm. Generally strictures are defined under two broad categories. Strictures are generally either classified as intrinsic or extrinsic. Intrinsic strictures include peptic acid-induced, pill-induced, chemical, post-NG intubation, infectious esophagitis, sclerotherapy-induced, irradiation-induced, esophageal/gastric malignancies, congenital, systemic inflammatory disease and epidermolysis bullosa. Extrinsic causes of strictures include pulmonary/mediastinal malignancies, anomalous vessels and aneurysms, and metastatic submucosal infiltration by breast cancer, mesothelioma, and adenocarcinoma of the gastric cardia. The foundation of treatment of esophageal strictures is esophageal dilation. There are several types of esophageal dilator available and include the Maloney bougies and Savary-Gilliard dilators. Recently balloon dilators have become increasingly more popular in esophageal dilation. Esophageal dilation is not without complications. Most commonly, bacteremia occurs in 20-50% of patients. Esophageal dilation incurs a 0.5% risk of perforation. Also, there is a 0.3% risk of bleeding. Radiation and malignancy-induced strictures are at greater risk of perforation. To safely dilate the esophagus, many experts agree to follow the "rule-of-threes." In one setting for esophageal dilation, one should not dilate more than three times in one session. The goal of dilation is to get the esophageal objective diameter of greater than 15 mm. Studies have shown that 90% of patients in whom the esophagus is dilated to 15 mm do not have recurrence of their dysphagia at 24 month follow-up. Esophageal strictures that do not resolve after standard treatment are termed refractory esophageal strictures. Often the causes of these strictures include nonsteroidal anti-inflammatory drugs, pill injury, uncontrolled acid reflux, and inability to achieve adequate lumen diameter by dilation. Treatment for refractory strictures includes removing the offending agent and gentle dilation to 15 mm. Studies have shown intralesional steroid injections are safe and may be effective in refractory strictures.

Rings or webs are structural abnormalities that are often found incidentally in asymptomatic patients. An esophageal web is defined as a circumferential lesion of the esophagus that can consist of either mucosa or muscle. Esophageal rings generally occur in the distal esophagus. There are two major types of esophageal ring. A B ring or Schatzki's ring occurs at the distal margin of the LES. Schatzki's ring is the most common cause of intermittent solid food dysphagia and food impaction in the general population. There is controversy among experts in regards to the cause of Schatzki's ring. There is also an entity known as an A ring which is a muscular ring that occurs generally at the proximal margin of the LES 2 cm proximal to the squamocolumnar junction. Webs generally only occupy part of

the lumen. Esophageal webs are always mucosal unlike rings and mostly occur in the proximal esophagus. For rings and webs, barium esophagram is the most sensitive diagnostic tool. Interestingly, esophageal webs may be related to a specific syndrome known as Plummer-Vinson or Paterson-Kelly Syndrome. Two Gastroenterologists in the US and two Otolaryngologists in the UK noted the association of proximal esophageal webs, iron-deficiency anemia and dysphagia.

One of the newer pathology that has become more and more relevant in esophagology is eosinophilic esophagitis. On esophagoscopy, there are findings of multiple esophageal rings. Diagnosis of eosinophilic esophagitis is made by biopsy showing esophageal eosinophilia. Definitive diagnosis requires greater than 15 eosinophils per high-power field of mucosa that then does not clear after appropriate treatment with a PPI. In many patients, this finding is associated with other atopic diseases including eczema and asthma. There is often a strong family history of atopy as well. In the pediatric and adolescent population, eosinophilic esophagitis is increasingly recognized as the cause of dysphagia and food impaction in young adults. In the pediatric population, dietary modification and food elimination are effective treatment. Often times, dilation is necessary in the older population.

Barrett's Esophagus

Barrett's esophagus is the most significant outcome of chronic GERD. This condition predisposes patients to the development of esophageal adenocarcinoma. The histopathology of Barrett's is such that normal stratified squamous epithelium of the distal esophagus is replaced by intestinal columnar metaplasia. Studies find that 6-12% of patients undergoing endoscopy for GERD have Barrett's esophagus and in this population the risk of esophageal adenocarcinoma is 0.5% annually. Current endoscopic surveillance guidelines require four quadrant biopsies at 2 cm intervals along the entire length of Barrett's esophagus every 3 years. As will be discussed later, there is a distinct role of the Otolaryngologist in this practice.

In-office Dysphagia Consult

Dr. Jonathan Aviv has developed a guideline for patients who present in the clinic with the chief complaint of dysphagia. Up to 10% of patients who see an Otolaryngologist are presenting with laryngopharyngeal reflux. LPR is present in up to 50% of patients with hoarseness and in 51% of patients who present with dysphagia. It is important to have a step-by-step process in the work-up and management of these patients. Dr. Aviv divides patients into two categories. Patient who present with cough, throat clearing and hoarseness but deny dysphagia are separated from patients who present with a chief complaint of dysphagia. Dr. Aviv has helped to develop a testing system that utilizes laryngeal sensory testing. The endoscope delivers a discrete pulse of air to the epithelium innervated by the internal branch of the superior laryngeal nerve to elicit the laryngeal adductor reflex. This air pulse is 50 ms in width and is delivered by a channel associated with the flexible laryngoscope. Normal sensory threshold is less than 4 mm Hg air pulse pressure. Moderate deficit is defined as 4.1-60. mm Hg air pulse pressure. Severe deficit is defined as greater than 6.1 mm Hg air pulse pressure.

Patients complaining of cough, throat clearing or hoarseness first undergo flexible laryngoscopy with laryngeal sensory testing. Patients with findings of LPR and no sensory deficits are then started on standard PPI treatment for 6-8 weeks with follow-up. Any patients with persistent symptoms afterward, are asked to undergo trans-nasal esophagoscopy. Those patients with LPR and asymmetrical sensory deficit undergo imaging to rule out a neoplasm. If the imaging is negative, those patients undergo trans-nasal esophagoscopy. Patients with tumors or malignancy found under imaging or initial evaluation or referred to the appropriate physician.

Patients who present with a chief complaint of dysphagia are offered a flexible endoscopic evaluation of swallowing with sensory testing (FEESST). This testing assesses both airway protection and bolus transport of patients. The testing utilizes laryngeal sensory testing and endoscopic swallowing evaluation. The first phase of the testing involves flexible laryngoscopy where the anatomy of the nasopharynx, tongue base, hypopharynx, larynx and vocal folds are evaluated. The physician should assess proper velopharyngeal closure, vocal fold mobility, baseline secretion management, pharyngeal muscle strength, and laryngeal elevation. The second phase of the test involves laryngeal sensory testing of the laryngopharynx. The third phase of testing is the motor evaluation of swallowing with the administration of food consistencies varying from thin liquids to solids. Patients undergoing this testing generally require a trans-nasal esophagoscopy for further evaluation.

Trans-nasal Esophagoscopy

Trans-nasal esophagoscopy is quickly growing in its importance in esophagology. The technique is an important advance in the care of patients with reflux, dysphagia, and esophageal pathology. Advances in technology allow for brilliant illumination and excellent image quality. The endoscope is capable of air-insufflation and irrigation along with a 2 mm working channel. For trans-nasal esophagoscopy there is no conscious sedation required. This is a decided advantage for the general population as the risk of sedation is negated. Relatively new to esophagoscopy, TNE has a long list of indications which continues to grow. These indications are divided into esophageal and extra-esophageal indications. According to the ASGE and ACG, indications for TNE include: esophageal symptoms persisting despite adequate therapy, dysphagia, odynophagia, weight loss, anorexia, radiologically-demonstrated lesions, acute injury after caustic ingestion, greater than 5 years symptoms of GERD or GERD and greater than 50 years old, continuous anti-reflux therapy, foreign body evaluation and possible removal, cirrhosis screening for varices, and guide-wire placement of esophageal manometry. Therapeutic indications include: dilation of strictures, placement of a feeding tube, botox treatment of achalasia, laser therapy, and placement of wireless pH telemetry capsules. Relative extra-esophageal indications include: globus pharyngeus, chronic cough, cervical dysphagia, asthma or COPD, odynophagia, hemoptysis, LPRD, and head and neck cancer. Trans-nasal esophagoscopy performs a limited evaluation of the upper gastrointestinal tract and often does not view the distal stomach or duodenum. A study by Wildi et al shows that patients with reflux symptoms without abdominal complaints including daily pain and nausea with a history of ulcer disease are highly unlikely to have a major disease involving the stomach or duodenum. Their study shows that daily abdominal pain and nausea with a history of ulcer disease is a strong predictor of major gastric and duodenal diseases.

The technique for trans-nasal esophagoscopy is straight-forward and requires little set-up. It is preferable that patients be NPO for 3 hours. The technique does not require any IV or conscious sedation; however, adequate topical nasal anesthesia and decongestion is required. Many authors agree that there should be minimal hypopharyngeal anesthesia to prevent secretion build-up and subsequent coughing or aspiration of the secretions. The esophagoscope can be held in a variety of manners. Most utilize either the standard or the fishing pole technique. The lubricated esophagoscope is passed through the nare and then inserted into the esophagus. There are generally two techniques to intubate the esophagus. The first technique asks the patient to burp, then the scope is passed posterior to the cricoids and into the cervical esophagus. The other technique involves the patient tucking their chin toward the chest and then swallowing. The esophagoscope tip is then passed above the arytenoids or into the left pyriform. Gentle pressure is then applied as the scope is passed through the esophagus. Any excessive

resistance that is found in intubating the esophagus should terminate the procedure. The patient should then undergo a bariums swallow or modified barium swallow. After intubation of the esophagus, the scope is then passed to the region of the squamocolumnar junction and LES. Patients are asked to swallow which will then open the LES. A sniffing technique will allow evaluation for a diaphragmatic hernia. The scope is then passed through the LES into the stomach. At that point, the scope is retroflexed to view the LES. After evaluation of the stomach and LES, the stomach is suctioned free of air to help decrease post-procedure belching and vomiting. The middle and proximal esophagus is then evaluated on withdrawal. With generous air insufflations, the post-cricoid area is then visualized as the endoscope is removed. Often, this evaluation requires post-procedure evaluation of the procedure video to ensure no masses or lesions are missed. Postma et al evaluated over 700 patients undergoing TNE and found that the procedure was well-tolerated in 98% of the patients.

During esophagoscopy, there are several landmarks that were mentioned earlier. The aorta compresses the esophagus generally 24 mm from the nasal ala. The left mainstem bronchus compresses the esophagus at 26 mm from the nasal ala. The third major esophageal landmark is the diaphragm which compresses the esophagus at 41 mm from the nasal ala.

As discussed earlier, Barrett's esophagus is a predisposing factor for developing adenocarcinoma of the esophagus. Esophageal carcinoma is the fastest growing cancer of the USA and Western Europe in the past 25 years. Unfortunately, esophageal carcinoma is generally detected in an advanced stage when symptoms occur. The 5 year survival rate for symptomatic patients is less than 10% in advanced stages. Early stage adenocarcinoma of the esophagus has a much better survival rate with 5 year survival rates reaching 90%. Saeian et al studied the accuracy of trans-nasal esophagoscopy in the detection of Barrett's metaplasia and dysplasia. TNE utilizes a 2 mm working channel and the biopsies are generally smaller. The study took 32 patients with Barrett's metaplasia who underwent quadrant biopsies with standard equipment and TNE biopsy forceps at least 1 week apart. Two pathologists blinded to the study evaluated the specimens. The study found excellent agreement in the quality of both biopsy specimens. Trans-nasal esophagoscopy allows for a safe and effective manner to detect and survey Barrett's metaplasia in patients. One of the best advantages is the ability to perform the procedure without conscious sedation. This is often the cause of most complications from esophagogastroduodenoscopy.

There are multiple procedures that can be performed in the unsedated patient through trans-nasal esophagoscopy. Biopsies can be safely performed using a 1.8 mm cupped forceps through the working channel of the scope. Biopsies can be safely taken in any area of the upper aerodigestive tract with proper topical anesthesia. TNE can be utilized to place wireless pH probes. The esophagoscope can localize the upper and lower esophageal sphincters and determine the accurate position of the pH probes. Generally, the distal pH sensor is 6 cm above the squamocolumnar junction. The hypopharyngeal sensor is placed 1 cm above the proximal border of the upper esophageal sphincter. The working channel of the scope can be used to pass a flexible laser wire. Often, the KTP laser is utilized. This technique is most often used for laryngeal and hypopharyngeal lesions. Esophageal dilation can be safely performed without sedation. Generally this is performed with a guide wire placed through the trans-nasal esophagoscope. A Savary dilator or hydrostatic balloon dilator can be utilized in dilation. The Savary dilator utilized a guide wire which is placed under direct visualization with the esophagoscope. The esophagoscope is then removed while the guide wire is kept in place. The guide wire is then extracted through the oral cavity with a Kelly clamp. The dilator is then passed over the guide wire. Balloon dilation is a newer technique that allows for visualization of the dilation.

Unfortunately, the working channel of the trans-nasal esophagoscope is too small for passing the balloon through the channel. In a similar fashion to the Savary dilator, a guide wire is passed under direct visualization of the esophagoscope through the working channel. The esophagoscope is then removed with the guidewire in place. The balloon is passed over the guide wire with the esophagoscope to allow for direct visualization of the balloon passage.

Trans-nasal esophagoscopy can also be utilized to perform a secondary trachea-esophageal puncture. A secondary TEP can be safely performed using TNE in the office. The technique utilizes an 18 gauge needle, a TEP dilatory, local anesthesia and a 15 blade scalpel.

Botulinum toxin injections can be safely performed in patients using trans-nasal esophagoscopy. Botulinum toxin injections are useful in the treatment of patients with achalasia, hypertensive lower esophageal sphincter, distal esophageal spasm, nutcracker esophagus, and obstructing muscular rings. The technique involves an endoscopic sclerotherapy needle through the working channel of the scope. 100 units of Botulinum toxin is suspended into 4 ml of sterile saline. In preparing the needle, one must account for the dead space in the long injection needle. 0.5 ml of toxin is injected into the LES musculature with 2 injections per quadrant as the esophagus is divided into 4 quadrants.

Conclusions

Esophagology is an important aspect in the world of the Otolaryngologist. Otolaryngologists often see patients with both extra-esophageal and esophageal complaints. These patients often will benefit from trans-nasal esophagoscopy. Utilizing both a good understanding of anatomy, physiology, and pathology, the Otolaryngologist can play a crucial role in diagnosis and treatment of patients with many complaints and conditions. Trans-nasal esophagoscopy is safe to perform and utilizes the skills in endoscopy the Otolaryngologist utilizes daily. There are currently multiple indications for TNE and these continue to grow. As we understand more about Barrett's esophagus and the role of the Otolaryngologist, trans-nasal esophagoscopy will likely expand. It is likely that trans-nasal esophagoscopy will become more relevant in the next 5 to 10 years.

Comments by Dr. Underbrink on Esophagology by Dr. Walton

That was an excellent talk, very well done, and I think that this information is becoming more and more important as we delve into this new technology that we've become accustomed to. The esophagus is a difficult structure because it's partitioned and based on the anatomical regions, it is the province of different specialists, the gastroenterologist, the thoracic surgeon, and the otolaryngologist.

Many esophageal problems present first to the otolaryngologist, in the form of dysphagia, regurgitation, and laryngopharyngeal reflex symptoms. Thus it is important for us to gain a good knowledge of the anatomy and physiology of this structure as we go forward into practice with our transnasal esophagoscopes.

My second point is about Dr. Aviv's sensory testing, which is a wonderful thing. Unfortunately the pump portion of the apparatus is no longer available to us, and it would have been helpful to measure sensation before administering a barium swallow, so as to avoid aspiration

in the insensate portion of the swallowing mechanism. We generally start with a thick barium bolus, but some aspiration is always present.

The indications for TNE (transnasal esophagoscopy) are all over the place, and I think that as more and more otolaryngologists are doing TNE the indications become softer and softer, but the important thing is that if you treat empirically for things that you think might be reflex with dysphagia or silent reflux you might consider assessing the esophagus or at least advising the patient to undergo TNE after about three months of treatment because the incidence of adenocarcinoma in Barrett's esophagus is going up.

Our problem is "When do we refer this patient to a gastroenterologist?" Generally, if during the TNE you see something you're worried about or don't understand you're probably better off putting the referral in. Now, I like to look into the stomach, peek into the duodenum, but if I see anything suggestive of peptic ulcer disease, or if my biopsy for *H. pylori* comes back positive, I'll go ahead and refer to our gastroenterologist. It is unusual, however, for that type of patient, one with abdominal pain and ulcer symptoms, to come to the otolaryngologist.

Bibliography

Amin MR, Postma GN, Setzen M, Koufman JA. "Transnasal esophagoscopy: A position statement from the American Bronchoesophagological Association (ABEA)." Otolaryngology-Head and Neck Surgery. 2008; 138: 411-414.

Aviv JE. "Prospective, Randomized Outcome Study of Endoscopy Versus Modified Barium Swallow in Patients with Dysphagia." The Laryngoscope. 2000; 110: 564-574.

Aviv JE. "The Office Dysphagia Consult: Diagnosis, Management, and Coding." Presented at AAO-HNSF Annual Meeting, October 2009.

Aviv JE, Murry T, Cohen M, Zschommler, Gartner C. "Flexible Endoscopic Evaluation of Swallowing and Sensory Testing: Patient Characteristics and Analysis of Safety in 1,340 Consecutive Examinations." Ann Otol Rhinol Laryngol. 2005; 114: 173-176.

Chheda NN, Postma GN. "Transnasal Esophagoscopy." Flint: Cummings Otolaryngology: Head & Neck Surgery, 5th ed. Mosby, 2010.

Dale OT, Alhamarneh O, Young K, Mohan S. "Laryngeal sensory testing in the assessment of patients with laryngopharyngeal reflux." The Journal of Laryngology & Otology. 2010; 124: 330-332.

Falcone MT, Garrett CG, Slaughter JC, Vaezi M. "Transnasal esophagoscopy findings: Interspecialty comparison." Otolaryngology-Head and Neck Surgery. 2009; 140: 812-815.

Ford CN. "Evaluation and Management of Laryngopharyngeal Reflux." JAMA. 2005; 294: 1534-15400.

Kahrilas PJ, Pandolfino JE. “Esophageal Neuromuscular Function and Motility Disorders.” Feldman: Sleisenger and Fordtran’s Gastrointestinal and Liver Disease, 9th ed. Saunders, 2010.

Koufman JA, Aviv JE, Casiano RR, Shaw GY. “Laryngopharyngeal reflux: Position statement of the Committee on Speech, Voice, and Swallowing Disorders of the American Academy of Otolaryngology–Head and Neck Surgery.” Otolaryngol Head Neck Surg. 2002; 127: 32-35.

Long JD, Orlando RC. “Anatomy, Histology, Embryology, and Developmental Anomalies of the Esophagus.” Feldman: Sleisenger and Fordtran’s Gastrointestinal and Liver Disease, 9th ed. Saunders, 2010.

Postma GN, Cohen JT, Belafsky PC, Halum SL, Supta SK, Bach KK, Koufman JA. “Transnasal Esophagoscopy: Revisited (over 700 Consecutive Cases).” The Laryngoscope. 2005; 115: 321-323.

Postma GN, Belafsky PC, Aviv JE. Atlas of Transnasal Esophagoscopy. 1st edition. Lippincott Williams & Wilkins, 2007.

Rees CJ. “In-office transnasal esophagoscope-guided botulinum toxin injection of the lower esophageal sphincter.” Current Opinion in Otolaryngology & Head and Neck Surgery. 2007;15: 409-411.

Rees CJ. “In-office unsedated transnasal balloon dilation of the esophagus and trachea.” Current Opinion in Otolaryngology & Head and Neck Surgery. 2007; 15: 401-404.

Saeian K, Staff DM, Vasilopoulos S, Townsend WF, Almagro UA, Komorowski RA, Choi H, Shaker R. “Unsedated transnasal endoscopy accurately detects Barrett’s metaplasia and dysplasia.” Gastrointestinal Endoscopy. 2002; 56: 472-478.

Vaezi, MF. “The Esophagus: Anatomy, Physiology, and Diseases.” Flint: Cummings Otolaryngology: Head & Neck Surgery, 5th ed. Mosby, 2010.

Wildli SM, Glenn TJ, Woolson RF, Wang W, Hawes RH, Wallace MB. “Is esophagoscopy alone sufficient for patients with reflux symptoms?” Gastrointestinal Endoscopy. 2004; 59: 349-354.