Continuing Education Independent Study Series

SELECTED ASPECTS OF DYSPHAGIA: WEBS, RINGS, AND DIVERTICULA
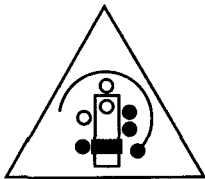
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PREFACE

"Selected Aspects of Dysphagia: Webs, Rings, and Diverticula" is part of the AST Continuing Education Independent Study Series. The series has been specifically designed for surgical technologists to provide independent study opportunities that are relevant to the field and to support the educational goals of the profession and the Association.

Acknowledgments

INTRODUCTION

Purpose

The purpose of this module is to acquaint the learner with the association between the symptom of dysphagia and a variety of underlying esophageal disorders; the methods of diagnosis and treatment currently used to address the manifestations of dysphagia; and the means of treating esophageal webs, rings, and diverticula associated with dysphagia. Upon completing this module, the learner will receive 2 continuing education (CE) credits in category 3 (applicable to CFA certification).

Objectives

Upon completing this module, the learner will be able to do the following:

1. Accurately describe the anatomy and physiology of the esophagus, differentiate the swallowing phases, and identify the role of the esophageal sphincters in dysphagia.
2. Describe the different types of dysphagia, including their respective etiology, complications, and comorbidities.
3. Delineate the methods used to diagnose the variety of ulterior disorders in dysphagic patients; in addition, describe the clinical purposes of each method.
4. List the characteristics of the following esophageal disorders: gastroesophageal reflux disease (GERD), achalasia, diffuse esophageal spasm (DES), and esophageal webs, rings, and diverticula.
5. Describe the methods of diagnosis and treatment (both surgical and nonsurgical) that currently are used to address such disorders, and provide an historical perspective in each instance.
6. Where possible, list the postsurgical complications associated with the different types of surgical procedures, and mention any complications that can arise in patients whose esophageal disorders remain untreated.

Using the Module

1. Read the information provided, referring to the appropriate figures.
2. Complete the enclosed exam without referring back to the text. The questions are in a multiple-choice format. Select the best answer from the alternatives given.
3. Mail the completed exam to AST, CEIS Series, 7108-C S. Alton Way, Suite 100, Englewood, CO 80112-2106. Please keep a copy of your answers before mailing the exam. You must return the original copy of the answer sheet; this exam may not be copied and distributed to others.
4. Your exam will be graded, and you will be awarded continuing education credit upon achieving a minimum passing score of 70%. If you are an AST member, your credits will be automatically recorded and you do not need to submit the credits with your yearly CE report form.
5. You will be sent the correct answers to the exam. Compare your answers with the correct answers to evaluate your level of knowledge and determine what areas you need to review.

Studying Technical Material

To study technical material, find a quiet place where you can work uninterrupted. Sitting at a desk or work table will be most conducive to studying.

Having a medical dictionary available as you study is very helpful so you can look up any words with which you are unfamiliar. Make notes in the margins of any new definitions so that you can review them.

The ultimate test of how well you learn this material is your ability to relate your knowledge to what is happening in the surgical field. Apply your knowledge to the assessment of patients with these disorders and their post-treatment results.

SELECTED ASPECTS OF DYSPHAGIA: WEBS, RINGS, AND DIVERTICULA

Esophageal Anatomy and Physiology

The esophagus generally is considered in terms of its three segments: upper, middle, and lower thirds. Three areas of natural constriction occur in the esophagus: the cricopharyngeus muscle, the crossing of the aorta with the left mainstem bronchus anteriorly, and the lower esophageal sphincter (LES). The esophagus consists of an inner mucosal layer, a middle submucosa, and an outer muscular layer. Stratified squamous epithelium lines the esophagus with the exception of the distal 3 cm, which is lined by columnar epithelium. Blood supply follows a segmental organization, and innervation occurs through the vagus and sympathetic nerves. Motor function generally is thought to be under vagal control, but other sources may be contributory.

The esophagus transports nutrients, prevents regurgitation, and ventilates excess gaseous materials from the stomach; furthermore, it is one component of the swallowing mechanism. Three separate phases of swallowing occur: oral, pharyngeal, and esophageal. The **oral phase** consists of bolus preparation, segregation, and transport to the pharynx. This process is under voluntary control and involves striated muscles innervated by cranial nerves. The nasopharynx and larynx are closed during the **pharyngeal phase**, accompanied by relaxation of the upper esophageal sphincter (UES) and transfer of bolus to the esophagus. The **esophageal phase** requires LES receptive relaxation, transfer of bolus to the stomach, and subsequent reestablishment of LES tone. The speed of peristaltic progression is most rapid in the upper, striated muscle portion (upper one-third) and slower in the distal, smooth muscle segment. Primary peristalsis provides bolus transport throughout the length of the esophagus; secondary peristalsis clears any remaining food when initiated by distention or reflux. Tertiary contractions are nonperistaltic.

The UES consists of the cricopharyngeus muscle and is a transitional segment between the pharynx and the esophagus. It has resting pressures between 50 mm and 100 mm Hg. Relaxation of the UES occurs just prior to arrival of the pharyngeal peristaltic contraction. The swallowing center in the medulla oblongata of the brain coordinates this complex sequence of events, and cranial nerves V, VII, IX, X, and XII all play a role. The LES measures 2 cm to 4 cm in length and is a zone of increased pressure in the range of 10 mm to 40 mm Hg. Incompetence of the LES leads to reflux, whereas incomplete relaxation can contribute to dysphagia. The LES is under the control of autonomic nerves and is affected by intrinsic (smooth) muscle activity as well as hormones.

Dysphagia

Dysphagia indicates an abnormality in the swallowing mechanism and is a common disorder. Dysphagia is associated with many diseases; therefore, obtaining a detailed history of the patient is crucial in directing the patient's workup. Of interest are the patient's account of his or her initial awareness of a change in swallowing and such details as the types of food being ingested and under what circumstances the symptoms appear. In addition, noting the progression from initial appearance of symptoms to the current state of illness is important. Abnormalities in any of the three swallowing phases can cause dysphagia.

Oral dysphagia is an uncommon, isolated disorder that often has a neurogenic cause; other causes include decreased salivary flow or painful oropharyngeal lesions. Neurogenic dysphagia creates a situation in which the patient appears to have forgotten what to do when a bolus is taken orally. The neuro-

logic defect involves cortical function and/or corticobulbar impulse transmission. Sudden onset of oral dysphagia can be attributed to cerebrovascular disease, whereas a slowly progressive course is a classic indicator of degenerative or demyelinating diseases, for example, Alzheimer's, Huntington's, amyotrophic lateral sclerosis, or multiple sclerosis. Neurologic causes often are associated with weakness of the lips, drooling, or speech difficulties. Diminished production of saliva renders oral bolus preparation difficult. Salivary flow can be affected by more than 400 commonly used drugs, such as antihistamines, anticholinergics, antidepressants, antihypertensives, and diuretics. Autoimmune disorders such as Sjogren's syndrome can cause dry mucous membranes. Oropharyngeal lesions causing odynophagia also may lead to dysphagia: In most instances, these lesions are caused by self-limiting disorders such as viral pharyngitis. Immunosuppressed patients, however, may be afflicted with lesions associated with such serious conditions as candidiasis or herpes.

Pharyngeal dysphagia often is described as a sensation of difficult passage of the bolus through the region above the suprasternal notch. This experience frequently is accompanied by coughing, choking, nasal regurgitation, or even aspiration. This type of dysphagia is most frequently caused by neuromuscular disorders of striated muscles and less frequently by a structural narrowing. Most neurologic conditions causing pharyngeal dysphagia tend to involve the peripheral nervous system, which is manifested by weakness, lack or coordination, or a movement disorder. Conversely, myasthenia gravis is a myoneural disease that involves the cranial nerves, and early symptoms include ptosis, diplopia, voice change, or dysphagia. In this disorder, use of muscles increases muscular weakness. Dermatomyositis is another disorder that can cause dysphagia with the classic features of a heliotrope rash over the eyelids, Grotton's papules over interphalangeal joints, polyarthritis, and myalgia. Thyroid-related myopathy must be considered in elderly patients whose symptoms include proximal weakness, tachycardia, fasciculation, and a fine tremor. Structural narrowing of the esophagus may be caused by the presence of a neoplasm, Zenker's diverticulum, or mucosal web, among other possible forms of obstruction.

Patients with **esophageal dysphagia** may experience a difficulty in swallowing that is described as occurring at any point between the suprasternal notch and the epigastrium. Two general causes of esophageal dysphagia are a fixed anatomic narrowing and an interference with the mechanisms of receptive relaxation and propulsive peristalsis. The central nervous system normally is not found to be a significant cause because of its minimal effect on smooth muscle. Peptic strictures, neoplasms, and esophageal webs and rings all can cause a structural narrowing. This type of dysphagia usually begins with difficulty in swallowing solids and often progresses to a stage at which liquids can not pass. Rapid progression of symptoms is suspicious for carcinoma. Gastroesophageal motility abnormalities produce intermittent dysphagia when ingesting both solids and liquids and can worsen when the patient is distracted. Esophageal dysphagia frequently is associated with diffuse esophageal spasm, achalasia, and inflammation secondary to conditions such as reflux, ulceration, infectious esophagitis, or radiation exposure. Localized esophagitis can occur if medications are held in contact with esophageal mucosa for an extended time. This condition is particularly common in the elderly who ingest tablets at bedtime. Bullous dermatoses may cause inflammation in the esophagus; examples of these disorders include Stevens-Johnson syndrome, toxic epidermal necrolysis, dystrophic epidermolysis bullosa, and pemphigoid.

Identification of the source of dysphagia can be precise when pertaining to the pharynx, but less so for the esophagus. Associated symptoms can aid the physician in the location of the source. Coughing or choking often implies a neuromuscular disorder involving the pharyngeal swallowing phase. If these symptoms occur immediately after swallowing, a Zenker's diverticulum or high-obstructing lesion may be the cause. Symptoms occurring later during sleep are characteristic of either Zenker's diverticulum, achalasia, or reflux. Odynophagia often indicates the presence of an ulcerative lesion if the pain is

described as sharp or as a spasm if it is dull or squeezing. Whereas regurgitation while eating is nonspecific, later regurgitation of undigested food suggests the presence of a diverticulum or distal esophageal obstruction. Regurgitation of sour or bitter material is a classic characteristic of gastroesophageal reflux. The sensation of a lump or fullness in the throat (globus) can accompany dysphagia, particularly when the conditions of reflux or esophageal spasm are present.

Patient Evaluation

The diagnostic workup of a patient with dysphagia varies depending on the clinical assessment of signs and symptoms that are elicited during history-taking and physical examination. The physical examination should include indirect or flexible laryngoscopy as well as palpation of the neck. If no problems are identified during the head and neck examination, some clinicians may elect to initiate treatment for a benign process such as reflux. Otherwise, the sequence of diagnostic testing depends on whether the symptoms are primarily obstructive or painful.

A complete blood count should be obtained to rule out leukocytosis or anemia. Immunologic studies can be conducted if collagen diseases such as scleroderma are suspected. Cytologic examination of exfoliated mucosal cells is recommended for patients older than 40 or for those experiencing rapidly progressive symptoms or weight loss. A full-column esophagography (barium swallow) is the classic radiographic study employed to assess motility and lumen integrity and to detect the presence of ulceration, diverticula, narrowing, hernia, and extrinsic masses. A modified barium swallow utilizes a cookie, pellet, or marshmallow to assess motility and milder forms of stricture. The mucosal relief technique involves x-ray filming with the esophagus coated and collapsed and is used ideally for identifying small plaquelike or polypoid lesions, esophagitis, and varices. An air-contrast barium study involves distention of a coated esophagus with air and identifies small lesions and subtle mucosal irregularities. Videorecording during a barium swallow enhances the evaluation of both oral and pharyngeal swallowing phases. Computed tomography (CT) and magnetic resonance imaging (MRI) are reserved for detecting suspected neoplasms or locating causes of extrinsic compression.

Manometry is preferred in the evaluation of motility disorders; in this test, the pressure wave is transduced to show duration, amplitude, and velocity of the peristaltic wave. Manofluorographic analysis combines manometry with videostroboscopy. This test is particularly useful for evaluating postsurgical changes. An acid perfusion (Bernstein) test is an excellent means of diagnosing gastroesophageal reflux if pain can be reproduced during acid administration. Reflux can also be diagnosed during pH monitoring when pH is lowered to 4.1 or less at a site 5 cm above the LES. Use of endoscopy is often indicated in the diagnostic workup of patients with dysphagia. Advantages of rigid esophagoscopy include the following: the pharynx and UES are better visualized; large biopsies are made possible; foreign bodies are more easily removed; and the adjunctive use of laser can be accommodated. Advantages of the flexible technique include (1) only sedation and local anesthesia are required, (2) the stomach and upper small intestine can be inspected, (3) videotaping is facilitated, and (4) flexibility is increased when maneuvering in anatomically difficult areas.

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) is a common disorder that accounts for or contributes to the vast majority of esophageal diseases. The most common symptoms of GERD include heartburn and epigastric or retrosternal pain. The pain itself may radiate to the arms, hands, back, neck, or ear; thus, a

cardiac origin must always be considered. Other typical symptoms include regurgitation of gastric contents, dysphagia, aspiration during sleep, and nausea. Patients usually complain of experiencing the greatest discomfort after meals (especially those consisting of fatty or spicy foods) and when lying supine.

Incompetence of the LES is the most significant cause of GERD. Other contributors include slow esophageal clearance, high proportion of acid and pepsin in the refluxed material, high volume of refluxed material, and delayed gastric emptying. Poor nutrition and weakened resistance of the esophageal mucosa to the effects of gastric acid play a role in the development of GERD. Diagnosis is most often made clinically. A barium swallow is useful in excluding either a stricture or hiatal hernia. Approximately 90% of those afflicted with GERD have an associated hernia. Esophagoscopy reveals esophagitis in nearly half of those diagnosed with GERD. Some patients can be diagnosed using the pH test, the Bernstein test, or through esophageal biopsy.

Three phases of treatment are instigated in antireflux therapy. The first phase consists of lifestyle and dietary changes and the use of over-the-counter antacids. Patients are advised to stop smoking, elevate the head area of the bed, lose weight, and avoid wearing tight-fitting clothing. Alcohol, fatty foods, caffeine, chocolate, mints, and carbonated beverages should be avoided. Patients must not overeat, nor should they eat just prior to bedtime.

The second phase of treatment consists of H_2-receptor blocking agents, prokinetic drugs, cytoprotective agents, and proton pump inhibitors. Ranitidine hydrochloride, cimetidine hydrochloride, and famotidine are H_2-blockers that decrease acid secretion. Approximately half of all patients with GERD have disturbances in gastroesophageal motility; pharmacologic agents such as metoclopramide, bethanechol chloride, domperidone, and cisapride are beneficial to these patients in promoting faster gastric emptying. These drugs, in their respective order, act as a central and peripheral dopamine antagonist, cholinergic agonist, peripheral dopamine antagonist, or by enhancing the release of acetylcholine. Sucralfate (Carafate) appears to be most beneficial in the treatment of ulcers and reflux esophagitis. Proton pump inhibition with omeprazole has also been shown to be effective in treating severe esophagitis. Prostaglandins have also been tried in the second phase of therapy as well.

Antireflux surgical procedures constitute the third phase. Surgery generally is reserved for those patients for whom the first and second phases of treatment proved ineffective. The usual indications for surgery include many of the complications of reflux, such as severe esophagitis, ulcer, stricture, bleeding, large hiatal hernia, and problematic respiratory conditions (for example, aspiration, asthma, and bronchitis).

Achalasia

Achalasia, or megaesophagus, is a neuromuscular disorder in which occurs degeneration of the ganglion cells of Auerbach's plexus (plexus myentericus). The pathophysiology is unclear, and hallmarks of this condition include esophageal dilatation, aperistalsis, and failure of the LES to relax. Symptoms include slowly progressive, intermittent dysphagia, pain, and regurgitation with associated respiratory problems including chronic cough, asthma, and pneumonitis. Later in the condition's progression, bleeding, anemia, weight loss, and a psychological aversion to food can occur. Esophagitis resulting from chronic food retention is a common symptom.

Diagnosis of achalasia primarily is made through radiography. Classic signs and symptoms observed at the time of diagnosis include gross esophageal distention, an abnormal air-fluid level, aperistalsis, and inability of the LES to relax. Manometry can be used to confirm the diagnosis. Esophagoscopy (performed after decompression of the contents of esophageal retention) is recommended to evaluate the extent of esophagitis and to dismiss the possibility of malignancy. Treatment is initiated with the patient's taking

more frequent and smaller meals as well as liquids if tolerated. Administration of a calcium-channel blocker (ie, nifedipine) or a longer-acting nitrate (ie, isosorbide mononitrate) can be beneficial, though long-term efficacy of the latter is not proved. Esophageal dilatation is often required in the early stages of treatment; use of Hurst or Maloney dilators is successful in nearly 85% of patients, with a 3% perforation rate. Transthoracic or transabdominal surgical intervention is required for those patients in whom dilatation fails or who can not undergo dilatation because of such contraindications such as a history of previous perforation, aortic aneurysm, or epiphrenic diverticulum. Improvement is achieved in approximately 80% of those who undergo a Heller or modified Heller myotomy, procedures in which incisions are confined to the region of the LES.

Diffuse Esophageal Spasm

Diffuse esophageal spasm (DES) is a disorder characterized by repetitive, synchronous muscular contractions. In contrast to achalasia, there is normal relaxation of the LES. Primary symptoms include retrosternal or epigastric pain, as well as dysphagia when ingesting both solids and liquids. The dysphagia can worsen during times of stress. This condition often is found incidentally in patients in whom esophageal symptoms are absent. Barium swallow reveals the classic corkscrew pattern in the esophageal mucosa. Endoscopy and manometry are used to confirm the diagnosis and exclude other possible abnormalities. Treatment primarily is a pharmacologic one consisting of calcium-channel blockers, nitroglycerin, and diazepam. A transthoracic myotomy and/or reflux procedure can be performed for treatment of intractable pain and dysphagia.

Esophageal Webs and Rings

Esophageal webs and rings are thin, fragile, diaphragmatic structures that interrupt the esophageal lumen and often are found incidentally.[1,2] Webs consist of mucosa and submucosa, whereas rings are thicker and contain a muscular layer as well. Differential diagnoses include peptic stricture, annular neoplasm, abnormal muscular contractions, and inflammatory stenosis. The large variety of webs and rings normally are divided into four categories: cervical webs, midesophageal and lower esophageal webs, esophageal rings, and muscular rings.

Cervical esophageal webs typically occur in the anterior postcricoid area. They can be missed easily during radiography (routine barium swallow) if no special attention is given to that area. A study by Ekberg[3] in 1981 noted the existence of this type of web in nearly 15% of dysphagic patients. Women are more frequently affected; those with anemia and glossitis are said to suffer from Paterson-Kelly or Plummer-Vinson syndrome. Other associated disorders include thyroid disease, esophageal diverticula (especially Zenker's), bullous dermatoses, and an increased incidence of carcinoma occurring in the postcricoid and upper esophageal areas.

Most patients who have cervical webs are asymptomatic or experience varying degrees of intermittent dysphagia with ingestion of solid foods. Some may experience aspiration or complain of pallor, brittle nails, or other symptoms consistent with anemia. Cineradiography is essential in the diagnosis of this type of web, which can be difficult to visualize by endoscopy.[2] The web is often ruptured inadvertently upon the introduction of instrumentation. Treatment normally involves deliberate endoscopic rupture and possibly esophageal bougienage, as well as iron replacement if indicated. These measures prove effective for most patients.

Midesophageal and lower esophageal webs are uncommon and may occur in multiple forms. They are most often caused by an underlying disorder such as chronic graft versus host disease arising after transplantation, long-standing inflammatory conditions, or the bullous dermatologic conditions mentioned earlier. Most patients complain of dysphagia and occasionally of odynophagia. Reflux and its associated symptoms generally are not present with this type of web. Diagnosis and treatment measures are similar to those used to address cervical webs. Studies have shown that perforation of mid- and lower esophageal webs during endoscopy is an unusually common occurrence.[1]

Schatzki's ring is the most common of the lower esophageal rings. These rings occur at the esophageal squamocolumnar junction in older individuals. Most patients are asymptomatic; however, this type of ring is found to occur in up to 14% of patients undergoing routine barium swallows.[1,2] The pathogenesis of esophageal rings is unclear, although most affected individuals also have severe GERD. Ring caliber is an important factor: Schatzki[4] in 1963 showed that symptoms were nearly always evident when ring diameter measured less than 13 mm. Symptomatic patients experience intermittent solid-food dysphagia, reflux, and occasionally acute bolus obstruction, also known as "steakhouse syndrome."[2] A modified barium swallow is effective in revealing this type of ring. Endoscopy may be helpful, although the ring's existence may be missed unless the lower esophagus is distended with air.

No treatment is indicated for asymptomatic patients; however, they should be encouraged to eat slowly and chew carefully. In 1984, Eastridge et al[5] showed that a single, large mercury-filled bougie was effective in relieving patients' discomfort. Only 9 of 65 patients required repeat dilatation. For those who fail to improve, pneumatic dilatation and/or laser excision has been attempted with varying degrees of success. Reflux must be treated if present.

The existence of muscular esophageal rings is controversial: though rare, they are seen most often in children with narrowed, hypertrophic muscular tissue in the esophageal body. This type of ring usually is located a few centimeters proximal to the squamocolumnar junction. On barium swallow, it appears as a broad area of constriction, the variable character of which alters with peristalsis. Therapy consists of dilatation and surgery, although experience with this type of ring is limited.

Esophageal Diverticula

An esophageal diverticulum is an outpouching of the esophagus wall from the lumen that may contain all or none of the muscular coats. Diverticula may be classified on the basis of pulsion versus traction, congenital versus acquired, and true versus false, in addition to anatomic location. The most common are pulsion diverticula, which often coexist with other disorders such as DES, in which pressures are elevated. Traction diverticula are pulled outward by an inflammatory process adjacent to the esophagus and are usually found near tracheobronchial lymph nodes. Most diverticula are described histologically as being "false" in that they lack a muscular coat. A "true" diverticulum, in its pouch wall, entails all of the muscle coats. The four standard anatomic classifications of diverticula are hypopharyngeal or pharyngoesophageal (Zenker's) diverticula, midesophageal or midthoracic diverticula, epiphrenic diverticula, and intramural pseudodiverticula.

Protrusion of hypopharyngeal mucosa between the oblique fibers of the inferior constrictor and the transverse fibers of the cricopharyngeus muscle is known as Zenker's diverticulum. The region of muscular weakness from which the diverticulum protrudes is called Killian's dehiscence or triangle (Figure 1, p 9). Ludlow[6] is credited with the first description of a hypopharyngeal diverticulum in 1769. Zenker, a German pathologist, and von Ziemssen accurately described, in 1874,[7] the pertinent anatomy associated with this type of diverticulum. Pathogenesis is a matter of debate, but generally is thought to be a lack of

Inferior
constrictor
muscle

Superior
constrictor
muscle

Killian's triangle

Cricopharyngeus
muscle

Upper esophageal
sphincter

Proximal esophageal
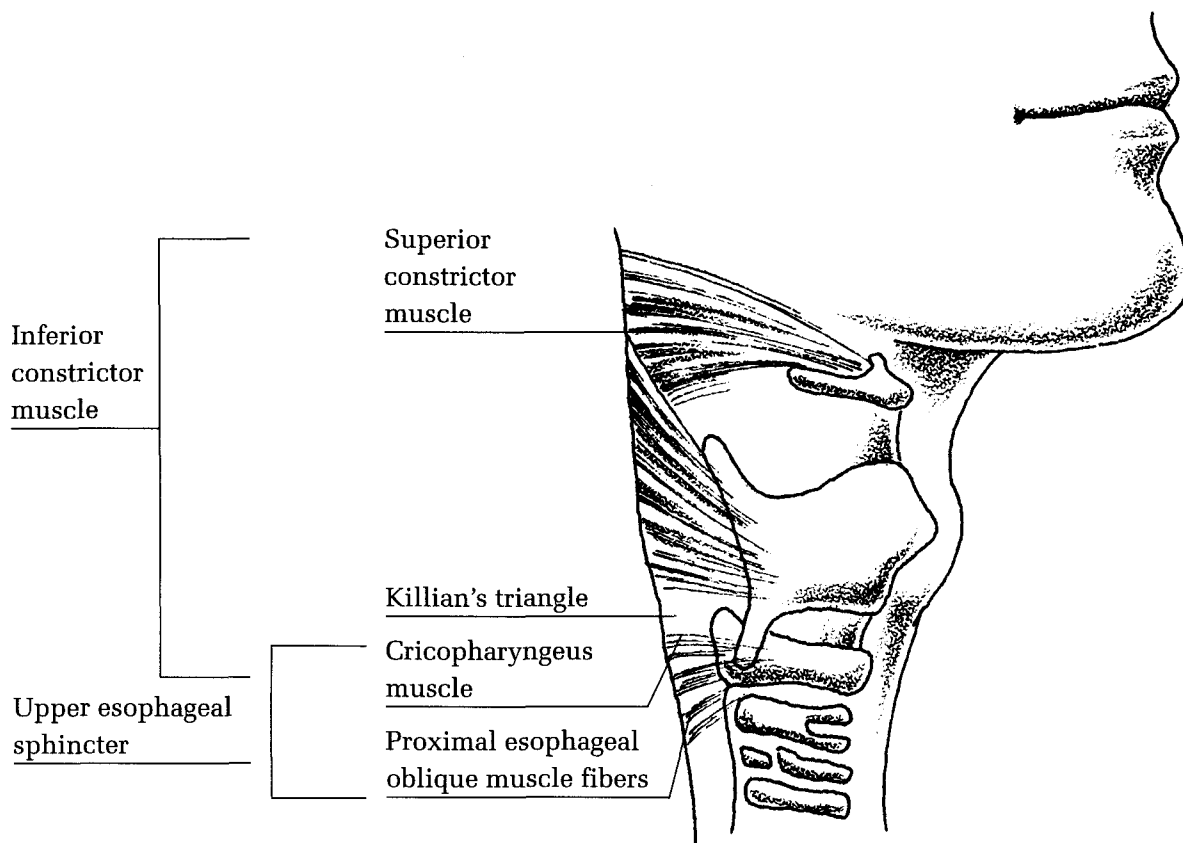oblique muscle fibers

Figure 1. Musculature of the hypopharynx and esophagus.

coordination between pharyngeal contraction and UES relaxation. Causes of UES dysfunction include tonic spasm or failure of relaxation (as occurs in achalasia, for example). Increased intraluminal pressure contributes to formation of this pulsion diverticula. Zenker's is the most common of the esophageal diverticula and accounts for approximately 2% of patients diagnosed with dysphagia.[2] Nearly all cases are found to occur in men older than 50.

Zenker's diverticulum often is found incidentally, but dysphagia and regurgitation are the most common complaints of symptomatic patients. Excessive salivation, expectoration, throat discomfort, and chronic cough are evident in early stages. Later symptoms include progressive regurgitation, throat gurgling, chronic halitosis, pulmonary aspiration, retrosternal pain, and an enlarging anterior neck mass. Such enlargement can cause the esophageal lumen to become compressed, resulting in obstruction and weight loss. Many patients are forced to learn new swallowing maneuvers. Less than 10% of those afflicted with Zenker's diverticula also suffer from significant pulmonary complications, although many experience bronchitis or mild bronchiectasis. Other complications include loss of drug bioavailability (if medications can not be absorbed), ulcer, fistula, and development of a carcinoma. A study reported in 1987[8] found 38 patients having the dual diagnoses of Zenker's diverticulum and carcinoma; a common factor among them was long-standing existence of symptoms. Nearly half of all Zenker's diverticula occur in conjunction with a cervical web.

Physical examination can occasionally reveal such signs as gurgling or anterior neck crepitus in association with a mass; however, barium swallow is the most valuable diagnostic tool. Endoscopy can be
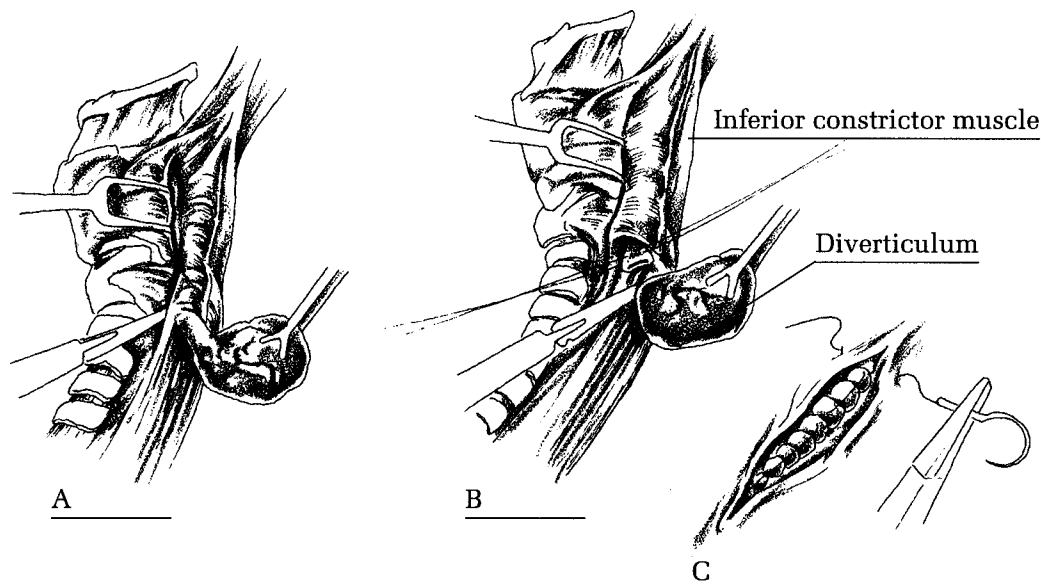
Figure 2. Diverticulectomy. A, Cricopharyngeal myotomy completed (with dilator). B, Traction sutures placed and diverticulum excised. C, Two-layer closure—mucosa and submucosa.

hazardous because of the risk of perforation. Manometry is useful in the diagnostic process in that it enables exclusion of motility disorders. Therapy for small, asymptomatic diverticula consists only of observation. Patients are advised to eat slowly, with careful, meticulous chewing.

Numerous surgical options exist for those who have severe, persistent symptoms. The five surgical methods of correction are diverticulectomy, cricopharyngeal myotomy, diverticulopexy, the Dohlman endoscopic procedure, and imbrication. Even today, much controversy surrounds the preferred surgical procedure.

Excision of the diverticulum is referred to as **diverticulectomy** (Figure 2). Butlin[9] is credited with the first successful diverticulectomy in 1903. Goldman[10] popularized the two-stage procedure in which the diverticular sac is immobilized in the first stage and excised 10 days later: This method was considered the treatment of choice until the early 1960s, when the one-stage procedure was found to have a reduced mortality (0.8%) and morbidity (4.8%) associated with it. Recurrence after diverticulectomy alone has been identified through radiography in nearly 85% of patients; however, only 7% of those are clinically significant. Packing the diverticular sac endoscopically prior to transcervical excision is beneficial to patients, as concluded by authors of numerous studies.

Seifert[11] performed the first successful **cricopharyngeal myotomy** in 1932. The rationale for performing this procedure arose from the theoretical role of UES dysfunction in pathogenesis. Following myotomy of the pharyngoesophageal junction, closure of the muscularis can be done in a longitudinal or transverse direction. According to a few current authors, myotomy alone is preferred, having minimal associated morbidity when treating a small diverticulum. This procedure can be combined with a one-stage diverticulectomy, diverticulopexy, or imbrication procedure.

Diverticulopexy, in combination with cricopharyngeal myotomy, was introduced by Schmid[12] in 1912 and is recommended by other authors (Figure 3, p 11). The sac is dissected free and its fundus is sus-

Myotomy completed
onto upper esophagus

Diverticulum sutured
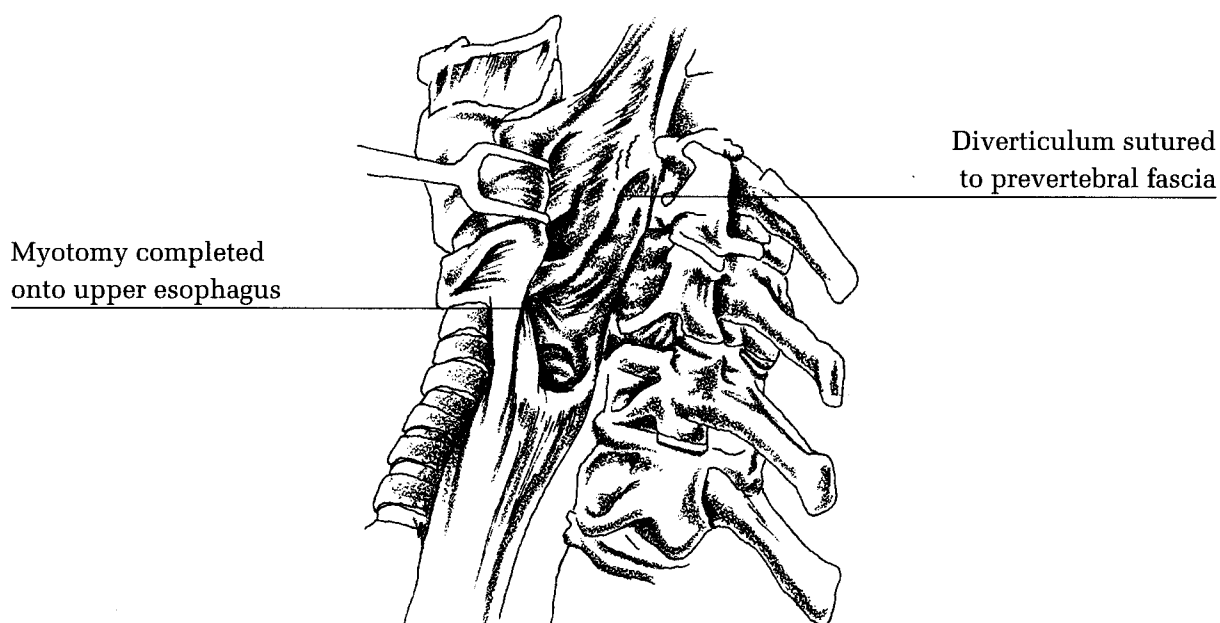to prevertebral fascia

Figure 3. Diverticulopexy in combination with cricopharyngeal myotomy.

pended and affixed to prevertebral fascia superiorly to permit dependent drainage. Many have reported patients' having a shorter hospital stay and sooner oral intake compared with those who undergo diverticulectomy. Diverticulopexy is the preferred procedure for patients who are not good candidates for excision of the sac. Furthermore, it can be performed under local anesthesia. The risks of pharyngoesophageal leakage or esophageal stricture are, at least theoretically, reduced when suspension rather than excision of the sac is performed. However, no retrospective study exists that can document a clear advantage of myotomy and suspension over diverticulectomy with or without myotomy.

Endoscopic division of the esophagodiverticular wall is known as the **Dohlman procedure**. After being described initially by Mosher[13] in 1917, it was popularized in the early 1960s by Dohlman and Mattsson.[14] The wall itself is divided intraluminally by electrocautery using a special slotted, double-lipped laryngoscope. The cricopharyngeus, which is contained in the esophagodiverticular wall, is transected. A communication is created between the diverticulum and the esophagus. Laser transection has been attempted, but this technique has not proved to be superior to the diathermy/cautery approach. Recent studies[15] have shown improvement of symptoms in approximately 90% to 95% of those patients undergoing endoscopic diverticulotomy performed by experienced surgeons. Mediastinitis is an ever-present risk in this procedure, having a 5% occurrence rate.

Morton and Giles[16] provided the first detailed description of **inversion** (instead of excision) of a Zenker's diverticulum in 1986 (Figure 4, p 12). The technique involves initial endoscopic packing of the sac and placement of a cuffed endotracheal tube into the esophageal lumen with the cuff inflated. The pouch is carefully dissected free through an external approach. A cricopharyngeal myotomy is performed prior to inversion of the sac, and the defect is closed using purse-string sutures. Inversion has proved to be a successful technique: in 1992, Johnson and Weissman[17] reported that, of six patients who had undergone inversion with myotomy, all were found to be symptom-free. Morton and Bartley[18] proved a
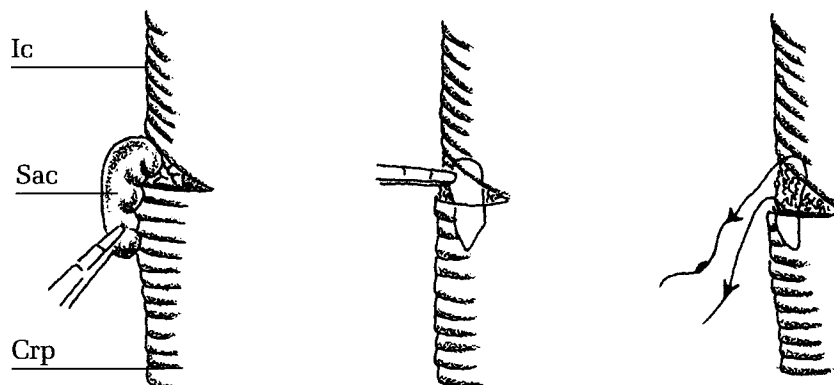
Figure 4. Inversion of a Zenker's diverticulum. A, The diverticulum is located between the inferior constrictor muscle and the cricopharyngeus muscle. B, The sac is inverted or imbricated into the hypopharyngeal lumen. C, Surrounding edges are then closed in a single layer.

shorter operative time, decreased length of hospital stay, and fewer complications in 18 patients treated with inversion, when compared with the same number of patients treated with myotomy and/or diverticulectomy. Despite the relatively short follow-up period of the study, the authors concluded that inversion combined with myotomy was an excellent choice for treating diverticula of limited duration. In instances of long-standing diverticula, the possibility of the development of carcinoma is enhanced; thus, excision may be the most prudent of treatment methods.

The most common complications of surgical treatment of a Zenker's diverticulum include wound infection, hematoma, and pharyngocutaneous fistulization. Other problems arising postoperatively may include recurrent laryngeal nerve injury, stricture, hemothorax, and recurrence of a diverticulum. The Dohlman procedure frequently results in mediastinitis, as noted earlier.

A true diverticular outpouching in the middle one-third of the esophagus is known as a **midesophageal** or **midthoracic diverticulum**. These were first described by Mondiere[19] in 1833. Zenker and von Ziemssen[7] showed that almost all were located adjacent to fibrous tissue in conjunction with long-standing lymphadenopathy. The vast majority of these diverticula are thought to be the traction type resulting from a chronic inflammatory process in the peribronchial tree, particularly at the tracheal bifurcation. Some can form by pulsion secondary to a motility disorder. Midesophageal diverticula are relatively rare and account for only 20% of esophageal diverticula. Although most of these are asymptomatic, chest pain and dysphagia may exist. Complications of an untreated midesophageal diverticulum include rupture with subsequent fistula formation, hematemesis, vocal cord paresis, and coexistence of carcinoma. Diagnosis is obtained by barium swallow, endoscopy, and manometry. Often, no treatment is required; however, any underlying motility disorder or complication should be dealt with, and thoracotomy with diverticulectomy eventually may be required.

An **epiphrenic diverticulum** is an uncommon pulsion outpouching from the distal esophagus. The phenomenon was first identified on autopsy in 1804 and was not understood until characterized by Vinson in 1934.[20] Normally, diverticula of this type are found proximal to a mechanical or functional obstruction. Disorders frequently associated with epiphrenic diverticula include DES, achalasia, hiatal hernia with reflux, and neoplasm.[2] Approximately 80% are asymptomatic, although some patients
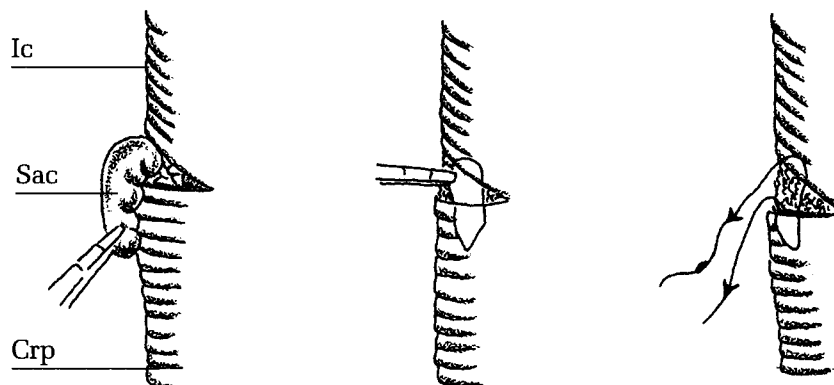
Figure 4. Inversion of a Zenker's diverticulum. A, The diverticulum is located between the inferior constrictor muscle and the cricopharyngeus muscle. B, The sac is inverted or imbricated into the hypopharyngeal lumen. C, Surrounding edges are then closed in a single layer.

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experience dysphagia, regurgitation, chest pain, or obstruction. Complications of untreated epiphrenic diverticula include esophagitis, bezoar formation, carcinoma, perforation, hematemesis, and aspiration. Barium swallow and manometry are useful diagnostic tools, and endoscopy must be performed with caution because of perforation risk. Diverticulectomy or diverticulopexy, regardless of whether any underlying disorder is treated, is the form of therapy generally recommended. Fortunately, few patients progress to the point of needing surgery; the majority simply require observation.

Esophageal intramural pseudodiverticulosis is a condition characterized by multiple, small flask-shaped outpouchings. This disorder is quite rare, with only 70 cases reported through 1988.[1] The pseudodiverticula actually are dilated excretory ducts of deep mucous glands, and their development is associated with increased intraluminal pressure and chronic inflammation. Dysphagia experienced when ingesting solids is the most common symptom. Barium esophagography, CT scan, and endoscopy assist in diagnosing this condition. Treatment usually is limited to observation, although any underlying problem such as stricture or esophagitis should be addressed.

Conclusions

Dysphagia is a common symptom indicating an abnormality in one of the three swallowing phases. Obtaining a detailed history from the patient that elicits accompanying signs and symptoms is crucial in directing the diagnostic workup. Barium swallow (and variations in this radiographic technique), endoscopy, as well as manometry are the diagnostic tools most frequently used. Such diagnostic tests as CT, MRI, pH studies, and laboratory studies are useful adjuncts in some cases. The existence of GERD or a gastroesophageal motility disorder must not be overlooked when evaluating the possible causes of dysphagia, and these disorders may require particular forms of therapy. Esophageal webs often are found incidentally during evaluation of one of the many conditions associated with dysphagia. Endoscopy is generally useful in diagnosis and treatment of both webs and rings. When performing endoscopy to diagnose esophageal diverticula, care must be taken to avoid diverticular perforation. Surgical management of a Zenker's diverticulum remains controversial and long-term prospective studies are needed. Inversion is gaining attention as an exciting new option in the treatment of Zenker's diverticulum.

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