

The role of endoscopy in the evaluation and management of dysphagia

This is one of a series of statements discussing the use of GI endoscopy in common clinical situations. The Standards of Practice Committee of the American Society for Gastrointestinal Endoscopy (ASGE) prepared this update of a previous ASGE guideline.¹ In preparing this guideline, a search of the medical literature was performed by using PubMed for the period 1990-2013. Additional references were obtained from the bibliographies of the identified articles and from recommendations of expert consultants. When few or no data exist from well-designed prospective trials, emphasis is given to results from large series and reports from recognized experts. Guidelines for appropriate use of endoscopy are based on a critical review of the available data and expert consensus at the time that the guidelines are drafted. Further controlled clinical studies may be needed to clarify aspects of this guideline. This guideline may be revised as necessary to account for changes in technology, new data, or other aspects of clinical practice. The recommendations are based on reviewed studies and are graded on the strength of the supporting evidence (Table 1).² The strength of individual recommendations is based on both the aggregate evidence quality and an assessment of the anticipated benefits and harms. Weaker recommendations are indicated by phrases such as “We suggest...” whereas stronger recommendations are typically stated as “We recommend...”

This guideline is intended to be an educational device to provide information that may assist endoscopists in providing care to patients. This guideline is not a rule and should not be construed as establishing a legal standard of care or as encouraging, advocating, requiring, or discouraging any particular treatment. Clinical decisions in any particular case involve a complex analysis of the patient's condition and available courses of action. Therefore, clinical considerations may lead an endoscopist to take a course of action that varies from these guidelines.

ETIOLOGIES OF ESOPHAGEAL DYSPHAGIA

Dysphagia may result from structural or neuromuscular disorders of the esophagus. Patients with structural disorders of the esophagus typically have dysphagia with solids

alone, in contrast to patients with motility disorders who present with both liquid and solid food dysphagia.³ Structural disorders include inflammatory and malignant conditions. Benign inflammatory strictures result from collagen and fibrous tissue deposition in patients with severe or chronic inflammation in the esophagus,⁴ whereas malignant strictures result from intrinsic luminal tumor growth or extrinsic esophageal compression.

The most common causes of esophageal dysphagia are listed in Table 2. Peptic strictures, a sequela of GERD, have been reported to account for up to 80% of all benign esophageal strictures.⁵ However, their incidence appears to have decreased in the last decade because of the widespread use of proton pump inhibitors. With the reported increase in its prevalence, eosinophilic esophagitis (EoE) is now recognized as a common benign cause of dysphagia.⁶ Motility disorders that cause dysphagia include achalasia, diffuse esophageal spasm, and hypomotility secondary to scleroderma and other connective tissue disorders.

THE ROLE OF ENDOSCOPY IN THE EVALUATION OF DYSPHAGIA

Endoscopy is indicated in patients with dysphagia to determine the underlying etiology, exclude malignant and premalignant conditions, assess the need for therapy, and perform therapy, such as dilation. Esophageal dilation is a therapeutic procedure performed for the management of dysphagia. The primary indication for dilation is to provide immediate and durable symptomatic relief of dysphagia. Most of the data on esophageal dilation is compiled from the adult population, but its safety and efficacy also have been confirmed in the pediatric population.^{7,8} In contrast to mechanical stenoses, motility disorders may not respond to dilation, with achalasia being the notable exception.

EGD is an effective tool for the diagnostic evaluation and management of patients with dysphagia. One study reported a diagnostic yield of 54% with EGD in the initial evaluation of patients aged >40 years, who presented with dysphagia and concomitant heartburn, odynophagia, and weight loss.⁹ A cost analysis also showed that EGD with therapeutic intent is more cost effective than an initial diagnostic approach with barium swallow in patients with histories suggestive of benign esophageal obstruction.¹⁰

TABLE 1. GRADE system for rating the quality of evidence for guidelines²

Quality of evidence	Definition	Symbol
High quality	Further research is very unlikely to change our confidence in the estimate of effect.	⊕⊕⊕⊕
Moderate quality	Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.	⊕⊕⊕○
Low quality	Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.	⊕⊕○○
Very low quality	Any estimate of effect is very uncertain.	⊕○○○

During endoscopic evaluation of an esophageal stricture, biopsy specimens should be obtained when a malignancy is suspected on the basis of clinical presentation or endoscopic findings. Biopsies should be obtained from the proximal and distal esophagus to evaluate for EoE in patients with dysphagia and endoscopic findings suggestive of the disorder as well as in the absence of typical endoscopic findings of EoE in patients without esophageal mechanical obstruction.^{11,12} Mucosal biopsies performed in conjunction with dilation do not appear to confer any additional risk for perforation.¹³ Retroflexion of the endoscope before dilation, when possible, to evaluate for malignancy or varices in the gastric cardia, is another important part of the examination and is considered to be one of the quality indicators for EGD.¹⁴

Adults are usually able to tolerate a modified diet at an esophageal luminal diameter of 15 mm and a regular diet at an esophageal luminal diameter of 18 mm.^{15,16} An esophageal luminal diameter of ≤ 13 mm results in dysphagia. Esophageal strictures can be classified as simple or complex, based on their diameter and associated anatomic abnormalities. A simple stricture is defined as a short stricture with a symmetric or concentric lumen and a diameter of ≥ 12 mm that can be traversed easily with an endoscope. A complex stricture is usually longer than 2 cm, may be angulated or irregular, and has a diameter of < 12 mm. It may be associated with a large hiatal hernia, esophageal diverticula, or tracheoesophageal fistula.³ Complex strictures have a higher rate of recurrence and an increased risk for dilation-related adverse events, compared with simple strictures.^{17,18} The severity of a stricture can be estimated by the resistance encountered with passage of the diagnostic endoscope, which has a typical external diameter of 9 mm. A mild stricture allows passage of the endoscope without resistance, a moderate stricture offers increased resistance, whereas a severe stricture may not be traversable.¹⁹ However, this estimation is limited by the subjective perception of the endoscopist. The diameter of a stricture can be objectively measured on barium radiography or by determining the maximal sized barium tablet that can pass through the lumen.¹⁶

Although some endoscopists have advocated the role of large-bore (50F) dilators in patients with dysphagia and normal endoscopic findings,²⁰ several studies have failed to demonstrate improvement in dysphagia scores with this approach.²¹⁻²³ The risk of perforation with large-bore dilators may outweigh the benefits, especially in patients with undiagnosed EoE.^{11,24}

Patients with dysphagia caused by esophageal cancer or extrinsic compression present a challenge to the endoscopist. Most malignant strictures respond to dilation, but symptomatic relief may be only short term, and additional treatment with stent placement may be necessary in these patients.^{25,26} Dysphagia caused by extrinsic compression of the esophagus responds poorly to esophageal dilation.²⁷ In patients with malignant strictures, dilation facilitates feeding gastrostomy tube placement, palliative management with esophageal stenting, and completion of the endoscopic examination, including staging with EUS.²⁸⁻³⁰

Types of dilators

Esophageal dilators include the weighted push type (Maloney; Medovations, Milwaukee, Wis; Teleflex Medical, Research Triangle Park, NC), polyvinyl wire-guided dilators (Savary-Gilliard; Cook Medical, Winston-Salem, NC, and American ConMed, Utica, NY), and balloon dilators (wire-guided and through-the-scope [TTS]).³¹

Bougie dilators rely on tactile perception to determine the amount of resistance encountered with passage through the esophagus. Maloney dilators range in size from 16F to 60F. They can be passed into the esophagus blindly or under fluoroscopic guidance. Maloney dilators can be used without sedation and may be used for self-dilation by select patients.¹⁸ These dilators should not be used for narrow, complex strictures because of the possibility that they could buckle above the stricture and result in perforation. Polyvinyl dilators (Savary-Gilliard and American) have a more tapered and rigid tip than Maloney dilators and a central hollow core for passage of a guidewire. They also range in size from 16F to 60F. The Savary-Gilliard dilators are marked with a radiopaque band at the level of their maximal

TABLE 2. Common etiologies of esophageal dysphagia

Common etiologies	Amenable to dilation
Benign etiologies	
Peptic stricture	Yes
Schatzki ring	Yes
Esophageal web	Yes
Eosinophilic esophagitis	Yes
Caustic injury	Yes
Anastomotic stricture	Yes
Radiation injury	Yes
Pill-induced stricture	Yes
Post-endoscopic therapy stricture	Yes
Congenital esophageal anomalies (tracheoesophageal fistula)	Yes
Cricopharyngeal bar	Yes
Malignant etiologies	
Esophageal adenocarcinoma	Yes
Esophageal squamous cell carcinoma	Yes
Pseudoachalasia	Yes
Extrinsic compression	No
Motility disorders	
Achalasia	Yes
Diffuse esophageal spasm	No
Hypomotility (secondary to connective tissue disorders)	No

diameter for radiographic visualization during fluoroscopy. American dilators have a shorter tapered tip and are impregnated with barium throughout.

Polyethylene balloon dilators include single-diameter and multiple-size balloon dilators that may be used over a guidewire (over-the-wire [OTW]) or passed through the endoscope (TTS). The multiple-size balloons are designed to expand to 3 diameters and are useful for sequential dilations with a single passage of the dilator. Smaller balloons (up to 20 mm) are used for dilation of strictures, whereas larger balloons (30 mm to 40 mm) are used for pneumatic dilation in patients with achalasia. TTS dilators are used more commonly than OTW dilators because of their safety and advantage of dilation under direct visualization.³²

Large-caliber (0.89 mm) monofilament or coiled wires are predominantly used for esophageal dilation. However, smaller caliber wires may be advantageous in cannulation of severe strictures.³³

Preparation

Esophageal dilation usually is performed in the outpatient setting. Patients are instructed to refrain from intake of solids for 6 hours and clear liquids for 2 hours before the procedure.³⁴ Those who have esophageal stasis because of underlying achalasia, diverticula, or tight strictures may require a prolonged fast or nasogastric tube placement to minimize the risk of aspiration.³⁵ Moderate sedation is used for dilation in the majority of patients, whereas deep sedation or general anesthesia may be required for complex procedures and patients with significant comorbidities.³⁶

The management of patients on antithrombotic agents undergoing endoscopic procedures is discussed in detail in a different ASGE guideline.³⁷ Esophageal dilation is considered a high-risk procedure for bleeding adverse events. In patients who are considered low-risk for thromboembolic events, oral anticoagulation with warfarin should be held for 5 to 7 days before the procedure. Bridging therapy is often recommended for patients who are at high risk for thromboembolic events. Thienopyridines (eg, clopidogrel) usually are held for 7 to 10 days before the procedure. Clinicians may elect to continue aspirin before esophageal dilation, depending on the indication for antiplatelet therapy and individual patient characteristics. In patients who are receiving dual antiplatelet therapy, dilation should be deferred, if possible, until the patient has received the minimum length of therapy recommended by the American College of Cardiology/American Heart Association (AHA) guidelines.^{37,38}

Although esophageal dilation is associated with rates of bacteremia of 12% to 22%, the overall risk of infective endocarditis is extremely low.³⁹⁻⁴¹ Current AHA and ASGE guidelines do not recommend prophylactic antibiotics before dilation solely for the prevention of infective endocarditis.^{42,43}

It is important to confirm that all necessary equipment is available in the endoscopy suite before initiation of the procedure. Standard, pediatric, and ultrathin endoscopes and fluoroscopy should be available when dilation of a complex stricture is anticipated. Additional accessories that may be necessary include biopsy forceps, needle-knife papillotome, and steroids (triamcinolone) for injection. The endoscopist should be supported by assistants who are experienced in monitoring patient comfort and safety throughout the examination and who are familiar with the endoscopic tools and dilators being used. Patients should be closely monitored during and after esophageal dilation to detect adverse events.

Techniques of dilation

Bougie dilators exert both radial and axial forces along the entire length of the stricture. The amount of radial force exerted depends on several factors, including caliber of the dilator relative to the stricture diameter, surface

friction of the dilator, angle of taper, and intrinsic characteristics of the stricture.⁴⁴ Radial force is inversely proportional to the shear force, and a bougie with a shorter taper results in a more effective and safer dilation.¹⁶

Bougie dilation with a Maloney dilator may be performed with the patient in the left lateral or upright position. The shaft of the dilator is held with the thumb and medial 3 fingers of the right hand, which enables better tactile perception compared with a closed-hand grip.⁴⁵ The dilator is slowly passed into the esophagus with clockwise and counterclockwise rotations until the widest diameter is distal to the stricture. The dilator is then withdrawn in a single slow movement.

In the technique of wire-guided bougie dilation, a guidewire is passed through the esophagus so that its tip is positioned in the antrum. This length is approximately 60 cm from the incisors in a patient without prior esophago-gastric surgery. The guidewire can be passed under direct endoscopic visualization or fluoroscopic guidance into the stomach. Pediatric and small-caliber endoscopes are compatible with the guidewires. The dilator is then passed over the guidewire with a single smooth movement until the maximal diameter is beyond the stricture. A slight wire retraction may be necessary to ensure that the guidewire is maintained in place. The dilator is then gradually withdrawn while the position of the guidewire is maintained in a one-to-one exchange method. After passage of the last dilator, both the dilator and guidewire are withdrawn together.

Balloon dilators exert only radial force along the length of the stricture. This circumferential pressure, called hoop stress, is a product of the diameter and pressure within the balloon. The opposing static force of the stricture creates an hourglass waist in the balloon. The dilating force of a balloon dilator is inversely proportional to the diameter of the waist. A larger balloon that exerts a higher radial force requires less pressure for dilation but may be associated with a higher risk for perforation.⁴⁴ The dilating force is also dependent on the surface area of the stricture, with more effective dilation of longer strictures.¹⁶

For TTS balloon dilation, the endoscope is positioned at the proximal end of the stricture. The balloon dilator is advanced through the accessory channel of the endoscope through the stricture. Alternatively, if the stricture allows passage of the endoscope through it, the balloon may be advanced and then the endoscope withdrawn to position the balloon within the stricture. The balloon is inflated and maintained at the inflation pressure under direct visualization for approximately 30 to 60 seconds or until there is a sudden drop in manometric pressure. There are no data on the optimal time the balloon should remain inflated. TTS balloons require a 2.8-mm working channel and are not compatible with most small-caliber and pediatric endoscopes. With OTW balloon dilation, a guidewire is passed into the stomach, and the balloon is advanced under fluoroscopic guidance. The dilator is then centered within

the stricture by visualization of radiopaque markers at the center and ends of the balloon. Care should be taken to maintain the wire in position by applying a slight retraction.

Peptic strictures

Patients with peptic strictures may be treated with Maloney, push-type dilators and balloon dilators with similar efficacy.^{19,46} Patients undergoing dilation of peptic strictures should be treated with acid suppressive therapy to prevent stricture recurrence.⁴⁷⁻⁴⁹

Dilation under fluoroscopic guidance, guidewire assistance, or direct visualization is recommended for complex peptic strictures.¹⁸ The degree of dilation in a session should be based on the severity of the stricture. The "rule of 3" for bougie dilation has been accepted but not formally studied for its safety.⁵⁰⁻⁵² The initial dilator is selected based on the stricture diameter. This is estimated as approximately the same size as, and not more than, 1 mm to 2 mm larger than the lumen of the stricture. Sequential dilation is then performed. After moderate resistance is encountered, typically no greater than 3 consecutive dilators in increments of 1 mm are passed in a single session. The "rule of 3" does not apply to balloon dilation, and inflation of a single, appropriately sized balloon dilator should be done. Incremental dilations of >3 mm may be safe for simple strictures.⁵³⁻⁵⁵

Schatzki ring

Dilation with a single, large (16 mm to 20 mm) dilator leads to rupture of the Schatzki ring, and symptomatic relief in almost all patients.^{56,57} Adjunctive methods that have been used with dilation are electrocautery incision with a needle-knife papillotome^{58,59} and 4-quadrant biopsies of the ring.⁶⁰ Several studies have reported an association between EoE and Schatzki ring; biopsies of the esophagus should be considered if there is a clinical suspicion of EoE.^{61,62} One study that compared 4-quadrant biopsies alone versus bougie dilation reported comparable results with both techniques.⁶³

If a Schatzki ring cannot be distinguished from a peptic stricture, graded stepwise dilation is recommended. A peptic stricture is a smooth, concentric, fixed narrowing most commonly seen in the lower esophagus, which may occur in the presence or absence of esophagitis.⁶⁴ In contrast, a Schatzki ring is a diaphragm-like web that is located at the squamocolumnar junction and usually marks the proximal margin of a hiatal hernia.⁶⁵ This is best detected on a barium swallow because it may disappear with air insufflation at endoscopy. Similar to patients with peptic strictures, patients with Schatzki rings may present with recurrent symptoms and require repeated dilation.^{56,66}

Eosinophilic esophagitis

In adults with suspected EoE, initial evaluation should include esophageal biopsies to confirm the diagnosis, followed by medical management. Both bougie and balloon

dilation have been described in the management of patients with EoE.²⁴ Several case reports and case series have reported both spontaneous as well as endoscopic adverse events of esophageal perforation and Boerhaave syndrome.⁶⁷⁻⁷⁰ However, based on the results of two recent systematic reviews, the overall risk of perforation with esophageal dilation is <1% in patients with EoE.^{71,72} Postprocedural pain and mucosal lacerations are common in this population. Risk factors for dilation-associated adverse events in patients with EoE include younger age, multiple dilations, upper esophageal strictures, and inability to traverse the stricture with the endoscope.⁷³

A novel balloon pull-through technique for assessment and dilation of EoE-related strictures was described recently in a series of 13 patients. A TTS multiple-size balloon is selected based on initial assessment of the esophageal luminal diameter at endoscopy. The balloon is positioned across the gastroesophageal junction and inflated to the smallest diameter. The catheter is grasped with the left hand to assess the tension during pull-through. The endoscope is then slowly withdrawn to the proximal esophagus. If no significant mucosal trauma is noted, the procedure is repeated by using a sequentially larger diameter balloon until adequate dilation is achieved. In this series, mucosal tears occurred in 67% of patients, but there were no perforations.⁷⁴

Clinical efficacy of dilation for EoE has been demonstrated in several studies. A recent review of 12 studies reported improvement in dysphagia in 92% of patients with EoE after dilation.⁷¹ A large, retrospective study of 207 adults with EoE found that dilation with or without medical management with steroids resolved or nearly-resolved dysphagia in up to half of patients, 45% remaining symptom free for ≥ 2 years.⁷⁵ A consensus committee on EoE recommended that dilation be reserved for patients who have a dominant esophageal stricture or ring as well as those who remain symptomatic despite medical therapy. In these patients, dilation should be performed cautiously with small-caliber dilators, followed by slow advancement, not exceeding a maximal diameter of 18 mm.^{6,76} Most studies and the consensus committee have suggested bougie dilation as the preferred method because EoE may involve the entire esophagus, whereas others have recommended TTS balloons for dilation under direct visualization.^{6,71,73}

Postesophagectomy anastomotic strictures

Anastomotic strictures have been reported in 9% to 48% of patients after esophagectomy for esophageal cancer.⁷⁷⁻⁸⁰ The diagnosis is made in patients with dysphagia in whom the standard flexible esophagoscope cannot be passed across the anastomosis.⁸¹ Risk factors for developing an anastomotic stricture include anastomotic leakage, ischemia, a stapled as opposed to a hand-sewn anastomosis, gastric pull-up instead of colonic interposition, and medical

comorbidities of cardiovascular disease and diabetes mellitus.^{77,82,83}

Endoscopy allows evaluation for local recurrence of malignancy as well as the performance of dilation for benign anastomotic strictures. Both bougie and balloon dilation have been used for treatment of anastomotic strictures, with a success rate of up to 93%.^{77,83-85} However, there is a high recurrence rate and patients often require frequent and multiple sessions (median 2-9 per patient) to achieve effective dilation.^{79,83,84,86} Electrocautery needle-knife treatment has been described in the management of anastomotic strictures that are resistant to dilation, but long-term outcomes in large series are unavailable.^{87,88} Although short strictures (<1 cm) respond to a single electrocautery treatment, longer strictures may require multiple sessions.⁸⁷ Tissue remodeling with temporary placement of fully covered self-expandable metal stents has been increasingly applied for the management of these benign, refractory, esophageal strictures.⁸⁹

Post-radiation strictures

Proximal esophageal strictures occur in 2% to 16% of patients after radiation therapy for head and neck or lung cancer.^{90,91} The majority of the radiation-induced strictures are complex, and several sessions of bougie dilation may be necessary for adequate treatment. Adequate relief of dysphagia is reported in up to 84% of patients.⁹²⁻⁹⁴ A combined antegrade-retrograde rendezvous approach has been described in case reports and case series for the management of severe radiation-induced strictures with complete occlusion of the proximal esophagus. In this technique, a standard endoscope (after dilation) or a small-caliber endoscope is passed via an existing gastrostomy tract through the stomach into the esophagus. The proximal side of the closed lumen is visualized by using a rigid or flexible endoscope by a second endoscopist. Both endoscopes are aligned by using fluoroscopy and transillumination. The stricture is dissected from above, and an ERCP guidewire is passed from below to traverse the stricture. Serial Savary-Gilliard dilators are passed over the guidewire until moderate resistance is encountered. A small-caliber nasogastric tube is left in place to maintain patency of the lumen and enable subsequent dilations.^{95,96}

Recurrent or refractory esophageal strictures

A refractory or recurrent stricture has been defined as an anatomic restriction due to cicatricial luminal compromise or fibrosis that results in dysphagia in the absence of endoscopic evidence of inflammation. This may occur as the result of either an inability to successfully dilate the stricture to a diameter of 14 mm over 5 sessions at 2-week intervals (refractory) or as a result of an inability to maintain a satisfactory luminal diameter for 4 weeks once the target diameter of 14 mm has been achieved (recurrent). This does not include patients with inflammatory strictures (which will not resolve successfully until the

inflammation subsides) or those with satisfactory stricture diameters who have dysphagia on the basis of neuromuscular dysfunction.⁹⁷

Despite the use of acid suppressive therapy, up to 40% of patients with peptic strictures have recurrent dysphagia requiring repeat dilations.^{46,55,98,99} The most common causes for recurrence include the presence of complex strictures, untreated acid reflux, and undiagnosed EoE.^{47,100,101} For patients who require repeat dilations, the maximal sized dilator used at the prior dilation may be used as the initial dilator for the subsequent session. There is no reported limit to the number of dilation sessions a patient can undergo.

Steroid injection into refractory, benign strictures immediately before or after dilation has been shown to increase the post-dilation diameter (50F vs 40F for peptic strictures; $P = .027$) (47F vs 42F for radiation strictures; $P = .004$),¹⁰² decrease the need for repeat dilations (13% vs 60%; $P = .02$),¹⁰³ and increase the interval between dilations (167 days vs 23 days; $P < .05$).¹⁰²⁻¹⁰⁴ The mechanism of action is considered to be inhibition of matrix protein genes by the steroids, which leads to a decrease in deposition of collagen and fibrous tissue in the esophagus. The most common steroid used for this purpose is triamcinolone acetonide, 40 mg/mL, 0.2 mL to 0.5 mL aliquots injected into 4 quadrants of the stricture.¹⁰⁵ A few investigators have suggested the use of an ultrasound (US) probe to enable injection into the thickest portion of the stricture,^{102,106} but this is not routinely performed in clinical practice.

Temporary esophageal stent placement is an adjunct to dilation in the management of patients with refractory, benign, esophageal strictures. Because of the high rate of tissue ingrowth, uncovered metal stents have been largely replaced by plastic or fully-covered metal stents for this indication.^{17,107,108} A systematic review of 10 studies with 130 patients reported successful plastic stent placement in 98% of patients with benign strictures. Successful dilation was achieved in 52% of patients. Clinical success was significantly lower for cervical strictures compared with strictures in the remainder of the esophagus (33% vs 54%; $P < .05$). There was a high rate of stent migration in 24% of patients. The rate of major adverse events was 9%, including bleeding, perforation, and 1 death.¹⁰⁹

Fully-covered metal stents and biodegradable stents are not U.S. Food and Drug Administration approved for the management of benign esophageal strictures but have been evaluated for this indication. A study of 25 patients being treated with fully-covered metal stents reported rates of stent migration of 80%, new stricture formation of 48%, and development of esophagobronchial fistulae of 4%.¹¹⁰ In a recent study of 15 patients with benign, esophageal strictures, stents were removed prematurely in 60% of patients because of migration, tissue ingrowth, or pain. Recurrent dysphagia occurred in all patients after stent removal.¹¹¹ A biodegradable stent made of poly-L-lactic acid monofilaments has been studied in a trial of

13 patients. Symptomatic improvement was reported in only 2 patients, and the rate of stent migration was 77%.¹¹² A study by Hirdes et al¹¹³ evaluated the role of single and sequential biodegradable stent placement in the management of 28 patients with benign strictures. In total, 59 stents were placed in these patients. Thirteen patients underwent sequential biodegradable stent placement (median 3, range 2-8) during the study period. After initial stent placement, the median dysphagia-free period was 90 days (range 14-618 days). Clinical success, described as absence of dysphagia for 6 months or longer after stent placement, was achieved in 7 patients (25%), and major adverse events occurred in 8 patients (29%). After placement of a second biodegradable stent, the median dysphagia-free period was 55 days (range 25-700 days), and clinical success was achieved in 15% of patients. After placement of a third stent, the median dysphagia-free period was 106 days (range 90-150 days), but clinical success was not achieved in any of the patients.

Self-bougienage is another option for patients who require multiple and frequent dilations. The initial dilation sessions should be performed under the supervision of a clinician in order to ensure that the patient learns the correct technique. A single Maloney dilator with a diameter of 42F, 45F, or 48F is used. The dilator should be marked at the required depth of insertion, and dilation performed with the patient in the sitting position. The dilator is lubricated with water, and the tapered end is introduced into the oropharynx with the left hand. The end of the dilator is raised above the head by using the right hand, which allows the tungsten to migrate to the tip. The dilator is slowly advanced into the esophagus until the marking is seen at the level of the incisors. The dilator is then slowly withdrawn.¹¹⁴⁻¹¹⁶

Achalasia

Esophageal dilation for achalasia involves forceful disruption of the lower esophageal sphincter. This usually is accomplished with 30 mm to 40 mm diameter pneumatic balloon dilators. Dilation is generally performed over a wire under fluoroscopic guidance,^{117,118} although nonfluoroscopically-guided dilation by using endoscopic visualization alone has been reported.^{119,120} Although short-term relief of dysphagia is favorable, recurrence has been reported in approximately one-third of patients,¹²¹ and long-term resolution of symptoms after the initial response has been reported to be as low as 40% to 50%.^{122,123} One study reported a 3-year success rate of 88%, which was attributed predominantly to the use of larger balloons (35 mm to 40 mm). Pneumatic dilation with 30 mm balloons failed in 42% of patients within 3 months.¹²⁴ The overall risk of perforation with pneumatic dilation is in the range of 3% to 5%.^{118,125,126} The strategy of 30 mm balloon dilation followed by 35 mm dilation may be a safer approach because initial dilation with the 35 mm balloon has a higher perforation rate (31% vs 4%; $P < .001$).¹²⁷

An alternative to dilation in patients with achalasia is the endoscopic injection of botulinum toxin. The symptomatic response to this treatment is often short lived, with greater than 50% recurrence by 6 months.¹²⁸ In randomized studies, pneumatic balloon dilation is more effective than botulinum toxin injection, with significantly higher cumulative remission rates (70%-89% compared with 32%-38%, respectively; $P < .01$).^{128,129} A large, randomized trial of 201 patients compared pneumatic dilation with laparoscopic Heller myotomy (LHM). There was no significant difference in therapeutic success between the 2 groups, 86% with pneumatic dilation and 90% with LHM after 2 years ($P = .46$). Perforation occurred in 4% of patients with pneumatic dilation, whereas mucosal tears occurred in 12% of patients with LHM.¹²⁷

A meta-analysis of 17 studies evaluated various treatment options for achalasia.¹³⁰ Pneumatic dilation demonstrated a better remission and lower relapse rate compared with botulinum toxin for the initial management of patients with achalasia. There was an increase in remission and no differences in adverse event rates with LHM compared with pneumatic dilation. Another recent meta-analysis of 36 studies with 3211 patients reported a mean 5-year remission rate of 61.9% and 10-year remission rate of 47.9% with pneumatic dilation, compared with 76.1% and 79.6%, respectively, with LHM. The perforation rate was 4.8% with LHM and 2.4% with pneumatic dilation ($P < .05$).¹³¹ Cost analysis models indicate that initial pneumatic dilation is a more cost-effective approach compared with botulinum toxin injection or LHM for healthy patients with achalasia.^{132,133} A study of 99 patients with achalasia diagnosed with high-resolution manometry showed that type II patients (achalasia with esophageal compression) are more likely to respond to any therapy (Botox 71%, pneumatic dilation 91%, or Heller myotomy 100%) than type I (achalasia with minimal esophageal pressurization) (56% overall) or type III (achalasia with spasm) (29% overall) patients. Type II achalasia was a predictor of positive treatment response, whereas type III and pretreatment esophageal dilatation were predictors of a negative treatment response.¹³⁴

Before endoscopic treatment, patients with achalasia should be informed of all therapeutic options available. Symptomatic patients with achalasia who are good surgical candidates should be given the option of either graded pneumatic dilation or cardiomyotomy. Open surgical repair with myotomy of early recognized endoscopic perforation offers outcomes similar to those of elective open myotomy. However, LHM may not be technically feasible after an endoscopic perforation.¹³⁵ In comparison, pneumatic dilation can be performed safely in patients after a failed myotomy.^{136,137} The subset of patients in whom the latter approach has failed may require esophagectomy. Botulinum toxin may be the preferred approach in patients who are poor candidates for surgery, as pneumatic dilation is not recommended in these high-risk surgical candidates.

Peroral endoscopic myotomy (POEM) is a new endoscopic procedure that has been used in the treatment of achalasia. The technique involves the creation of a 2-cm long mucosal incision in the esophagus, approximately 14 cm proximal to the lower esophageal sphincter (LES). A submucosal tunnel is then created from the incision to the LES followed by dissection of the circular muscle fibers over the distal 7 cm of the esophagus and proximal 2 cm of the gastric cardia. The mucosal incision is then closed using endoscopic clips.¹³⁸ A study that evaluated the role of POEM in 17 consecutive patients with achalasia reported a significant improvement in dysphagia scores (1.3 vs 10; $P < .0003$) and lower esophageal sphincter pressure (19.8 vs 52.4; $P < .0001$).¹³⁹ The success rate was significantly higher in patients who had a nontortuous esophagus compared to those with a tortuous esophagus. Another study by the same investigators reported treatment success in 94% of patients after peroral endoscopic myotomy (mean dysphagia score 1.4 vs 8.8; $P < .001$ and lower esophageal sphincter pressure 11.8 vs 27.2; $P < .001$).¹⁴⁰ Reflux esophagitis after POEM was reported in one patient in each of these studies. Long-term data and randomized trials comparing this technique to conventional modalities of management are necessary before it can be adopted into clinical practice, but the procedure is becoming more widely used in expert centers.

Dysphagia due to hypopharyngeal causes

Disorders of the upper esophageal sphincter or hypopharynx can cause oropharyngeal dysphagia, typically distinguishable from esophageal dysphagia based on a careful history and diagnostic evaluation of the swallowing mechanism. EGD should be performed in patients suspected of having oropharyngeal dysphagia in order to exclude alternative or additional pathologic conditions and should include a complete examination of the upper esophageal sphincter and hypopharynx. A variety of endoscopy based therapies including dilation and injection therapy have been described for the treatment of upper esophageal sphincter dysfunction and entities such as cricopharyngeal bars.¹⁴¹⁻¹⁴³

Contraindications and adverse events of esophageal dilation

The presence of an esophageal perforation is an absolute contraindication to esophageal dilation. Dilation should be performed with caution in patients who have had a recent, healed perforation or upper GI surgery. The main adverse events associated with dilation are perforation, bleeding, and aspiration. The perforation rate for esophageal strictures after dilation ranges from 0.1% to 0.4%¹⁸ and is higher with complex strictures¹⁸ and radiation-induced strictures.¹⁴⁴ The perforation rate may be influenced by endoscopist experience. One study indicated that the perforation rate was 4 times greater when

the endoscopist had performed fewer than 500 previous diagnostic upper endoscopic examinations.¹⁴⁵

Perforation after esophageal dilation can be intra-abdominal or intrathoracic at the site of the stricture. This adverse event should be suspected if a patient develops severe or persistent chest or abdominal pain, dyspnea, tachycardia, or fevers after dilation. The physical examination may reveal subcutaneous crepitus of the chest or cervical region. Although a chest radiograph may indicate a perforation, a normal study result does not exclude it, and a water-soluble contrast esophagram or contrast-enhanced computed tomogram of the chest may be necessary to confirm this adverse event.¹⁴⁶ The use of large-diameter covered metal stents and expandable, retrievable plastic stents has been effective in the management of perforation after dilation of benign and malignant strictures.^{147,148}

RECOMMENDATIONS

1. We recommend endoscopic dilation for patients with dysphagia secondary to benign intrinsic strictures of the esophagus. (⊕⊕⊕⊕)
2. We recommend wire-guided dilation, preferably under fluoroscopic guidance, or TTS balloon dilation for complex esophageal strictures. (⊕⊕⊕○)
3. We recommend antisecretory treatment in conjunction with dilation to reduce the recurrence rate of peptic strictures. (⊕⊕⊕⊕)
4. We recommend that dilation for adult patients with EoE be reserved for those who have a dominant esophageal stricture or ring and those who remain symptomatic despite medical therapy. (⊕⊕⊕○)
5. We suggest adjunctive treatment with corticosteroid injection into recurrent or refractory benign esophageal peptic strictures. (⊕⊕○○)
6. We suggest that esophageal stent placement be reserved for refractory esophageal strictures that do not respond to sequential dilation and/or steroid injection. (⊕⊕○○)
7. We recommend that both endoscopic and surgical treatment options for achalasia be discussed with the patient. In patients who opt for endoscopic management and are good surgical candidates, we recommend pneumatic dilation with large-caliber balloon dilators for the endoscopic treatment of achalasia. (⊕⊕⊕⊕)
8. We recommend botulinum toxin injection for endoscopic treatment of achalasia in patients who are poor candidates for surgery or pneumatic dilation. (⊕⊕⊕○)

DISCLOSURES

The following authors disclosed financial relationships relevant to this publication: Dr Muthusamy, consultant to Boston Scientific; Dr Khashbab, consultant to, honoraria from, and on the advisory board of Boston Scientific. All

other authors disclosed no financial relationships relevant to this publication.

Abbreviations: AHA, American Heart Association; EoE, eosinophilic esophagitis; LHM, laparoscopic Heller myotomy; OTW, over-the-wire; TTS, through-the-scope.

REFERENCES

1. Egan JV, Baron TH, Adler DG, et al. Esophageal dilation. *Gastrointest Endosc* 2006;63:755-60.
2. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ* 2008;336:924-6.
3. Lew RJ, Kochman ML. A review of endoscopic methods of esophageal dilation. *J Clin Gastroenterol* 2002;35:117-26.
4. Spechler SJ. Complications of gastroesophageal reflux disease. In: Castell DO, editor. *The Esophagus*, 2nd ed. Boston: Little, Brown; 1995. p. 533-44.
5. Richter JE. Peptic strictures of the esophagus. *Gastroenterol Clin North Am* 1999;28:875-91.
6. Liacouras CA, Furuta GT, Hirano I, et al. Eosinophilic esophagitis: updated consensus recommendations for children and adults. *J Allergy Clin Immunol* 2011;128:3-20.
7. Chang CF, Kuo SP, Lin HC, et al. Endoscopic balloon dilatation for esophageal strictures in children younger than 6 years: experience in a medical center. *Pediatr Neonatol* 2011;52:196-202.
8. Lan LC, Wong KK, Lin SC, et al. Endoscopic balloon dilatation of esophageal strictures in infants and children: 17 years' experience and a literature review. *J Pediatr Surg* 2003;38:1712-5.
9. Varadarajulu S, Eloubeidi MA, Patel RS, et al. The yield and the predictors of esophageal pathology when upper endoscopy is used for the initial evaluation of dysphagia. *Gastrointest Endosc* 2005;61:804-8.
10. Esfandyari T, Potter JW, Vaezi MF. Dysphagia: a cost analysis of the diagnostic approach. *Am J Gastroenterol* 2002;97:2733-7.
11. Arora AS. Management strategies for dysphagia with a normal-appearing esophagus. *Clin Gastroenterol Hepatol* 2005;3:299-302.
12. Furuta GT, Liacouras CA, Collins MH, et al. Eosinophilic esophagitis in children and adults: a systematic review and consensus recommendations for diagnosis and treatment. *Gastroenterology* 2007;133:1342-63.
13. Barkin JS, Taub S, Rogers AI. The safety of combined endoscopy, biopsy and dilation in esophageal strictures. *Am J Gastroenterol* 1981;76:23-6.
14. Cohen J, Safdi MA, Deal SE, et al. Quality indicators for esophagogastroduodenoscopy. *Am J Gastroenterol* 2006;101:886-91.
15. Schatzki R, Gary JE. Dysphagia due to a diaphragm-like localized narrowing in the lower esophagus (lower esophageal ring). *Am J Roentgenol Radium Ther Nucl Med* 1953;70:911-22.
16. Dryden GW, McClave SA. Methods of treating dysphagia caused by benign esophageal strictures. *Tech Gastrointest Endosc* 2001;3:135-43.
17. de Wijkerslooth LR, Vleggaar FP, Siersema PD. Endoscopic management of difficult or recurrent esophageal strictures. *Am J Gastroenterol* 2011;106:2080-91.
18. Hernandez LV, Jacobson JW, Harris MS. Comparison among the perforation rates of Maloney, balloon, and Savary dilation of esophageal strictures. *Gastrointest Endosc* 2000;51:460-2.
19. Scolapio JS, Pasha TM, Gostout CJ, et al. A randomized prospective study comparing rigid to balloon dilators for benign esophageal strictures and rings. *Gastrointest Endosc* 1999;50:13-7.
20. Marshall JB, Chowdhury TA. Does empiric esophageal dilation benefit dysphagia when endoscopy is normal? *Dig Dis Sci* 1996;41:1099-101.
21. Colon VJ, Young MA, Ramirez FC. The short- and long-term efficacy of empirical esophageal dilation in patients with nonobstructive

- dysphagia: a prospective, randomized study. *Am J Gastroenterol* 2000;95:910-3.
22. Scolapio JS, Gostout CJ, Schroeder KW, et al. Dysphagia without endoscopically evident disease: to dilate or not? *Am J Gastroenterol* 2001;96:327-30.
 23. Lavu K, Mathew TP, Minocha A. Effectiveness of esophageal dilation in relieving nonobstructive esophageal dysphagia and improving quality of life. *South Med J* 2004;97:137-40.
 24. Cohen MS, Kaufman AB, Palazzo JP, et al. An audit of endoscopic complications in adult eosinophilic esophagitis. *Clin Gastroenterol Hepatol* 2007;5:1149-53.
 25. Tietjen TG, Pasricha PJ, Kalloo AN. Management of malignant esophageal stricture with esophageal dilation and esophageal stents. *Gastrointest Endosc Clin N Am* 1994;4:851-62.
 26. Varadarajulu S, Banerjee S, Barth B, et al. Enteral stents. *Gastrointest Endosc* 2011;74:455-64.
 27. Bethge N, Sommer A, Vakil N. Palliation of malignant esophageal obstruction due to intrinsic and extrinsic lesions with expandable metal stents. *Am J Gastroenterol* 1998;93:1829-32.
 28. Pfau PR, Ginsberg GG, Lew RJ, et al. Esophageal dilation for endosonographic evaluation of malignant esophageal strictures is safe and effective. *Am J Gastroenterol* 2000;95:2813-5.
 29. Adler DG, Baron TH. Endoscopic palliation of malignant dysphagia. *Mayo Clin Proc* 2001;76:731-8.
 30. Adler DG, Baron TH, Geels W, et al. Placement of PEG tubes over previously placed self-expanding esophageal metal stents. *Gastrointest Endosc* 2001;54:237-41.
 31. ASGE Technology Committee. Tools for endoscopic stricture dilation. *Gastrointest Endosc* 2013;78:391-404.
 32. Lindor KD, Ott BJ, Hughes RW Jr. Balloon dilatation of upper digestive tract strictures. *Gastroenterology* 1985;89:545-8.
 33. Somogyi L, Chuttani R, Croffie J, et al. Guidewires for use in GI endoscopy. *Gastrointest Endosc* 2007;65:571-6.
 34. Apfelbaum JL, Caplan RA, Connis RT, et al. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures: an updated report by the American Society of Anesthesiologists Committee on Standards and Practice Parameters. *Anesthesiology* 2011;114:495-511.
 35. DeVita JJ, Reynolds JC. Esophageal dilation techniques (#4 in series). *Practical Gastroenterology* 2002;26:46-57.
 36. Lichtenstein DR, Jagannath S, Baron TH, et al. Sedation and anesthesia in GI endoscopy. *Gastrointest Endosc* 2008;68:815-26.
 37. Anderson MA, Ben-Menachem T, Gan SI, et al. Management of antithrombotic agents for endoscopic procedures. *Gastrointest Endosc* 2009;70:1060-70.
 38. King SB, 3rd, Smith SC, Jr, Hirshfeld JW, Jr, et al. 2007 Focused update of the ACC/AHA/SCAI 2005 guideline update for percutaneous coronary intervention: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2008;51:172-209.
 39. Zuccaro G Jr, Richter JE, Rice TW, et al. Viridans streptococcal bacteremia after esophageal stricture dilation. *Gastrointest Endosc* 1998;48:568-73.
 40. Nelson DB, Sanderson SJ, Azar MM. Bacteremia with esophageal dilation. *Gastrointest Endosc* 1998;48:563-7.
 41. Hirota WK, Wortmann GW, Maydonovitch CL, et al. The effect of oral decontamination with clindamycin palmitate on the incidence of bacteremia after esophageal dilation: a prospective trial. *Gastrointest Endosc* 1999;50:475-9.
 42. Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation* 2007;116:1736-54.
 43. Banerjee S, Shen B, Baron TH, et al. Antibiotic prophylaxis for GI endoscopy. *Gastrointest Endosc* 2008;67:791-8.
 44. Abele JE. The physics of esophageal dilatation. *Hepatogastroenterology* 1992;39:486-9.
 45. Boyce HW. Dilation of difficult benign esophageal strictures. *Am J Gastroenterol* 2005;100:744-5.
 46. Saeed ZA, Winchester CB, Ferro PS, et al. Prospective randomized comparison of polyvinyl bougies and through-the-scope balloons for dilation of peptic strictures of the esophagus. *Gastrointest Endosc* 1995;41:189-95.
 47. Smith PM, Kerr GD, Cockel R, et al. A comparison of omeprazole and ranitidine in the prevention of recurrence of benign esophageal stricture. Restore Investigator Group. *Gastroenterology* 1994;107:1312-8.
 48. Silvis SE, Farahmand M, Johnson JA, et al. A randomized blinded comparison of omeprazole and ranitidine in the treatment of chronic esophageal stricture secondary to acid peptic esophagitis. *Gastrointest Endosc* 1996;43:216-21.
 49. Swarbrick ET, Gough AL, Foster CS, et al. Prevention of recurrence of oesophageal stricture, a comparison of lansoprazole and high-dose ranitidine. *Eur J Gastroenterol Hepatol* 1996;8:431-8.
 50. Tulman AB, Boyce HW, Jr. Complications of esophageal dilation and guidelines for their prevention. *Gastrointest Endosc* 1981;27:229-34.
 51. Langdon DF. The rule of three in esophageal dilation. *Gastrointest Endosc* 1997;45:111.
 52. Nostrant TT. Esophageal dilatation. *Dig Dis* 1995;13:337-55.
 53. Kozarek RA, Patterson DJ, Ball TJ, et al. Esophageal dilation can be done safely using selective fluoroscopy and single dilating sessions. *J Clin Gastroenterol* 1995;20:184-8.
 54. Cox JG, Winter RK, Maslin SC, et al. Balloon or bougie for dilatation of benign oesophageal stricture? An interim report of a randomised controlled trial. *Gut* 1988;29:1741-7.
 55. Siersema PD, de Wijkerslooth LR. Dilation of refractory benign esophageal strictures. *Gastrointest Endosc* 2009;70:1000-12.
 56. Eckardt VF, Kanzler G, Willems D. Single dilation of symptomatic Schatzki rings. A prospective evaluation of its effectiveness. *Dig Dis Sci* 1992;37:577-82.
 57. Jalil S, Castell DO. Schatzki's ring: a benign cause of dysphagia in adults. *J Clin Gastroenterol* 2002;35:295-8.
 58. Burdick JS, Venu RP, Hogan WJ. Cutting the defiant lower esophageal ring. *Gastrointest Endosc* 1993;39:616-9.
 59. Guelrud M, Villamil L, Mendez R. Late results in patients with Schatzki ring treated by endoscopic electrosurgical incision of the ring. *Gastrointest Endosc* 1987;33:96-8.
 60. Som ML, Wolf BS, Marshak RH. Narrow esophagogastric ring treated endoscopically. *Gastroenterology* 1960;39:634-8.
 61. Nurko S, Teitelbaum JE, Husain K, et al. Association of Schatzki ring with eosinophilic esophagitis in children. *J Pediatr Gastroenterol Nutr* 2004;38:436-41.
 62. Desai TK, Stecevic V, Chang CH, et al. Association of eosinophilic inflammation with esophageal food impaction in adults. *Gastrointest Endosc* 2005;61:795-801.
 63. Chotiprasidhi P, Minocha A. Effectiveness of single dilation with Maloney dilator versus endoscopic rupture of Schatzki's ring using biopsy forceps. *Dig Dis Sci* 2000;45:281-4.
 64. Gupta N, Sharma P. Esophageal Diseases. In: Classen M, Tytgat GN, Lightdale CJ, editors. *Gastroenterological endoscopy*, 2nd ed. New York: Thieme; 2010. p. 488-510.
 65. Ott DJ, Gelfand DW, Wu WC, et al. Esophagogastric region and its rings. *AJR Am J Roentgenol* 1984;142:281-7.
 66. Groskreutz JL, Kim CH. Schatzki's ring: long-term results following dilation. *Gastrointest Endosc* 1990;36:479-81.
 67. Straumann A, Bussmann C, Zuber M, et al. Eosinophilic esophagitis: analysis of food impaction and perforation in 251 adolescent and adult patients. *Clin Gastroenterol Hepatol* 2008;6:598-600.
 68. Kaplan M, Mutlu EA, Jakate S, et al. Endoscopy in eosinophilic esophagitis: "feline" esophagus and perforation risk. *Clin Gastroenterol Hepatol* 2003;1:433-7.

69. Robles-Medranda C, Villard F, Bouvier R, et al. Spontaneous esophageal perforation in eosinophilic esophagitis in children. *Endoscopy* 2008;(40 Suppl 2):E171.
70. Prasad GA, Arora AS. Spontaneous perforation in the ringed esophagus. *Dis Esophagus* 2005;18:406-9.
71. Bohm ME, Richter JE. Review article: oesophageal dilation in adults with eosinophilic oesophagitis. *Aliment Pharmacol Ther* 2011;33:748-57.
72. Jacobs JW Jr, Spechler SJ. A systematic review of the risk of perforation during esophageal dilation for patients with eosinophilic esophagitis. *Dig Dis Sci* 2010;55:1512-5.
73. Dellon ES, Gibbs WB, Rubinas TC, et al. Esophageal dilation in eosinophilic esophagitis: safety and predictors of clinical response and complications. *Gastrointest Endosc* 2010;71:706-12.
74. Madanick RD, Shaheen NJ, Dellon ES. A novel balloon pull-through technique for esophageal dilation in eosinophilic esophagitis (with video). *Gastrointest Endosc* 2011;73:138-42.
75. Schoepfer AM, Gonsalves N, Bussmann C, et al. Esophageal dilation in eosinophilic esophagitis: effectiveness, safety, and impact on the underlying inflammation. *Am J Gastroenterol* 2010;105:1062-70.
76. Schoepfer AM, Gschossmann J, Scheurer U, et al. Esophageal strictures in adult eosinophilic esophagitis: dilation is an effective and safe alternative after failure of topical corticosteroids. *Endoscopy* 2008;40:161-4.
77. Briel JW, Tamhankar AP, Hagen JA, et al. Prevalence and risk factors for ischemia, leak, and stricture of esophageal anastomosis: gastric pull-up versus colon interposition. *J Am Coll Surg* 2004;198:536-41.
78. McLarty AJ, Deschamps C, Trastek VF, et al. Esophageal resection for cancer of the esophagus: long-term function and quality of life. *Ann Thorac Surg* 1997;63:1568-72.
79. Law S, Fok M, Chu KM, et al. Comparison of hand-sewn and stapled esophagogastric anastomosis after esophageal resection for cancer: a prospective randomized controlled trial. *Ann Surg* 1997;226:169-73.
80. Heitmiller RF, Fischer A, Liddicoat JR. Cervical esophagogastric anastomosis: results following esophagectomy for carcinoma. *Dis Esophagus* 1999;12:264-9.
81. Rice TW. Anastomotic stricture complicating esophagectomy. *Thorac Surg Clin* 2006;16:63-73.
82. van Heijl M, Gooszen JA, Fockens P, et al. Risk factors for development of benign cervical strictures after esophagectomy. *Ann Surg* 2010;251:1064-9.
83. Marjanovic G, Schrag HJ, Fischer E, et al. Endoscopic bougienage of benign anastomotic strictures in patients after esophageal resection: the effect of the extent of stricture on bougienage results. *Dis Esophagus* 2008;21:551-7.
84. Ikeya T, Ohwada S, Ogawa T, et al. Endoscopic balloon dilation for benign esophageal anastomotic stricture: factors influencing its effectiveness. *Hepatogastroenterology* 1999;46:959-66.
85. Kim HC, Shin JH, Song HY, et al. Fluoroscopically guided balloon dilation for benign anastomotic stricture after Ivor-Lewis esophagectomy: experience in 62 patients. *J Vasc Interv Radiol* 2005;16:1699-704.
86. Honkoop P, Siersema PD, Tilanus HW, et al. Benign anastomotic strictures after transhiatal esophagectomy and cervical esophagogastrotomy: risk factors and management. *J Thorac Cardiovasc Surg* 1996;111:1141-6.
87. Hordijk ML, Siersema PD, Tilanus HW, et al. Electrocautery therapy for refractory anastomotic strictures of the esophagus. *Gastrointest Endosc* 2006;63:157-63.
88. Thorsen G, Rosseland AR. Endoscopic incision of postoperative stenoses in the upper gastrointestinal tract. *Gastrointest Endosc* 1983;29:26-9.
89. Oh YS, Kochman ML, Ahmad NA, et al. Clinical outcomes after self-expanding plastic stent placement for refractory benign esophageal strictures. *Dig Dis Sci* 2010;55:1344-8.
90. Laurell G, Kraepelien T, Mavroidis P, et al. Stricture of the proximal esophagus in head and neck carcinoma patients after radiotherapy. *Cancer* 2003;97:1693-700.
91. Ahn SJ, Kahn D, Zhou S, et al. Dosimetric and clinical predictors for radiation-induced esophageal injury. *Int J Radiat Oncol Biol Phys* 2005;61:335-47.
92. Swaroop VS, Desai DC, Mohandas KM, et al. Dilation of esophageal strictures induced by radiation therapy for cancer of the esophagus. *Gastrointest Endosc* 1994;40:311-5.
93. Dhir V, Vege SS, Mohandas KM, et al. Dilation of proximal esophageal strictures following therapy for head and neck cancer: experience with Savary-Gilliard dilators. *J Surg Oncol* 1996;63:187-90.
94. Ahlawat SK, Al-Kawas FH. Endoscopic management of upper esophageal strictures after treatment of head and neck malignancy. *Gastrointest Endosc* 2008;68:19-24.
95. Maple JT, Petersen BT, Baron TH, et al. Endoscopic management of radiation-induced complete upper esophageal obstruction with an antegrade-retrograde rendezvous technique. *Gastrointest Endosc* 2006;64:822-8.
96. Dellon ES, Cullen NR, Madanick RD, et al. Outcomes of a combined antegrade and retrograde approach for dilatation of radiation-induced esophageal strictures (with video). *Gastrointest Endosc* 2010;71:1122-9.
97. Kochman ML, McClave SA, Boyce HW. The refractory and the recurrent esophageal stricture: a definition. *Gastrointest Endosc* 2005;62:474-5.
98. Patterson DJ, Graham DY, Smith JL, et al. Natural history of benign esophageal stricture treated by dilatation. *Gastroenterology* 1983;85:346-50.
99. Chiu YC, Hsu CC, Chiu KW, et al. Factors influencing clinical applications of endoscopic balloon dilation for benign esophageal strictures. *Endoscopy* 2004;36:595-600.
100. Said A, Brust DJ, Gaumnitz EA, et al. Predictors of early recurrence of benign esophageal strictures. *Am J Gastroenterol* 2003;98:1252-6.
101. Liacouras CA, Markowitz JE. Predictors of early recurrence of benign esophageal strictures: What about eosinophilic esophagitis? *Am J Gastroenterol* 2004;99:182-3.
102. Lee M, Kubik CM, Polhamus CD, et al. Preliminary experience with endoscopic intralesional steroid injection therapy for refractory upper gastrointestinal strictures. *Gastrointest Endosc* 1995;41:598-601.
103. Ramage JI Jr, Rumalla A, Baron TH, et al. A prospective, randomized, double-blind, placebo-controlled trial of endoscopic steroid injection therapy for recalcitrant esophageal peptic strictures. *Am J Gastroenterol* 2005;100:2419-25.
104. Altintas E, Kacar S, Tunc B, et al. Intralesional steroid injection in benign esophageal strictures resistant to bougie dilation. *J Gastroenterol Hepatol* 2004;19:1388-91.
105. Kochhar R, Makharia GK. Usefulness of intralesional triamcinolone in treatment of benign esophageal strictures. *Gastrointest Endosc* 2002;56:829-34.
106. Bhutani MS, Usman N, Shenoy V, et al. Endoscopic ultrasound miniprobe-guided steroid injection for treatment of refractory esophageal strictures. *Endoscopy* 1997;29:757-9.
107. Sharma P, Kozarek R. Role of esophageal stents in benign and malignant diseases. *Am J Gastroenterol* 2010;105:258-73.
108. Baron TH. Management of benign esophageal strictures. *Gastroenterol Hepatol* 2011;7:46-9.
109. Repici A, Hassan C, Sharma P, et al. Systematic review: the role of self-expanding plastic stents for benign oesophageal strictures. *Aliment Pharmacol Ther* 2010;31:1268-75.
110. Song HY, Jung HY, Park SI, et al. Covered retrievable expandable nitinol stents in patients with benign esophageal strictures: initial experience. *Radiology* 2000;217:551-7.
111. Hirdes MM, Siersema PD, Vleggaar FP. A new fully covered metal stent for the treatment of benign and malignant dysphagia: a prospective follow-up study. *Gastrointest Endosc* 2012;75:712-8.
112. Saito Y, Tanaka T, Andoh A, et al. Usefulness of biodegradable stents constructed of poly-L-lactic acid monofilaments in patients with benign esophageal stenosis. *World J Gastroenterol* 2007;13:3977-80.
113. Hirdes MM, Siersema PD, van Boeckel PG, et al. Single and sequential biodegradable stent placement for refractory benign

- esophageal strictures: a prospective follow-up study. *Endoscopy* 2012;44:649-54.
114. Dzeletovic I, Fleischer DE. Self-dilation for resistant, benign esophageal strictures. *Am J Gastroenterol* 2010;105:2142-3.
 115. Grobe JL, Kozarek RA, Sanowski RA. Self-bougienage in the treatment of benign esophageal stricture. *J Clin Gastroenterol* 1984;6:109-12.
 116. Dzeletovic I, Fleischer DE. Esophageal self-dilation: a teaching guide for physicians [video]: ASGE Learning Center Programs; 2011.
 117. Mikaeli J, Bishehsari F, Montazeri G, et al. Pneumatic balloon dilatation in achalasia: a prospective comparison of safety and efficacy with different balloon diameters. *Aliment Pharmacol Ther* 2004;20:431-6.
 118. Vaezi MF, Richter JE. Current therapies for achalasia: comparison and efficacy. *J Clin Gastroenterol* 1998;27:21-35.
 119. Lambroza A, Schuman RW. Pneumatic dilation for achalasia without fluoroscopic guidance: safety and efficacy. *Am J Gastroenterol* 1995;90:1226-9.
 120. Rai RR, Shende A, Joshi A, et al. Rigiflex pneumatic dilation of achalasia without fluoroscopy: a novel office procedure. *Gastrointest Endosc* 2005;62:427-31.
 121. Ghoshal UC, Kumar S, Saraswat VA, et al. Long-term follow-up after pneumatic dilation for achalasia cardia: factors associated with treatment failure and recurrence. *Am J Gastroenterol* 2004;99:2304-10.
 122. West RL, Hirsch DP, Bartelsman JF, et al. Long term results of pneumatic dilation in achalasia followed for more than 5 years. *Am J Gastroenterol* 2002;97:1346-51.
 123. Karamanolis G, Sgouros S, Karatzias G, et al. Long-term outcome of pneumatic dilation in the treatment of achalasia. *Am J Gastroenterol* 2005;100:270-4.
 124. Farhoomand K, Connor JT, Richter JE, et al. Predictors of outcome of pneumatic dilation in achalasia. *Clin Gastroenterol Hepatol* 2004;2:389-94.
 125. Metman EH, Lagasse JP, d'Altoche L, et al. Risk factors for immediate complications after progressive pneumatic dilation for achalasia. *Am J Gastroenterol* 1999;94:1179-85.
 126. Eckardt VF, Kanzler G, Westermeier T. Complications and their impact after pneumatic dilation for achalasia: prospective long-term follow-up study. *Gastrointest Endosc* 1997;45:349-53.
 127. Boeckxstaens GE, Annese V, des Varannes SB, et al. Pneumatic dilation versus laparoscopic Heller's myotomy for idiopathic achalasia. *N Engl J Med* 2011;364:1807-16.
 128. Vaezi MF, Richter JE, Wilcox CM, et al. Botulinum toxin versus pneumatic dilatation in the treatment of achalasia: a randomised trial. *Gut* 1999;44:231-9.
 129. Bansal R, Nostrant TT, Scheiman JM, et al. Intrasphincteric botulinum toxin versus pneumatic balloon dilation for treatment of primary achalasia. *J Clin Gastroenterol* 2003;36:209-14.
 130. Wang L, Li YM, Li L. Meta-analysis of randomized and controlled treatment trials for achalasia. *Dig Dis Sci* 2009;54:2303-11.
 131. Weber CE, Davis CS, Kramer HJ, et al. Medium and long-term outcomes after pneumatic dilation or laparoscopic Heller myotomy for achalasia: a meta-analysis. *Surg Laparosc Endosc Percutan Tech* 2012;22:289-96.
 132. Panaccione R, Gregor JC, Reynolds RP, et al. Intrasphincteric botulinum toxin versus pneumatic dilatation for achalasia: a cost minimization analysis. *Gastrointest Endosc* 1999;50:492-8.
 133. Imperiale TF, O'Connor JB, Vaezi MF, et al. A cost-minimization analysis of alternative treatment strategies for achalasia. *Am J Gastroenterol* 2000;95:2737-45.
 134. Pandolfino JE, Kwiatek MA, Nealis T, et al. Achalasia: a new clinically relevant classification by high-resolution manometry. *Gastroenterology* 2008;135:1526-33.
 135. Hunt DR, Wills VL, Weis B, et al. Management of esophageal perforation after pneumatic dilation for achalasia. *J Gastrointest Surg* 2000;4:411-5.
 136. Guardino JM, Vela MF, Connor JT, et al. Pneumatic dilation for the treatment of achalasia in untreated patients and patients with failed Heller myotomy. *J Clin Gastroenterol* 2004;38:855-60.
 137. Vela MF, Richter JE, Wachsberger D, et al. Complexities of managing achalasia at a tertiary referral center: use of pneumatic dilatation, Heller myotomy, and botulinum toxin injection. *Am J Gastroenterol* 2004;99:1029-36.
 138. Ponsky JL, Marks JM, Pauli EM. How I do it: per-oral endoscopic myotomy (POEM). *J Gastrointest Surg* 2012;16:1251-5.
 139. Inoue H, Minami H, Kobayashi Y, et al. Peroral endoscopic myotomy (POEM) for esophageal achalasia. *Endoscopy* 2010;42:265-71.
 140. von Renteln D, Inoue H, Minami H, et al. Peroral endoscopic myotomy for the treatment of achalasia: a prospective single center study. *Am J Gastroenterol* 2012;107:411-7.
 141. Zaninotto G, Marchese RR, Contantini BC, et al. The role of botulinum toxin injection and upper esophageal sphincter myotomy in treating oropharyngeal dysphagia. *J Gastrointest Surg* 2004;8:997-1006.
 142. Solt J, Bajor J, Moizis M, et al. Primary cricopharyngeal dysfunction: treatment with balloon catheter dilatation. *Gastrointest Endosc* 2001;54:767-71.
 143. Wang AY, Kadkade R, Kahrilas PJ, et al. Effectiveness of esophageal dilation for symptomatic cricopharyngeal bar. *Gastrointest Endosc* 2005;61:148-52.
 144. Clouse RE. Complications of endoscopic gastrointestinal dilation techniques. *Gastrointest Endosc Clin N Am* 1996;6:323-41.
 145. Quine MA, Bell GD, McCloy RF, et al. Prospective audit of perforation rates following upper gastrointestinal endoscopy in two regions of England. *Br J Surg* 1995;82:530-3.
 146. Fadoo F, Ruiz DE, Dawn SK, et al. Helical CT esophagography for the evaluation of suspected esophageal perforation or rupture. *AJR Am J Roentgenol* 2004;182:1177-9.
 147. Siersema PD, Homs MY, Haringsma J, et al. Use of large-diameter metallic stents to seal traumatic nonmalignant perforations of the esophagus. *Gastrointest Endosc* 2003;58:356-61.
 148. Gelbmann CM, Ratiu NL, Rath HC, et al. Use of self-expandable plastic stents for the treatment of esophageal perforations and symptomatic anastomotic leaks. *Endoscopy* 2004;36:695-9.

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