

## Esophageal dilation

*This is one of a series of statements discussing the use of gastrointestinal endoscopy in common clinical situations. The Standards of Practice Committee of the American Society for Gastrointestinal Endoscopy prepared this text. In preparing this guideline, a MEDLINE literature search was performed, and additional references were obtained from the bibliographies of the identified articles and from recommendations of expert consultants. When little 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. Further controlled clinical studies are needed to clarify aspects of this statement, and revision may be necessary as new data appear. Clinical consideration may justify a course of action at variance to these recommendations.*

### INTRODUCTION

The purpose of this updated guideline is to provide practical recommendations regarding the indications and techniques for the use of esophageal dilation. Esophageal dilation (EGD) is performed for treatment of documented anatomic, and sometimes functional, narrowing of the esophagus caused by a variety of benign and malignant conditions.<sup>1</sup> The formation of benign strictures of the esophagus is believed to be caused by the production of fibrous tissue and deposition of collagen stimulated by deep esophageal ulceration or chronic inflammation.<sup>1</sup> The most common form of an esophageal stricture, a peptic stricture, is a sequela of reflux esophagitis. In the recent past, nearly 80% of strictures were due to gastroesophageal reflux,<sup>2</sup> although this may be decreasing with the widespread use of proton pump inhibitors (PPIs). Other common benign causes include Schatzki's ring, radiation therapy, congenital strictures, caustic ingestion, and anastomotic strictures. Less common causes of benign esophageal strictures include those following endoscopic therapy of varices, photodynamic therapy (PDT),<sup>1</sup> reaction to a foreign body or pill, infectious esophagitis, and

eosinophilic esophagitis (Table 1). Narrowing of the esophagus from malignancy may result either from intrinsic luminal tumor growth or from extrinsic esophageal compression. During the endoscopic evaluation of an esophageal stricture, biopsy specimens should be taken to exclude malignancy when this diagnosis is suspected on the basis of clinical presentation or endoscopic appearance. In young patients with dysphagia with or without endoscopic abnormalities, especially with a history of food impaction, midesophageal biopsy specimens should be obtained to exclude eosinophilic esophagitis.<sup>3</sup> Endoscopic esophageal biopsy samples can be safely obtained before esophageal dilation.<sup>4</sup>

Patients with an esophageal stricture characteristically have dysphagia to solids and generally have no difficulty swallowing liquids, in contrast to those with an esophageal motility disorder in which liquid and solid dysphagia occurs.<sup>1</sup> Symptoms in the latter group of patients are generally not improved with esophageal dilation, with achalasia being the most notable exception.

### EOSINOPHILIC ESOPHAGITIS

Eosinophilic esophagitis deserves special mention because it is becoming increasingly common,<sup>5</sup> there is available therapy in addition to dilation,<sup>6</sup> there are recognizable endoscopic<sup>7-9</sup> and histologic features,<sup>10</sup> and there appears to be an increased risk for mucosal tearing during endoscopy.<sup>11</sup> The latter may translate into an increased risk perforation during dilation.<sup>12</sup> Eosinophilic esophagitis is common in young patients with otherwise unexplained dysphagia. A clinical presentation of food impaction is not uncommon.<sup>13</sup>

### INDICATIONS FOR DILATION

The primary indication for esophageal dilation is to relieve dysphagia. Cost analysis evaluations have suggested that initial EGD with therapeutic intent is less costly than a barium swallow in patients with a history suggesting esophageal obstruction.<sup>14</sup> Additionally, early endoscopy should be the initial diagnostic test performed in patients with dysphagia who are  $\geq 40$  years old and those with concomitant heartburn, odynophagia, or weight loss because of the high yield of finding significant pathology in these patients.<sup>15</sup>

**TABLE 1. Common causes of esophageal strictures/obstruction**

Gastroesophageal reflux disease (peptic)
Schatzki's ring
Esophageal cancer
Radiation therapy
Esophageal surgery
Eosinophilic esophagitis
Sclerotherapy
Caustic injury
PDT

Esophageal strictures can be structurally categorized into two groups: simple and complex.<sup>16</sup> Simple strictures are symmetric or concentric with a diameter of  $\geq 12$  mm or easily allow passage of a diagnostic upper endoscope. Complex strictures have one or more of the following features: asymmetry, diameter  $\leq 12$  mm or inability to pass an endoscope. Regardless of the cause, dysphagia is an indication for dilation of benign strictures.<sup>1</sup> Although some endoscopists suggest that large-bore dilators be passed empirically if the endoscopy has normal results,<sup>3</sup> results from two of three studies<sup>17-19</sup> have shown that empiric dilation does not improve dysphagia scores. Thus, because of the potential risk of perforation with use of large-bore dilators, particularly in patients with unrecognized eosinophilic esophagitis,<sup>12</sup> empiric dilation cannot be routinely recommended if no structural abnormalities are seen at endoscopy.

Most data regarding management of esophageal strictures have been gathered in the adult population. The safety and efficacy of esophageal dilation in children has also been confirmed.<sup>20,21</sup>

Endoscopic dilation of malignant strictures can be done to assist the completion of endoscopic procedures such as endoscopic ultrasonographic tumor staging<sup>22,23</sup> or to aid the placement of an esophageal stent to achieve temporary palliation.<sup>24</sup> Most malignant strictures respond to dilation, but relief of dysphagia is transient and more definitive treatment is usually needed. The dysphagia caused by malignant extrinsic compression of the esophagus responds poorly to esophageal dilation.

## DILATOR TYPES

Three general types of dilators are currently in use. These are (1) mercury or tungsten-filled bougies (Maloney or Hurst), (2) wire-guided polyvinyl dilators (Savary-Gilliard or American), and (3) TTS ("through-the-scope") balloon dilators. The Maloney type bougies have a tapered

tip and can be passed either blindly<sup>25</sup> or under fluoroscopic control. Fluoroscopy may lead to better functional results and fewer adverse events.<sup>26</sup> This type of dilator is used for simple strictures with a diameter of 12 to 14 mm. The risk of esophageal perforation may be higher with blind passage of Maloney dilators than with Savary or TTS balloons, particularly in patients with a large hiatal hernia, a tortuous esophagus, or those with complex strictures.<sup>16</sup> Savary and American dilators are passed over a guidewire that has been positioned with the tip in the gastric antrum, with or without fluoroscopic guidance.<sup>27</sup> There are a variety of available TTS balloon dilators available in either single or multiple diameters that may be passed with or without wire guidance. A new endoscopically guided bougie has recently become available (InScope) but clinical experience with it is limited.

## PREPARATION

Anticoagulants should be discontinued.<sup>28</sup> Routine antibiotic coverage is not recommended; endocarditis prophylaxis guidelines should be followed.<sup>29</sup> During the informed consent process, patients should be informed about the risk of perforation and the possible need for surgery should it occur. Esophageal dilation is routinely performed in an outpatient setting. Patients should fast for 4 to 6 hours before the procedure. Patients with achalasia are susceptible to esophageal stasis and a prolonged fast or esophageal lavage may be required to empty the esophagus. Although some patients may tolerate dilation with use of only topical anesthesia, conscious sedation is generally used.<sup>30</sup> When bougie dilators are used, neck extension may facilitate passage of the dilator.

## TECHNIQUES

The degree of dilation within a session should be based on the severity of the stricture. A conservative approach to dilation may reduce the risk of perforation. The "rule of 3" has been accepted and applied to bougie dilation of esophageal strictures.<sup>31</sup> Specifically, the initial dilator chosen should be based on the known or estimated stricture diameter. Serial increases in diameter are then performed. After moderate resistance is encountered with the bougie-type dilator, no greater than 3 consecutive dilators in increments of 1 mm should be passed in a single session. Although this rule does not apply to balloon dilators, a recent study suggested that inflation of a single large-diameter dilator ( $>15$  mm) or incremental dilation of greater than 3 mm may be safe in simple esophageal strictures.<sup>32</sup> There are no data on the optimal duration the balloon should remain inflated. Dilation therapy for symptomatic Schatzki's ring is directed toward achieving rupture of the ring; therefore, larger caliber dilators (16-20 mm) may be needed.<sup>33</sup> If a lower esophageal ring

cannot be distinguished from a short peptic stricture, graded stepwise dilation is recommended.

During esophageal dilation the endoscopist should be supported by assistants who are familiar with the endoscopic and dilating devices considered for use and are capable of monitoring patient comfort and safety throughout the examination. Patients should be closely observed after esophageal dilation, with pulse, blood pressure, and temperature measured regularly to detect complications.

Steroid injection into benign strictures immediately before or after dilation has been advocated to improve outcomes by decreasing the need for repeat dilation in strictures that have not responded to initial dilation. Most of the published studies to date have been small, nonrandomized, and uncontrolled.<sup>34,35</sup> Additionally, not all causes of stricture respond similarly to steroid injection. A recent randomized trial of intralesional steroid injection with PPI therapy versus sham injection with PPI therapy in patients with recalcitrant peptic esophageal strictures showed that the need for repeat dilation was significantly diminished in the steroid group.<sup>36</sup>

## RESULTS

Regardless of the specific method of dilation, early improvement in the ability to swallow is achieved in virtually all patients; however, longer-term outcomes are influenced by the underlying pathologic condition. If a luminal diameter of at least 13 to 15 mm can be achieved, nearly all patients will be relieved of dysphagia. In patients with benign peptic strictures, a graded stepwise dilating approach between 13 and 20 mm yields relief in 85% to 93%.<sup>4</sup> Bougie-type dilators exert not only radial forces as they are passed but also longitudinal forces as the result of a shearing effect.<sup>37</sup> Longitudinal forces are not transmitted with balloon dilators because the entire dilating force is delivered radially and simultaneously over the entire length of the stenosis rather than progressively from its proximal to distal extent.<sup>37</sup> Despite these differences, no clear advantage has been demonstrated between the two dilator types.<sup>38-40</sup> Factors associated with a poor response to balloon dilation of benign strictures are a length of >8 cm and a small predilation luminal diameter.<sup>41</sup> In patients with benign peptic strictures, the long-term benefits of dilation appear greatest when a luminal diameter of greater than 12 mm is achieved.<sup>42</sup>

Several clinical features are associated with outcome. For peptic strictures, smaller lumen diameter, presence of a hiatal hernia >5 cm, persistence of heartburn after dilation, and number of dilations needed for initial dysphagia relief were significant predictors of early symptomatic recurrence.<sup>43</sup> A multivariate analysis revealed that a non-peptic etiology of strictures was a significant predictor of early symptomatic recurrence within 1 year of initial dilation.<sup>41</sup> One study suggested that patients with peptic stric-

tures but without heartburn or patients with weight loss may be more likely to require frequent dilations.<sup>44</sup>

Patients with peptic strictures should be treated with PPI therapy. Compared with histamine receptor antagonist therapy, PPI use decreases stricture recurrence and the need for repeat stricture dilation.<sup>45-49</sup> Recent studies suggest that acid suppression may prevent recurrence of Schatzki's rings after dilation.<sup>50</sup>

## ACHALASIA

Esophageal dilation for achalasia involves the forceful disruption of the lower esophageal sphincter (LES). This is usually accomplished with 30- to 40-mm diameter pneumatic balloon dilators. Several balloon types are available. Although short-term relief of dysphagia is good, recurrence occurs in approximately one third<sup>51</sup> and, in some series, long-term resolution of symptoms after initial response may be as low as 40% to 50%.<sup>52,53</sup> The risk of perforation with balloon dilation in achalasia is in the range of 3% to 4% with a mortality rate of <1%.<sup>54,55</sup> Dilation is generally performed over a wire under fluoroscopic guidance initially using a 30-mm balloon,<sup>56</sup> although nonfluoroscopically guided dilation using endoscopic visualization alone has been reported.<sup>57,58</sup>

An alternative to dilation is the endoscopic injection of botulinum toxin. Botulinum toxin acts by inhibiting the calcium-dependent release of acetylcholine from nerve terminals. The proposed mechanism of action is relaxation of the LES, but the effect on manometrically determined LES pressure is variable.<sup>59</sup> Botulinum toxin is injected at 4 to 5 sites at the endoscopically identified LES. The usual total dose is 100 units diluted in 5 to 10 mL. Injection of botulinum toxin into the LES is effective in relieving symptoms in about 85% of patients. This response, however, is short lived, with symptom recurrence in greater than 50% by 6 months.<sup>58</sup> 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%).<sup>59,60</sup>

Surgical treatment of achalasia has yielded greater therapeutic efficacy than either pneumatic dilation or botulinum toxin injection. Myotomy offers good to excellent symptom improvement in 83% of patients.<sup>55</sup> Laparoscopic cardiomyotomy has shown similar results; however, longer-term follow-up is continuing.<sup>61</sup> Cardiomyotomy may be more difficult and less effective in patients treated previously with botulinum toxin due to submucosal scarring.<sup>62</sup> A randomized controlled trial comparing laparoscopic myotomy and botulinum toxin injection showed similar safety, but with better outcomes achieved with surgery.<sup>61</sup>

Before endoscopic treatment, patients with achalasia should be informed of the various 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 an outcome similar to that of elective open myotomy.<sup>63</sup> However, if endoscopic perforation occurs after pneumatic dilation, laparoscopic myotomy is usually not technically feasible.<sup>63</sup> In patients with failed myotomy, pneumatic dilation can be safely performed.<sup>64,65</sup> The subset of patients in whom this approach has failed may require esophagectomy. In patients who are poor candidates for surgery, initial therapy with botulinum toxin may be the preferred approach. In prohibitive operative candidates, pneumatic dilation is not recommended.

Cost analysis models indicate that, for otherwise healthy patients with achalasia, initial pneumatic dilation was the least costly strategy compared with botulinum toxin injection<sup>66</sup> or laparoscopic Heller myotomy.<sup>67</sup>

## CONTRAINDICATIONS AND COMPLICATIONS

The principal complications of esophageal dilation are perforation, bleeding, and aspiration. The most serious complication of esophageal dilation is perforation. The perforation rate for esophageal strictures after dilation has been reported to be 0.1% to 0.4%.<sup>16</sup> The risk of perforation is lower in simple strictures and higher in more complex strictures.<sup>16</sup> Perforation may be more common and severe with radiation-induced strictures.<sup>68</sup> The perforation rate may be influenced by endoscopist experience level; one study indicated that the perforation rate was 4 times greater when the operator had performed fewer than 500 previous diagnostic upper endoscopic examinations.<sup>69</sup> Perforation after esophageal dilation usually occurs at the site of the stricture, either intraabdominally or intrathoracically. This complication should be suspected if severe or persistent pain, dyspnea, tachycardia, or fever develops. 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 this diagnosis and a water-soluble contrast esophagram or contrast chest computed tomogram may be necessary to delineate a perforation.<sup>70</sup> The use of large-diameter covered metal stents and the use of expandable, removable plastic stents have been shown to be effective in the management of perforations after dilation of benign and malignant esophageal strictures, although the routine use of these devices in benign disease is not recommended.<sup>71,72</sup>

Esophageal dilation should be performed with caution in patients who have had a recent, healed perforation or upper gastrointestinal surgery. Continuing esophageal perforation is an absolute contraindication to esophageal dilation.

## SUMMARY

For the following points: (A), prospective controlled trials; (B), observational studies; (C), expert opinion.

- Dilation is indicated in patients with symptomatic esophageal strictures (B).
- Fluoroscopy is recommended when using non-wire-guided dilators during dilation of complex esophageal strictures or in patients with a tortuous esophagus (B).
- Bougie and balloon dilators are equally effective in relief of dysphagia in patients with esophageal strictures (A).
- The rule of 3 should be followed when dilation of esophageal strictures is performed with bougie dilators (B).
- Injection of corticosteroids into recurrent or refractory benign esophageal strictures may improve the outcome after esophageal dilation (B).
- Pneumatic dilation with large-diameter balloons is effective for the treatment of achalasia (A).
- Botulinum toxin therapy is the preferred endoscopic treatment for achalasia in poor operative and nonoperative patients (B).
- Administration of PPIs is effective in preventing recurrence of esophageal strictures and the need for repeat esophageal dilation (A).

## REFERENCES

1. Lew RJ, Kochman ML. A review of endoscopic methods of esophageal dilation. *J Clin Gastroenterol* 2002;35:117-26.
2. Richter JE. Peptic strictures of the esophagus. *Gastroenterol Clin North Am* 1999;28:875-91.
3. Arora AS. Management strategies for dysphagia with a normal-appearing esophagus. *Clin Gastroenterol Hepatol* 2005;3:299-302.
4. Riley SA, Attwood SE. Guidelines on the use of oesophageal dilatation in clinical practice. *Gut* 2004;53(1 Suppl):i1-6.
5. Noel RJ, Rothenberg ME. Eosinophilic esophagitis. *Curr Opin Pediatr* 2005;17:690-4.
6. Noel RJ, Putnam PE, Collins MH, et al. Clinical and immunopathologic effects of swallowed fluticasone for eosinophilic esophagitis. *Clin Gastroenterol Hepatol* 2004;2:568-75.
7. Potter JW, Saeian K, Staff D, et al. Eosinophilic esophagitis in adults: an emerging problem with unique esophageal features. *Gastrointest Endosc* 2004;59:355-61.
8. Croese J, Fairley SK, Masson JW, et al. Clinical and endoscopic features of eosinophilic esophagitis in adults. *Gastrointest Endosc* 2003;58:516-22.
9. Vasilopoulos S, Murphy P, Auerbach A, et al. The small caliber esophagus: an unappreciated cause of dysphagia for solids in patients with eosinophilic esophagitis. *Gastrointest Endosc* 2002;55:99-106.
10. Parfitt JR, Gregor JC, Suskin NG, et al. Eosinophilic esophagitis in adults: distinguishing features from gastroesophageal reflux disease: a study of 41 patients. *Mod Pathol* 2006;19:90-6.
11. Straumann A, Rossi L, Simon HU, et al. Fragility of the esophageal mucosa: a pathognomonic endoscopic sign of primary eosinophilic esophagitis? *Gastrointest Endosc* 2003;57:407-12.
12. Kaplan M, Mutlu EA, Jakate S, et al. Endoscopy in eosinophilic esophagitis: "feline" esophagus and perforation risk. *Clin Gastroenterol Hepatol* 2003;1:433-7.
13. Desai TK, Stecevic V, Chang CH, et al. Association of eosinophilic inflammation with esophageal food impaction in adults. *Gastrointest Endosc* 2005;61:795-801.
14. Esfandyari T, Potter JW, Vaezi MF. Dysphagia: a cost analysis of the diagnostic approach. *Am J Gastroenterol* 2002;97:2733-7.
15. 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.



16. 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.
17. 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.
18. Scolapio JS, Gostout CJ, Schroeder KW, et al. Dysphagia without endoscopically evident disease: to dilate or not? *Am J Gastroenterol* 2001;96:327-30.
19. 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.
20. Lan LC, Wong KK, Lin SC, et al. Endoscopic balloon dilation of esophageal strictures in infants and children: 17 years' experience and a review of the literature. *J Pediatr Surg* 2003;38:1712-5.
21. Wilsey MJ Jr, Scheimann AO, Gilger MA. The role of upper gastrointestinal endoscopy in the diagnosis and treatment of caustic ingestion, esophageal strictures, and achalasia in children. *Gastrointest Endosc Clin North Am* 2001;11:767-87, vii-viii.
22. 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.
23. Wallace MB, Hawes RH, Sahai AV, et al. Dilation of malignant esophageal stenosis to allow EUS-guided fine needle aspiration: safety and effect on patient management. *Gastrointest Endosc* 2000;51:309-13.
24. Adler DG, Baron TH. Endoscopic palliation of malignant dysphagia. *Mayo Clin Proc* 2001;76:731-8.
25. Ho SB, Cass O, Katsman RJ, et al. Fluoroscopy is not necessary for Maloney dilation of chronic esophageal strictures. *Gastrointest Endosc* 1995;42:11-4.
26. McClave SA, Brady PG, Wright RA, et al. Does fluoroscopic guidance for Maloney esophageal dilation impact on the clinical endpoint of therapy: relief of dysphagia and achievement of luminal patency? *Gastrointest Endosc* 1996;43:93-7.
27. Wang YG, Tio TL, Soehendra N. Endoscopic dilation of esophageal stricture without fluoroscopy is safe and effective. *World J Gastroenterol* 2002;8:766-8.
28. Eisen GM, Baron TH, Dominitz JA, et al. Guideline on the management of anticoagulation and antiplatelet therapy for endoscopic procedures. *Gastrointest Endosc* 2002;55:775-9.
29. Hirota K, Petersen K, Baron TH, et al. Antibiotic prophylaxis for GI endoscopy. *Gastrointest Endosc* 2003;58:475-82.
30. Petrini J, Egan J. Risk management regarding sedation/analgesia. *Gastrointest Endosc Clin North Am* 2004;14:401-14.
31. Langdon DF. The rule of three in esophageal dilation. *Gastrointest Endosc* 1997;45:111.
32. 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.
33. Jalil S, Castell DO. Schatzki's ring: a benign cause of dysphagia in adults. *J Clin Gastroenterol* 2002;35:295-8.
34. Zein NN, Greseth JM, Perrault J. Endoscopic intralesional steroid injections in the management of refractory esophageal strictures. *Gastrointest Endosc* 1995;41:596-8.
35. 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.
36. 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.
37. McLean GK, LeVeen RF. Shear stress in the performance of esophageal dilation: comparison of balloon dilation and bougienage. *Radiology* 1989;172:983-6.
38. 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.
39. 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.
40. Joyce A, Ginsberg G, Katzka DA, et al. Esophageal dilation at a tertiary referral center [abstract 36]. *Gastrointest Endosc* 2005;61:1168.
41. 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.
42. Saeed ZA, Ramirez FC, Hepps KS, et al. An objective end point for dilation improves outcome of peptic esophageal strictures: a prospective randomized trial. *Gastrointest Endosc* 1997;45:354-9.
43. Said A, Brust DJ, Gaumnitz EA, et al. Predictors of early recurrence of benign esophageal strictures. *Am J Gastroenterol* 2003;98:1252-6.
44. Agnew SR, Pandya SP, Reynolds RP, et al. Predictors for frequent esophageal dilations of benign peptic strictures. *Dig Dis Sci* 1996;41:931-6.
45. Barbezat GO, Schlup M, Lubcke R. Omeprazole therapy decreases the need for dilatation of peptic oesophageal strictures. *Aliment Pharmacol Ther* 1999;13:1041-5.
46. Marks RD, Richter JE, Rizzo J, et al. Omeprazole versus H2-receptor antagonists in treating patients with peptic stricture and esophagitis. *Gastroenterology* 1994;106:907-15.
47. 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.
48. Stal JM, Gregor JC, Preiksaitis HG, et al. A cost-utility analysis comparing omeprazole with ranitidine in the maintenance therapy of peptic esophageal strictures. *Can J Gastroenterol* 1998;12:43-9.
49. 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.
50. Sgouros SN, Vlachogiannakos J, Karamanolis G, et al. Long-term acid suppressive therapy may prevent the relapse of lower esophageal (Schatzki's) rings: a prospective, randomized, placebo-controlled study. *Am J Gastroenterol* 2005;100:1929-34.
51. 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.
52. 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.
53. 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.
54. Metman EH, Lagasse JP, d'Alteroche L, et al. Risk factors for immediate complications after progressive pneumatic dilation for achalasia. *Am J Gastroenterol* 1999;94:1179-85.
55. Vaezi MF, Richter JE. Current therapies for achalasia: comparison and efficacy. *J Clin Gastroenterol* 1998;27:21-35.
56. Mikaeli J, Bishehsari F, Montazeri G, et al. Pneumatic balloon dilation in achalasia: a prospective comparison of safety and efficacy with different balloon diameters. *Aliment Pharmacol Ther* 2004;15:431-6.
57. Lambroza A, Schuman RW. Pneumatic dilation for achalasia without fluoroscopic guidance: safety and efficacy. *Am J Gastroenterol* 1995;90:1226-9.
58. 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.
59. 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.

60. 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.
61. Zaninotto G, Annese V, Costantini M, et al. Randomized controlled trial of botulinum toxin versus laparoscopic Heller myotomy for esophageal achalasia. *Ann Surg* 2004;239:364-70.
62. Vaezi MF, Richter JE. Diagnosis and management of achalasia: American College of Gastroenterology Practice Parameter Committee. *Am J Gastroenterol* 1999;94:3406-12.
63. Hunt DR, Wills VL, Weis B, et al. Management of esophageal perforation after pneumatic dilation for achalasia. *J Gastrointest Surg* 2000;4:411-5.
64. 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.
65. 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.
66. 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.
67. 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.
68. Clouse RE. Complications of endoscopic gastrointestinal dilation techniques. *Gastrointest Endosc Clin North Am* 1996;6:323-41.
69. 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.
70. 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.
71. 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.
72. 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.

Disclosure: This article was not subject to the peer review process of GIE.

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