Pneumatic Dilation

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History

Achalasia is caused by the selective loss of inhibitory neurons in the myenteric plexus resulting in failure of the lower esophageal sphincter (LES) to relax. Currently there are no treatments to reverse the underlying neurologic dysfunction and restore normal esophageal motor function. Existing therapies aim to palliate symptoms via reduction of LES pressure to allow esophageal emptying by gravity and improve bolus transit through the cardia. The primary therapeutic options for achalasia are pneumatic dilation (PD), laparoscopic Heller myotomy (LHM), botulinum toxin injections, and pharmacotherapy.

PD leads to stretching and controlled mechanical disruption of the circular smooth muscle fibers of the LES and resultant fracture of the muscularis propria. Forceful dilation of the LES dates back to 1674 when Sir Thomas Willis used a carved whalebone with a sponge affixed to the distal end as a prototypic bougie to accomplish distraction of the muscular fibers at the gastroesophageal junction (GEJ) [1]. Willis first described achalasia as a "spasm of the lower esophageal sphincter". In 1937, Frederick Lendrum proposed the modern-day concept that the syndrome is caused by incomplete relaxation of the LES. He branded the disease process *achalasia*, a word of Greek origin with the literal translation being "without loosening" [2].

The technique of PD has evolved through several models of balloon dilators, many of which are no longer manufactured. Standard balloon dilators or bougienage are ineffective in the degree of disruption of the LES muscle fibers needed for symptomatic relief [3].

Early metal dilators (Starck) were modified in the early 1990s so that expanding balloons were incorporated onto flexible shafts so that they could be placed at the LES to forcefully dilate. The first balloon, called the Plummer hydrostatic dilator, utilized water as an expander. Subsequent dilators replaced water with air and were therefore referred to as pneumatic dilators [4, 5].

The Browne-McHardy and Hurst-Tucker pneumatic dilators consisted of mercury-filled tubes with a rubber covered silk bag at the distal end. The Mosher bag contained barium strips embedded within the wall of the bag to facilitate fluoroscopic visualization. The Rider-Mueller

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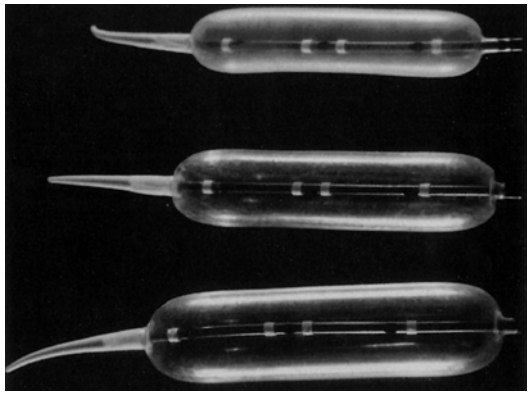

Fig. 6.1 Rigiflex pneumatic balloon dilators with three diameter sizes: 3.0, 3.5, and 4.0 cm (Adapted by permission from Richter and Roberts [6])

dilator was the first dilator to be available in a number of sizes and consisted of a dumbbell-shaped bag that could be positioned across the GEJ via guidewire placement. The Sippy pneumatic dilator employed two latex balloons covered by a nylon bag to limit expansion of the balloon. Each of the aforementioned dilators required fluoroscopy for proper positioning before dilation and ranged from 2.5 to 4.5 cm [6].

Currently, the most commonly employed balloon dilator in the United States is a non-radiopaque, non-compliant air-filled polyethylene balloon, known as the Rigiflex balloon (Boston Scientific, Marlborough, MA, USA) [3] (Fig. 6.1)

Technique of Pneumatic Dilation

PD is typically an outpatient procedure [7]. The patient is required to take nothing by mouth for 12 h preceding endoscopy with recommended adherence to a liquid diet for at least 1–2 days prior. In those with clinical or radiographic evidence of severe food retention and resultant dilation of the esophagus, a lavage with a large bore tube may be necessary [8]. All patients should be appropriate candidates for surgical intervention if an esophageal perforation were to arise.

The balloon system should first be inflated and checked for leaks or signs of malfunction. A comprehensive endoscopic evaluation should be performed prior to dilation with special attention to the gastric cardia during the retroflexed exam, to rule out mechanical obstruction or pseudoachalasia which can mimic achalasia [3]. Landmarks should also be determined, particularly the distance between the incisors and the gastroesophageal junction.

A guidewire is placed into the stomach via the working channel on the endoscope. The endoscope should be carefully removed to preserve the position of the guidewire in the stomach. The balloon and tip of the catheter are then lubricated and passed over the guidewire.

The previously noted distance between the gastroesophageal junction and incisors should be measured from the center of the balloon to ensure that the center will be across the LES. The position of the balloon should be so that the "waist" caused by the non-relaxing LES applies pressure on the center of the distending balloon [8].

This position is usually at or above the level of the diaphragm, except in patients after Heller myotomy, when the narrowing may be below the diaphragm. Minor re-adjustments in positioning may have to be made to ensure proper location, with deflation of the balloon with each adjustment. If performed under fluoroscopic guidance, a small volume of dilute contrast can be injected into the balloon to assist in radiographic visualization [9].

After proper positioning, the balloon is then connected to an external pressure gauge and is inflated until the pressure reaches 7–15 pounds per square inch (psi) of pressure (approximately 120 mL of air) and held for 6–60 s. Balloon distension time is variable, but studies have shown distension times as short as 6–15 s are as effective as longer distension times up 60 s [10, 11].

If a second inflation is necessary, the pressure will typically be at least 3 psi less than the initial pressure. A precipitous decrease of the intraballoon pressure signifies successful disruption of LES muscle fibers. After this is achieved, the balloon is then deflated and carefully removed.

Post-procedurally, it is recommended that patients routinely undergo a Gastrografin study followed by a barium esophogram to exclude perforation, however, in clinical practice, this is often only done if suspicious signs or symptoms

are present [12, 13]. Observation of the patient is generally recommended for 5–8 h to monitor for chest pain, fevers, and signs of perforation [14]. The patient is subsequently discharged if tolerating fluids without difficulty and the recovery was otherwise uneventful.

There is no clear consensus on the optimal method for performing PD with regard to balloon diameter and the amount and rate of inflation pressure. However, a 30 mm Rigiflex balloon is typically used for the initial dilation in most adults [8]. The standard approach is to perform one dilation per session, with repeat dilations being performed according to symptomatic recurrence.

Summary of Data Regarding Efficacy

Pneumatic dilation is considered to be the most cost-effective first-line therapy for achalasia over a 5–10 year post-procedure period [15–17]. The graded approach to dilation is effective in achieving symptom relief. The 3 year success rate for a single dilation with a 30-mm balloon is 37 % in comparison with 86 % for the graded dilation protocol [18].

Other studies estimate that with a graded dilation approach, symptomatic relief is achieved in 50–93 % of patients [19]. The 2013 American College of Gastroenterology guidelines regarding the diagnosis and management of achalasia cite that PD with 30, 35, and 40 mm balloon diameters result in symptomatic relief in 74, 86, and 90 % of patients, respectively, with an average follow-up of 1.6 years (range: 0.1–6 years) [3, 20].

Variability in these results is likely secondary to both inconsistent follow-up times as well as lack of consensus regarding a distension protocol. Data from retrospective studies are more limited by lack of follow-up, while prospective studies may be more accurate in predicting efficacy. One recent prospective study reported that 70 % of patients who underwent PD maintained control of symptoms after a median of 5.6 years of follow-up [21]. A prospective randomized controlled trial comparing PD to LHM found that there was no significant difference in rates of therapeutic success. In patients who underwent PD, clinical remission was reported in 90 % and 86 % after 1 and 2 years of follow up, respectively [22].

Persistent symptoms, especially in conjunction with impaired esophageal emptying or an LES pressure above 10 mmHg warrants repeat dilation with incrementally larger balloons. Generally, if symptom relief is not achieved with a 40 mm balloon or with three consecutive dilations, surgical intervention is then pursued [8].

While symptom-free periods at shorter term follow up times have been reported, approximately one-third of treated patients are expected to experience symptom relapse over 4–6 years of follow up, despite adherence to a graded pneumatic dilation approach [3, 23].

The lack of strong long-term data makes the efficacy of repeated dilations after relapse of symptoms difficult to definitively assess. However, existing studies suggest that patients who remain in clinical remission for 5 years are likely to benefit from the longstanding treatment effect of PD [24].

As there is no definitive cure for achalasia, the proportion of patients who remain in remission after successful graded PD or surgical myotomy declines over time and repeat intervention is typically warranted in 23–33 % of patients within 5–7 years [25].

Predictors of Success

Significant predictors of favorable clinical outcomes after PD include LES pressure after dilation of less than 10 mmHg, older age, female gender, and type II achalasia pattern on high resolution manometry [3].

Post-dilation LES pressure has been considered the single most valuable factor for predicting the long-term clinical response [26]. A post-dilation LES pressure to approximately 10 mmHg has been suggested as a goal of PD. Prospective studies of patients over 10 years also found that those patients with a post-dilation

LES pressure of less than 10–15 mmHg were more likely to achieve sustainable clinical response compared to those with higher LES pressures [24, 27].

Young males, aged less than 45 years, have a greater failure after 30 mm PD as well as after graded PD as compared to older men or women in general, which may be secondary to thicker LES musculature [28]. Age younger than 40 years, irrespective of gender, also predicts a poor response to pneumatic dilation [24, 26, 29].

Females have a better clinical outcome after PD when compared to males [30]. Young men initially treated with a 30 mm balloon were found to require repeat dilations more often than young women [28]. For this reason, initial PD with a 35 mm balloon or surgical myotomy is often considered as first-line therapy in young male patients [8].

The use of high-resolution esophageal manometry has stratified achalasia into three main subtypes which influence the response to therapeutic interventions. While each subtype is unified by the presence of impaired LES relaxation and aperistalsis, each has a distinct manometric finding. Type I, known as classic achalasia, is defined as no pressure generation in the esophageal body. Type II patients exhibit rapidly propagated compartmentalized pressurization, localized to the distal esophagus or present across the entire esophagus. In Type III, or spastic achalasia, patients have lumen-obliterating contractions in the distal esophagus, causing a functional obstruction. A study investigating clinical response to botulinum toxin injections, LHM, and PD found that Type II patients are most likely to respond to any therapy (botulinum toxin injections [71 %], PD [91 %], or LHM [100 %]) than type I (56 % overall) or type III (29 % overall) patients [31]. Severe esophageal dilation associated with any subtype of achalasia also is associated with a decreased response to therapeutic attempts.

Several other variables, such as pre-treatment LES pressure, duration of symptoms, size of balloon dilators utilized, and results of post-dilation barium esophograms have been studied but not found to significantly affect therapeutic response to PD [28, 30, 32].

Complications

The overall PD-associated complication rate is estimated to be lower than 10 % and most commonly include perforation, chest pain, bleeding, fever, aspiration pneumonia, and formation of diverticula [33, 34].

The most important and serious complication of PD is esophageal perforation, with an overall reported rate of 1.9 % (range 0–21 %) [3, 18, 35]. Perforations are typically small and located above the cardia along the left side of the esophagus, where there is an anatomic area of weakness and usually occur during the first dilation session.

Age greater than 60 years and initial dilation performed with 35 mm balloon compared with 30 mm balloon have been identified as risk factors in predisposing to perforation [17, 22]. Other risk factors for transmural perforation have been identified and include inappropriate positioning and distension of the balloon, balloon instability, higher dilation pressures, minimal weight loss, malnutrition, longer duration of symptoms, high-amplitude contractions, and pre-existing esophageal diverticula [17]. Incidence of perforation is generally considered to be lower with the serial, graded balloon dilation approach.

Prompt recognition of possible perforation is crucial either by routinely performing a post-dilation radiograph of the esophagus using water-soluble contrast or by recognizing signs and symptoms of perforation such as persistent chest pain or tachycardia [36]. Assessment of pain evoked by ingestion of water 1–2 h after the procedure can also be diagnostic. Should perforation arise, broad-spectrum antibiotics should be initiated and immediate surgical consultation should be sought. In some clinical situations, conservative management with antibiotics and initiation of parenteral nutrition may be sufficient [17].

PD-induced disruption of the LES, which is the principal barrier to acid reflux, commonly results in resultant gastroesophageal reflux disease (GERD). This has been reported in 15–35 % of patients post-dilation, the majority of which respond to proton-pump inhibitors [18].

Other minor complications have been reported including post-procedural chest pain, intramural

hematomas, and new diverticula, particularly at the gastric cardia. When bleeding does occur, there is usually not an associated drop in hemoglobin [37].

It is recommended that patients should undergo PD only at high-volume centers [3].

References

1. Tolone S, Limongelli P, Del Genio G, Brusciano L, Russo A, Cipriano L, et al. Recent trends in endoscopic management of achalasia. World J Gastrointest Endosc. 2014;6(9):407–14.
2. Spiess AE, Kahrilas PJ. Treating achalasia: from whalebone to laparoscope. JAMA. 1998;280(7): 638–42.
3. Vaezi MF, Pandolfino JE, Vela MF. ACG clinical guideline: diagnosis and management of achalasia. Am J Gastroenterol. 2013;108(8):1238–49. quiz 1250.
4. Bennett JR, Hendrix TR. Treatment of achalasia with pneumatic dilatation. Mod Treat. 1970;7(6):1217–28.
5. Kadakia SC, Wong RK. Pneumatic balloon dilation for esophageal achalasia. Gastrointest Endosc Clin N Am. 2001;11(2):325–46. vii.
6. Richter JE, Roberts JR. Achalasia. In: Richter JE, Castell DO, editors. The esophagus. 5th ed. Oxford, UK: Wiley-Blackwell; 2012. p. 257–301.
7. Barkin JS, Guelrud M, Reiner DK, Goldberg RI, Phillips RS. Forceful balloon dilation: an outpatient procedure for achalasia. Gastrointest Endosc. 1990; 36(2):123–6.
8. Richter JE, Boeckxstaens GE. Management of achalasia: surgery or pneumatic dilation. Gut. 2011; 60(6):869–76.
9. Allescher HD, Storr M, Seige M, Gonzales-Donoso R, Ott R, Born P, et al. Treatment of achalasia: botulinum toxin injection vs. pneumatic balloon dilation. A prospective study with long-term follow-up. Endoscopy. 2001;33(12):1007–17.
10. Khan AA, Shah SW, Alam A, Butt AK, Shafqat F, Castell DO. Pneumatic balloon dilation in achalasia: a prospective comparison of balloon distention time. Am J Gastroenterol. 1998;93(7):1064–7.
11. Gideon RM, Castell DO, Yarze J. Prospective randomized comparison of pneumatic dilatation technique in patients with idiopathic achalasia. Dig Dis Sci. 1999;44(9):1853–7.
12. Ott DJ, Donati D, Wu WC, Chen MY, Gelfand DW. Radiographic evaluation of achalasia immediately after pneumatic dilatation with the Rigiflex dilator. Gastrointest Radiol. 1991;16(4):279–82.
13. Ott DJ, Richter JE, Wu WC, Chen YM, Castell DO, Gelfand DW. Radiographic evaluation of esophagus immediately after pneumatic dilatation for achalasia. Dig Dis Sci. 1987;32(9):962–7.
14. Ciarolla DA, Traube M. Achalasia. Short-term clinical monitoring after pneumatic dilation. Dig Dis Sci. 1993;38(10):1905–8.
15. Karanicolas PJ, Smith SE, Inculet RI, Malthaner RA, Reynolds RP, Goeree R, et al. The cost of laparoscopic myotomy versus pneumatic dilatation for esophageal achalasia. Surg Endosc. 2007;21(7): 1198–206.
16. Imperiale TF, O'Connor JB, Vaezi MF, Richter JE. A cost-minimization analysis of alternative treatment strategies for achalasia. Am J Gastroenterol. 2000; 95(10):2737–45.
17. Vanuytsel T, Lerut T, Coosemans W, Vanbeckevoort D, Blondeau K, Boeckxstaens G, et al. Conservative management of esophageal perforations during pneumatic dilation for idiopathic esophageal achalasia. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2012;10(2):142–9.
18. Moonen A, Boeckxstaens G. Current diagnosis and management of achalasia. J Clin Gastroenterol. 2014;48(6):484–90.
19. Vaezi MF, Richter JE. Current therapies for achalasia: comparison and efficacy. J Clin Gastroenterol. 1998; 27(1):21–35.
20. Richter JE. Update on the management of achalasia: balloons, surgery and drugs. Expert Rev Gastroenterol Hepatol. 2008;2(3):435–45.
21. Bravi I, Nicita MT, Duca P, Grigolon A, Cantu P, Caparello C, et al. A pneumatic dilation strategy in achalasia: prospective outcome and effects on oesophageal motor function in the long term. Aliment Pharmacol Ther. 2010;31(6):658–65.
22. Boeckxstaens GE, Annese V, des Varannes SB, Chaussade S, Costantini M, Cuttitta A, et al. Pneumatic dilation versus laparoscopic Heller's myotomy for idiopathic achalasia. N Engl J Med. 2011;364(19):1807–16.
23. Zerbib F, Thetiot V, Richy F, Benajah D-A, Message L, Lamouliatte H. Repeated pneumatic dilations as long-term maintenance therapy for esophageal achalasia. Am J Gastroenterol. 2006;101(4):692–7.
24. Eckardt VF, Gockel I, Bernhard G. Pneumatic dilation for achalasia: late results of a prospective follow up investigation. Gut. 2004;53(5):629–33.
25. Vela MF. Management strategies for achalasia. Neurogastroenterol Motil Off J Eur Gastrointest Motil Soc. 2014;26(9):1215–21.
26. Eckardt VF, Aignherr C, Bernhard G. Predictors of outcome in patients with achalasia treated by pneumatic dilation. Gastroenterology. 1992;103(6):1732–8.
27. Hulselmans M, Vanuytsel T, Degreef T, Sifrim D, Coosemans W, Lerut T, et al. Long-term outcome of pneumatic dilation in the treatment of achalasia. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2010;8(1):30–5.
28. Farhoomand K, Connor JT, Richter JE, Achkar E, Vaezi MF. Predictors of outcome of pneumatic dilation in achalasia. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2004;2(5):389–94.

29. Robertson CS, Fellows IW, Mayberry JF, Atkinson M. Choice of therapy for achalasia in relation to age. Digestion. 1988;40(4):244–50.
30. Ghoshal UC, Kumar S, Saraswat VA, Aggarwal R, Misra A, Choudhuri G. Long-term follow-up after pneumatic dilation for achalasia cardia: factors associated with treatment failure and recurrence. Am J Gastroenterol. 2004;99(12):2304–10.
31. Pandolfino JE, Kwiatek MA, Nealis T, Bulsiewicz W, Post J, Kahrilas PJ. Achalasia: a new clinically relevant classification by high-resolution manometry. Gastroenterology. 2008;135(5):1526–33.
32. Kadakia SC, Wong RK. Graded pneumatic dilation using Rigiflex achalasia dilators in patients with primary esophageal achalasia. Am J Gastroenterol. 1993;88(1):34–8.
33. Walzer N, Hirano I. Achalasia. Gastroenterol Clin North Am. 2008;37(4):807–25. viii.
34. Stavropoulos SN, Friedel D, Modayil R, Iqbal S, Grendell JH. Endoscopic approaches to treatment of achalasia. Ther Adv Gastroenterol. 2013;6(2):115–35.
35. Lynch KL, Pandolfino JE, Howden CW, Kahrilas PJ. Major complications of pneumatic dilation and Heller myotomy for achalasia: single-center experience and systematic review of the literature. Am J Gastroenterol. 2012;107(12):1817–25.
36. Nair LA, Reynolds JC, Parkman HP, Ouyang A, Strom BL, Rosato EF, et al. Complications during pneumatic dilation for achalasia or diffuse esophageal spasm. Analysis of risk factors, early clinical characteristics, and outcome. Dig Dis Sci. 1993;38(10):1893–904.
37. Vaezi MF, Baker ME, Achkar E, Richter JE. Timed barium oesophagram: better predictor of long term success after pneumatic dilation in achalasia than symptom assessment. Gut. 2002;50(6):765–70.