Management of achalasia: surgery or pneumatic dilation

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ABSTRACT
Achalasia is an esophageal motility disorder of unknown cause, characterised by aperistalsis of the esophageal body and impaired lower esophageal sphincter relaxation. Patients present at all ages, primarily with dysphagia for solids/liquids and bland regurgitation. The diagnosis is confirmed by barium esophagram or endoscopy and is supported by esophageal manometry. Achalasia cannot be cured. Instead, our goal is to relieve symptoms, improve esophageal emptying and prevent the development of megaesophagus. The most successful therapies are pneumatic dilation and surgical myotomy. The advantages of pneumatic dilation include an outpatient procedure, minimal pain, return to work the next day, mild if any GERD, and can be performed in any age group and even during pregnancy. Pneumatic dilation does not hinder future myotomy, and all cost analyses find it less expensive than Heller myotomy. Laparoscopic myotomy with a partial fundoplication has the advantage of being a single procedure, dysphagia relief is longer at the cost of more troubling heartburn, and a myotomy may be more effective treatment in adolescents and younger adults, especially men. Over a two year horizon, the clinical success of pneumatic dilation and laparoscopic myotomy are comparable in a recent large European randomised trial. The prognosis for achalasia patients to return to near-normal swallowing and good quality of life are excellent, but few are “cured” with a single treatment and intermittent “touch up” procedures may be required.

INTRODUCTION
Achalasia is a rare disorder with an estimated prevalence of 0.5–1 per 100 000 per year without a clear age predilection. Patients mainly present with symptoms such as dysphagia for both solids and liquids, regurgitation of undigested food, respiratory complications (nocturnal cough and aspiration), chest pain and weight loss. These symptoms result from impaired peristalsis and deficient or absent relaxation of the lower oesophageal sphincter which leads to stasis of food in the oesophagus. Treatment aims to disrupt or dissect the lower oesophageal sphincter to enhance transport of the bolus into the stomach. In this review, we will discuss the current treatment options and propose a therapeutic algorithm.

PATHOPHYSIOLOGY
Why the enteric neurons of the oesophagus gradually disappear in patients with achalasia remains unclear. However, evidence is accumulating that an auto-immune response targeted against these neurons, triggered by an infectious agent, may be involved. This hypothesis fits with previous studies showing that, similar to auto-immune diseases, the risk of developing achalasia is associated with a certain genetic background, rendering individuals susceptible to develop an ‘abnormal’ immune response to an infectious event.

Genes that are associated with MHC genes, in particular class II MHC genes, are linked to autoimmunity. Several previous reports indicated a significant association of HLA-DR and DQ alleles with achalasia, and even showed that anti-neuronal antibodies were especially found in patients carrying the DQA1*0105 and DQB1*0605 alleles. Recently, Facco et al6 provided evidence that after HSV-I infection, the virus persists in the neurons of the oesophagus triggering a persistent immune-activation, consisting of infiltration of the ganglia with cytotoxic CD8+ T cells and circulating anti-neuronal antibodies.7

Diagnosis
The first diagnostic step is to rule out anatomical lesions using endoscopy or radiology. In early stages, both endoscopy and radiology may be completely normal. In advanced cases, endoscopy may reveal a dilated oesophagus with retained food and some increased resistance at the gastro-oesophageal junction. Radiological examination may show a typical ‘bird-beak’ image at the junction, with a dilated oesophageal body, sometimes with an air-fluid level and absence of an intragastric air bubble. Endoscopy is diagnostic in about 1/3 and radiology in about 2/3 of the patients; diagnostic certainty is provided by manometry in over 90% of cases.4 Manometry typically shows an aperistaltic oesophageal body, sometimes with elevated intraoesophageal pressure due to stasis of food and saliva, and incomplete relaxation of the lower oesophageal sphincter upon deglutition (residual pressure >10 mm Hg). In addition, resting tone of the lower oesophageal sphincter will often be elevated. Recently, Pandolfini et al8 proposed new diagnostic criteria using high resolution manometry with improved accuracy to diagnose achalasia and identifying three different types: type I (classical achalasia; no evidence of pressurisation), type II (achalasia with compression or compartmentalisation in the distal oesophagus >30 mm Hg) and type III (vigorous achalasia or two or more spastic contractions). Interestingly, the therapeutic response differs between the manometric subtypes suggesting that this classification may be useful to predict outcome.

Although most cases are idiopathic, it should be emphasised that Chagasic achalasia and...
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**Bullet points**

- Achalasia is a motor disorder of the oesophagus presenting with dysphagia for liquids and solids, regurgitation of undigested food, respiratory complications (nocturnal cough and aspiration), chest pain and weight loss.
- It is characterised by loss of enteric neurons resulting in aperistalsis and impaired relaxation of the lower oesophageal sphincter.
- The underlying mechanisms leading to neuronal loss are unknown but autoimmune mechanisms may be involved.
- Manometry is the ‘gold standard’ for diagnosing achalasia.
- To date, no treatment can restore oesophageal function; treatment is confined to reduce the pressure in the lower oesophageal sphincter.
- Laparoscopic myotomy and pneumatic dilation are the most commonly used treatments with comparable clinical efficacy.
- Since we cannot cure achalasia, recurrences after any form of treatment will occur with higher recurrence rates with longer follow-up periods.

**TREATMENT OF ACHALASIA**

No treatment can restore muscular activity to the denervated oesophagus in achalasia. Oesophageal aperistalsis is rarely reversed by any mode of treatment. Therefore, every treatment for achalasia is directed at reducing the gradient across the lower oesophageal sphincter (LOS) with three goals:

1. Relieving patient’s symptoms, especially dysphagia and bland regurgitation
2. Improving oesophageal emptying
3. Preventing the long-term development of megaesophagus.

In the modern era of achalasia treatment, disruption of the LOS is best accomplished by pneumatic dilation using the RigiFlex balloons or laparoscopic myotomy and, less effectively, by pharmacological agents, such as botulinum toxin injection or calcium channel blockers. Symptoms of dysphagia and regurgitation are the easiest to treat; chest pain relief is more unpredictable. Overall, using single or multiple modalities of treatment, over 90% of achalasia patients will do well. However, achalasia is never ‘cured’ by our current treatments, therefore, recurrences will occur with higher recurrence rates with longer periods of follow-up.

**PNEUMATIC DILATION**

Pneumatic dilation aims at disrupting the LOS by forceful stretching using air-filled balloons. This procedure has become standardised with the Microinvasive RigiFlex balloon system (Boston Scientific Corp, Massachusetts, USA) (box 1). These noncompliant polyethylene balloons are available in three diameters (3.0, 3.5 and 4.0 cm), on a flexible catheter placed over a guidewire at endoscopy. Briefly, pneumatic dilation is performed with the balloon placed across the LOS with the aid of radiopaque markers on the catheter shaft within the balloon. The position is confirmed by fluoroscopy or endoscopy. The actual balloon distension protocol varies across centres. Generally, the balloon is gradually inflated until the waist, caused by the nonrelaxing LOS, is flattened (figure 1). The pressure required is usually 7–15 psi of air, held for 15–60 s. Sometimes, multiple balloon distensions are done at the same setting. Some investigators only perform one dilation, but most use a graded dilation protocol starting with 3.0 cm, followed by 3.5 cm and then 4.0 cm balloon, in subsequent sessions.

A few European centres perform serial progressive dilations over several days, until the LOS pressure is below 10–15 mm Hg. In the USA, the need for further dilations is determined by persistence of symptoms often correlated with esophageal emptying studies at 4–6 week intervals after treatment. Pneumatic dilation is now routinely done in an outpatient setting, with the patient observed for up to 6 h, to ensure no complications have occurred.

In an updated review through 2009 of 1144 patients across 24 studies with an average follow-up of 37 months, RigiFlex pneumatic dilation gave good to excellent symptom relief in a graded fashion in 74%, 86% and 90% of patients treated with the 3.0, 3.5 and 4.0 cm balloon, respectively. Over a third of treated patients will experience symptom relapse over 4–6 years of follow-up. Long-term remission can be achieved in virtually all these patients treated by repeated pneumatic dilation according to an ‘on demand’ strategy, based on symptom recurrence (figure 2A). With the standardisation of the RigiFlex balloons, we are beginning to define risk factors for relapse after pneumatic dilation. These are mainly young age (<40 years), male gender, single dilation with a 3.0 cm balloon, post-treatment LOS pressure >10–15 mm Hg and poor esophageal emptying on an upright barium oesophagram (timed barium swallow).

Pneumatic dilation is the most cost-effective treatment for achalasia over a time period of 5 to 10 years. Gut 2011;60:869–876. doi:10.1136/gut.2010.212423
The only absolute contraindication to pneumatic dilation is poor cardiopulmonary status, or other co-morbid illnesses preventing surgery, should an oesophageal perforation occur. Pneumatic dilation can be safely done after a failed Heller myotomy, although larger diameter balloons are often required. Complications after pneumatic dilation are reported in up to 33% of patients with most complications being minor. Oesophageal perforation is the most serious complication after pneumatic dilation with an overall rate in experienced hands of 1.9% (range 0–16%). Most perforations occur during the first dilation, with difficulty in keeping the balloon in position during the procedure as potential risk factor. Although no other predictive factors have been identified, a recent European study reported more perforations when the first dilation was performed with a 35 mm balloon compared to a 30 mm balloon.

For small perforations and deep, painful tears, treatment may be conservative with antibiotics and total parenteral nutrition for days to weeks. However, surgical repair through thoracotomy is best for large, symptomatic perforations with extensive soilage of the mediastinum. Severe complications of GERD are rare after pneumatic dilation, but 15%–35% of patients have heartburn responding to proton pump inhibitors. Other usually minor complications after pneumatic dilation include chest pain, aspiration pneumonia, bleeding usually without a decrease in haemoglobin, transient fever, oesophageal mucosal tear without perforation and oesophageal haematoma.

**LAPAROSCOPIC HELLER MYOTOMY**

The first successful surgery for achalasia was performed in 1913, by the German surgeon Ernest Heller. The most popular operation now is a minimally invasive single anterior myotomy introduced by Pellegrini and co-workers in 1992. Initially performed through the chest, the overall success of the laparoscopic operation through the abdomen is superior to the thoracoscopic approach. Patients are usually hospitalised for less than 48 h and return to work within 2 weeks. Recent surgical improvements have included extending the

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**Box 1 General techniques for pneumatic dilation using Rigiflex balloon**

1. Every patient should be on a liquid diet for several days and fast for at least 12 h before endoscopy. Patients with mega-oesophagus may require oesophageal lavage with a large-bore tube.
2. Generally done as an outpatient in the morning. This ensures that appropriate x-rays and clinical observation do not go late into the evening, especially if surgical complications should occur.
3. Standard conscious sedation and upper endoscopy in the left lateral position.
4. Savary guidewire placed in the stomach and Rigiflex balloon passed over it.
5. The smallest balloon (3.0 cm) is usually used first. In patients with prior failed pneumatic dilations, young patients or after prior Heller myotomy, beginning with a 3.5 cm balloon may be preferred.
6. Accurate placement of the balloon by fluoroscopy with the patient in the supine position. Key is to carefully locate the balloon so the waist caused by the non-relaxing LOS impinges on the middle portion (figure 1). This is usually at or above the level of the diaphragm. In patients after Heller myotomy, the narrowing may be below the diaphragm.
7. Balloon is gradually distended until the waist, caused by the non-relaxing LOS is flattened. The pressure required is usually 7–15 psi of air, held for 15–60 s.
8. Patient repositioned in the left lateral position to minimize aspiration and the balloon carefully removed after deflating.
9. Overall post-procedure observation for 4–6 h to exclude perforation and evaluate for chest pain and fever. Patients discharged to home after drinking fluids without difficulty. Patients with pain during this observation period should be sent for gastrograffin followed by barium swallow to exclude oesophageal perforation.
10. Outpatient clinic follow-up in 2–4 weeks to assess symptoms and oesophageal function, especially emptying. Tests should include upright timed barium swallow and/or oesophageal manometry.
11. Persistent symptoms, especially if associated with poor oesophageal emptying or an LOS pressure above 10 mm Hg warrants repeat dilation with the next larger size balloon. This sequence is completed until symptom relief or failure to respond to the 4.0 cm balloon, at which point the patient is usually referred to surgery.

![Figure 1](https://example.com/figure1.png)

**Figure 1** X-ray images showing the Rigiflex balloon (dotted lines) positioned across the oesophagogastric junction. The waist in the balloon is clearly visible before (left panel) but has completely disappeared at the end (right panel) of the pneumodilation procedure.
myotomy 2–3 cm onto the proximal stomach to cut the gastric sling fibres, further decreasing LOS pressure and improving dysphagia.26 The more aggressive myotomy accentuates postoperative gastro-oesophageal reflux; therefore, the consensus is to add a full fundoplication (anterior Dor or posterior Toupet), to decrease this complication.27 Oesophageal myotomy lowers LOS pressure more consistently than pneumatic dilation. Depending on the distal extent of the myotomy into the cardia, LOS pressure is lowered by 55% to 75% with the remaining residual LOS pressure usually less than 10 mm Hg.26

In a recent review of 39 studies including nearly 3100 patients, the good-to-excellent symptom relief with laparoscopic myotomy was 89.5% with an average follow-up of 35 months.28 Younger patients, especially men and patients with higher LOS pressure, may benefit most from primary surgery. The former may be related to the greater tensile strength of the LOS in younger male patients; while the latter is postulated to reflect overall less severely damaged oesophageal muscle function with better bolus clearance once the obstruction is relieved.29 Patients failing pneumatic dilation or Botox treatment can be successfully treated with surgical myotomy,12 29 30 although some groups suggest a 15–21% lower success rate.31 32 Long-term studies suggest deterioration of surgical success over 5–11 years (figure 2B). Three groups have recently reported the long-term results of laparoscopic myotomy in 179 patients with remarkable consistency.29 33 34: 18% required pneumatic dilation, 5% Botox injection and 5–10% repeat myotomy or esophagectomy.

Recurrence of dysphagia after myotomy is usually the result of an incomplete myotomy, particularly on the gastric side. These patients may do well with a subsequent pneumatic dilation.29 Other factors include oesophageal scarring, obstruction by the fundoplication, megaesophagus, or complications of severe gastro-oesophageal reflux disease (GORD), including oesophagitis and peptic stricture. The role of a sigmoid megaesophagus (maximum diameter >6–9 cm with a horizontal configuration) contributing to the failure of myotomy is controversial. Several series suggest29 32 many of these patients will do poorly after surgery with up to 50% having persistent dysphagia. Others report35 36 these patients did as well after surgery as those with minimal dilation. Therefore, we recommend initial treatment with a laparoscopic myotomy, reserving oesophagectomy for the failures. Nevertheless, 2–5% of patients with a mega-oesophagus will eventually require an oesophagectomy.9 27 Surgical expertise is key, with the most complications occurring in the first 50 operations.38 Surgery is the most costly treatment for achalasia,19 but may be cost-effective if symptom relief reliably lasts at least 10 years.20

Surgical complications of laparoscopic myotomy include death (0.1%) and oesophageal perforation (7–15%).13 The most common long-term problem is chronic GORD and its sequelae, occurring overall in 18% of patients (range 5–55%).28 Most of these patients have reflux symptoms improving on proton pump inhibitors (PPIs); some oesophagitis, and rarely Barrett’s oesophagus.39 Secondary adenocarcinoma of the oesophagus has been reported after Heller myotomy. The addition of an incomplete fundoplication decreases, but does not eliminate, the complications of GORD as the reflux barrier seems to deteriorate over time.27

**PNEUMATIC DILATION VERSUS SURGICAL MYOTOMY?**

Ideally, the choice between two treatment options is based upon prospective, randomised comparative studies. Recently, studies comparing pneumatic dilation and laparoscopic myotomy have been reported. These studies are appearing at a critical time, when many gastroenterologists, especially in the US, have stopped performing pneumatic dilations and the laparoscopic technique has made surgical myotomy the preferred treatment for achalasia in many centres.

A large study from the Cleveland Clinic compared 106 patients treated with Rigiflex balloon and 73 patients undergoing primarily laparoscopic myotomy.12 The success of graded pneumatic dilation and myotomy, defined as dysphagia/regurgitation <3 times a week or freedom from alternative treatment, was similar; 96% versus 98% at 6 months, decreasing to 44% versus 57% at 6 years. Causes of symptom recurrence were uncompleted treatment of achalasia (96% after dilation vs 64% after myotomy) and complications of GORD (4% after dilation vs 36% after surgery).
Another method of addressing this issue is to study large population databases comparing treatment outcomes in typical practice settings. This was recently reported from Ontario, Canada, in a retrospective longitudinal study from 1991 to 2002. A total of 1461 adult patients were treated for achalasia; 81% had pneumatic dilation and 19% had surgical myotomy as their first procedure. The cumulative risk of any subsequent treatment (dilation, myotomy or esophagectomy) after 1, 5 and 10 years was 36.8%, 56.2% and 63.5% after initial pneumatic dilation versus 16.4%, 30.3% and 37.5% after initial myotomy (hazard risk: 2.37; CI 1.86 to 3.02). This risk difference was observed only when repeat pneumatic dilation was recorded as an adverse outcome. Since ‘on demand’ pneumatic dilation is the accepted approach to treating achalasia, this cannot logically be viewed as failure of dilation. The second series (26 dilation, 25 surgery) observed six dilation failures and one after surgery over at least 12 months follow-up. The difference was significant (p = 0.04) in the per protocol analysis, but not the intention-to-treat analysis (p = 0.09). Most recently, a five-country European achalasia trial randomised 94 patients to Rigiflex dilation (3.0 and 3.5 cm) and 106 to laparoscopic myotomy with Dor fundoplication. In this study, patients who had recurrent symptoms after pneumatic dilation were allowed to be retreated to a maximum of three series of pneumatic dilation. Both treatments had comparable success at 2 years: 92% for dilation and 87% for myotomy. Barium emptying and LOS pressure were both improved to a similar extent in both groups. Pre-existing daily chest pain, the height of the barium contrast column after 5 min and an oesophageal width of less than 4 cm before treatment were identified as predictors of treatment failure in Cox regression analysis. These data confirm that monitoring oesophageal emptying after treatment is a helpful tool to predict recurrence and to decide whether further dilation is required.

Why a diameter of the oesophagus less than 4 cm before treatment is associated with treatment failure is unclear, unless this may be indicative of vigorous achalasia, known to have a worse outcome. Although age was not a predictive factor of clinical success for either treatment, patients younger than the age of 40 presented more often with recurrent symptoms requiring redilation. This finding seems to support the proposal to preferentially treat younger patients (especially men) with laparoscopic Heller myotomy. Although longer follow-up is certainly required, this study indicates that both treatments are equally effective.

PHARMACOLOGICAL TREATMENTS

Lower oesophageal sphincter pressure can be transiently reduced by smooth muscle relaxants. Nitrates increase the nitric oxide concentration in smooth muscle cells, which subsequently increases cyclic guanosine monophosphate levels, resulting in muscle relaxation. Calcium is necessary for oesophageal muscle contractions and its action is blocked by calcium antagonists. Nitrates and calcium channel blockers decrease LOS pressure in a dose-dependent manner, with a maximum effect of 50%, thereby temporarily relieving dysphagia. These drugs are taken 15–30 min before meals, the improvement is usually incomplete and short lived, efficacy decreases with time, and side effects (headache, dizziness, pedal oedema) are common. As a result, there is infrequently a place for these drugs in the clinical management of achalasia.

Botulinum toxin (Botox) is a potent inhibitor of acetylcholine release from nerve endings. Botox cleaves SNAP-25, a cytoplasmic protein involved in the fusion of acetylcholine containing presynaptic vesicles with the neuronal plasma membrane. Exocytosis of acetylcholine is inhibited and temporary paralysis of the innervated muscle occurs. Botox counteracts the unopposed LOS stimulation by cholinergic neurons, helping to restore the LOS to a lower resting pressure. On average, Botox injections decrease LOS pressure by 50%, while partially improving oesophageal emptying. A total dose of 100 units of Botox is endoscopically injected into the LOS in multiple aliquots in a ring around the sphincter. Increasing the Botox dose to 200 units does not improve the success rate, but repeating a second 100 units in 1 month may improve its efficacy. Based on numerous studies, some placebo controlled, Botox markedly improves symptoms in approximately 75% of achalasia patients. However, 50% of patients relapse within 6 months, probably due to regeneration of the affected receptors.

Most initial responders improve with a second injection, but the response generally decreases with further injections, probably from antibody production to the foreign protein. Less than 20% of patients failing the initial Botox injection improve with a second injection. Older patients (>60 years) and those with vigorous achalasia, are more likely to have a sustained response, sometimes up to 1.5–2 years after a single Botox injection. Serial injections of Botox are required to give sustained relief, and comparison studies demonstrate long-term efficacy inferior to pneumatic dilation or myotomy. Botulinum toxin is contraindicated in patients with allergy to egg protein. It should be given carefully to patients receiving aminoglycosides, as these medications may potentiate the effect of the toxin. The most common side effect of Botox is chest pain in 16–25% of patients. Repeated Botox injections significantly hinder the dissection of the mucosal plane at myotomy, increasing the chance of mucosal
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Figure 3  Suggested algorithm for the treatment of achalasia. Healthy patients with a low risk of complications after surgery can be offered potentially definitive therapy with either pneumatic dilation or laparoscopic myotomy. Patients younger than 40 years may preferentially be referred to surgery, as they frequently need more repeat dilations than older subjects. Failures are best referred to oesophageal centres of excellence with expertise in pneumatic dilation, repeat myotomy, and oesophagectomy. High-risk patients, especially the elderly, are best treated with botulinum toxin injections, or alternatively pneumatic dilation, if the latter procedure is done at an oesophageal centre of excellence. (Updated from the American College of Gastroenterology Practice Guidelines: Diagnosis and management of achalasia. Am J Gastro 1999;4:3406—12, with permission.)

perforation. Serial Botox injections are more expensive than pneumatic dilation, because of the need for repeated injections. This treatment may have a cost advantage for patients living <2 years.

GENERAL RECOMMENDATIONS

Figure 3 is our suggested treatment algorithm for treating achalasia. Healthy patients with achalasia should be given the option of graded pneumatic dilation or laparoscopic Heller myotomy. The advantages of pneumatic dilation include an outpatient procedure, minimal pain, return to work the next day, mild, if any, GORD, and can be performed in any age group and even during pregnancy. Pneumatic dilation does not hinder future myotomy, and all cost analyses find it less expensive than Heller myotomy. Laparoscopic myotomy has the advantage of being a single procedure, dysphagia relief is longer at the cost of more troubling heartburn, and a myotomy may be more effective treatment in adolescents and younger adults, especially men. Myotomy is definitely the choice in noncooperative patients and those in whom pseudoachalasia cannot be excluded. Botox injection may be first-line treatment for elderly patients or those with severe co-morbid illness because it is safe, improves symptoms, and generally older patients require treatment no more frequently than once a year. However, pneumatic dilation is a reasonable alternative in high surgical risk patients if performed in high volume centres with surgical expertise should the rare perforation occur. Initial treatment of uncomplicated achalasia patients can be done by experienced community gastroenterologists and surgeons. Failures, particularly after surgery, should be referred to oesophageal centres of excellence with a multidisciplinary team experienced with pneumatic dilation, repeat myotomy and oesophagectomy. Nearly 90% of achalasia patients can have near normal swallowing and good quality of life, but few are ‘cured’ with a single treatment, and intermittent ‘touch up’ procedures may be required.

Future therapies

The therapies discussed so far reduce the resistance to flow at the oesophagogastric junction thereby improving oesophageal emptying, but in fact fail to correct the underlying neuron abnormality. As recent studies suggest auto-immune mechanisms underlying the disappearance of oesophageal enteric neurons, one might consider treating achalasia patients with immunomodulatory drugs. However, at the time of diagnosis the number of neurons has already decreased to a critical level leading to significant dysfunction and symptoms. Although this approach may theoretically prevent further disappearance of neurons, treatment will be started too late to restore function. An interesting alternative approach would be to transplant neuronal stem cells. In mice, neuronal stem cells injected in the pylorus survived and even expressed nitric oxide synthase. The advantage of such approach would be that both sphincter function and peristalsis may be restored. One of the main challenges clearly is to harvest enough stem cells to perform such transplantation. Interestingly, mesenchymal stem cells, which can be harvested from bone marrow, adipose tissue, and umbilical cord blood, among many other sources, possess several qualities which may be used to treat diseases of the central nervous system. Recently, it was even shown that neural stem cells can be...
isolated from mucosal biopsies, creating exciting possibilities to treat aganglionic diseases, including achalasia.\textsuperscript{53} Clearly, a lot of research remains to be done further exploring this approach.

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