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controversial topics in surgery

Achalasia of the cardia: dilatation or division?

The relative rarity of achalasia dictates that most patients will receive treatment delivered by an enthusiast with a predetermined preference. Mr Beckingham argues strongly in favour of laparoscopic myotomy, emphasising the benefits of division (as opposed to disruption) of the sphincter muscle under direct vision and the ability to perform sutured repair should a perforation occur. Professor Griffin is a confirmed dilator: he expounds the advantages of out-patient therapy, without the need for general anaesthesia, the relative ease of treatment repetition and the low risk of inadvertent perforation. With modern digital cameras and flat screen technology, the anatomical details seen by the surgeon are unsurpassed, enabling highly accurate division of the sphincter muscle to be obtained.

Last week, I operated on an external referral who had undergone three balloon dilatations, a laparoscopic Heller's myotomy and a further two dilatations postoperatively … before undergoing a re-do laparoscopic myotomy: nothing appears to be foolproof!

It would, however, appear reasonable to choose a treatment enabling immediate repair of any mucosal perforation rather than run the risk (albeit small) of an unseen perforation following dilatation. In elderly or unfit patients, BoTox or dilatation may be suitable, but younger patients may benefit from surgery. As both authors point out, it is highly unlikely that enough evidence will accrue to provide a definitive answer.

Ultimately, local preferences and expertise will dictate the treatment delivered.

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The case for balloon dilatation

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Achalasia is a global oesophageal motility disorder characterised by failure of oesophageal body peristalsis and a non-relaxing lower oesophageal sphincter. It results from a loss of ganglion cells in the myenteric plexus, although in the UK the precise aetiology is unknown. Consequently, there is no specific therapy available to treat the underlying disease process, or that will restore effective peristaltic function. Once the diagnosis is confirmed by a combination of barium studies, manometry, and video-endoscopy, treatment strategies are directed at reducing the pressure of the lower oesophageal sphincter to improve oesophageal emptying and control the disabling symptoms of dysphagia, regurgitation, and retrosternal pain.

The optimal treatment for achalasia is, therefore, the most safe, effective, and cost-efficient method of reducing lower oesophageal sphincter pressure. As achalasia is a life-long condition and the majority of patients present between 20 and 40 years of age, the treatment must also demonstrate durability. Based on these criteria, the standard therapy for achalasia is balloon dilatation of the lower oesophageal sphincter, rather than pharmacological manipulation or surgical myotomy. Unfortunately, the low incidence of 1 per 100,000 in the UK means that there is a paucity of randomised, controlled trials comparing these therapies with most data derived from relatively small case series from specialist centres, often with limited follow-up.

The aim of balloon dilatation is to disrupt the circular muscle fibres of the lower oesophageal sphincter rendering it incompetent. After balloon dilatation, good symptomatic relief has been reported for 76–96% of patients in large case series.[1–4] Although the technique is operator-dependent, by adhering to certain key principles, the results of specialist centres have been reproduced in wider clinical practice. Optimal results have been reported using Rigiflex Microvasive balloons; in order to minimise the risk of perforation, graded dilatation is recommended commencing initially with a 30-mm balloon.[4,5] This will provide symptomatic relief for the majority of patients with the remainder, often younger males, requiring a further dilatation with a 35-mm or, rarely, a 40-mm balloon. It is vital that, during the procedure, the lower oesophageal sphincter is accurately localised and that the balloon is positioned under image intensification. It is then slowly insufflated with contrast medium to efface fully the waist created by the sphincter and pressure maintained for a further 60 s (Fig. 1). It has been shown that

Figure 1 Endoscopic balloon dilatation being performed under image intensification showing effacement of the waist created by the lower oesophageal sphincter.

greater or more prolonged dilatation is associated with an increased risk of perforation without additional symptomatic benefit.[5]

The most serious risk of balloon dilatation is oesophageal perforation usually occurring above the cardia on the left posterolateral oesophageal wall. For this reason, a check endoscopy must always be performed immediately following dilatation and patients observed for 6 h post-procedure. A high perforation rate is often misleadingly cited as a reason against balloon dilatation, as with correct technique the risk of perforation is less than 3%.[2,4,6] Following balloon dilatation, there is a long-term risk of gastro-oesophageal reflux; however, symptomatic reflux actually occurs in less than 10% of patients and is usually well controlled by a proton pump inhibitor or even simple antacids.

Current evidence also suggests that balloon dilatation is a durable treatment. Many reports demonstrate that the benefit is maintained beyond 5 years and that, for the small number of patients with recurrent symptoms, further dilatation is effective.[2–4] In one long-term follow-up study over a 25-year period, 61 of 72 patients maintained their symptomatic relief with only four patients requiring a further dilatation during this time.[1] The measured decrease in lower oesophageal pressure is also maintained in the long-term as demonstrated on sequential manometric assessment.[7] Another study concluded that patients in remission at 5 years had an excellent chance of requiring no further therapy.[8] This study reported a higher relapse rate for those patients with a high post-dilatation sphincter pressure on manometry. This is unsurprising, as the lower oesophageal sphincter had clearly not been disrupted in these cases and repeat dilatation with a larger diameter balloon was indicated.

Pharmacotherapy has been unable to match the symptom benefit of balloon dilatation, as there is no specific smooth muscle agonist or antagonist targeting the lower oesophageal sphincter. The two most commonly used classes of drugs – calcium channel blockers and long-acting nitrates – do reduce sphincter tone but do not improve sphincter relaxation in response to swallowing. The response to such agents tends to be partial and short-lived, and there is good evidence that they are less effective than balloon dilatation.[9] More recently, there has been a vogue for endoscopic intrasphincteric injection of botulinum toxin to inhibit the release of acetylcholine from the presynaptic nerve endings. Although this temporarily reduces sphincter pressure, the technique has now largely been abandoned due to poor long-term results when compared to balloon dilatation.[10]

Surgical cardiomyotomy divides the sphincter muscle and has long been a treatment option for achalasia. It was, in fact, the disappointing long-term results of open Heller's cardiomyotomy that originally encouraged the development of endoscopic dilatation. Interest has been renewed by the introduction of a minimally invasive, laparoscopic technique, which has been shown in specialist centres to have a similar efficacy to balloon dilatation.[2] However, it is highly operator-dependent with a fine line between failing to divide the sphincter and causing perforation or inducing reflux symptoms. Proton pump inhibitor usage is certainly higher after Heller's myotomy and, although performing a simultaneous antireflux procedure may reduce this, it is not without complication.[2]

It has been suggested that cardiomyotomy should be used as a second-line treatment for patients who remain symptomatic following two balloon dilatations.[8] For this small group of patients, it is vital to demonstrate that symptoms relate to a hypertensive or non-relaxing sphincter and not to oesophageal body dysmotility or reflux before further intervention is contemplated. Further graded dilatation with a larger balloon may then provide good symptomatic relief. It is important to remember that no operation will improve oesophageal body function or prevent a small number of patients developing severe nutritional problems secondary to oesophageal failure. In fact, in our own experience, it is only those who have had long myotomies that have actually come to oesophagectomy for nutritional failure.

When all treatment options for achalasia were compared in a decision analysis, it was concluded that pneumatic dilatation was the favoured strategy if the probability of reflux with dilatation was less than 19% or if the effectiveness of laparoscopic Heller's myotomy was less than 90%.[11] All cost-analysis studies also support the superiority of pneumatic dilatation over laparoscopic Heller's myotomy or BoTox injection.[12] Current evidence, therefore, supports balloon dilatation as

the most safe, effective, cost-efficient, and durable method of reducing lower oesophageal sphincter pressure in the treatment of achalasia.

As surgeons, we are always keen to develop new ways of helping and treating our patients. The results of open Heller's myotomy encouraged the development of endoscopic dilatation and there is no reason to believe that the laparoscopic procedure, although less invasive, will significantly improve the long-term results. As interest in laparoscopic cardiomyotomy increases similar to any new procedure, it is useful to remember that one of the most important attributes of a surgeon is knowing when not to operate.

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Is pneumatic balloon dilatation justifiable any more?

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Achalasia is a disease of the oesophagus the exact cause of which still remains unresolved. It may be congenital or may be acquired, possibly as a result of viral infection. An almost identical clinical picture is found in Chagas' disease in South America where it is associated with *Trypanosomiasis* infestation. The pathophysiology of the disease is a failure of transmission in the myenteric nerves (Auerbach's plexus) controlling the relaxation of the tonically contracted lower oesophageal sphincter (LOS), resulting in a failure of relaxation of the sphincter. Patients present at any age from childhood to old age with difficulty in swallowing, regurgitation of old food and, occasionally, with chest pain. Halitosis is also a common complaint from partners. Elderly patients may present with recurrent chest infections from aspiration together with weight loss. Manometry is the most reliable diagnostic tool showing incomplete relaxation of the lower oesophageal sphincter and usually aperistalsis. A few patients show abnormal high amplitude pressure waves in the oesophageal muscle which is thought to precede complete failure of the oesophageal body pump to overcome the hypertonic LOS.

The essence of treatment is simple – to induce relaxation of the lower oesophageal sphincter. Although many techniques are described, they can be grouped into three approaches: (i) pharmacological manipulation of the LOS; (ii) dilatation or stretching of the LOS; and (iii) surgical disruption of the LOS.

Oral calcium channel antagonists (*e.g.* nifedipine) and GTN have been used to relax the LOS, neither with great success due to a limited capacity in some cases due to insufficient numbers of nerve endings, and a short duration of action requiring medication to be given immediately prior to eating. More recently, the neuromuscular blocking properties of botulinum toxin (BoTox) has been used to 'paralyse' the LOS. There are technical problems in establishing accurate and adequate placement of the injections into a circumferential muscle with an average length of 3 cm, but some patients receive transient benefit from BoTox injections. Unfortunately, as elsewhere in the body, the effects of BoTox are temporary and few patients receive benefit for more than a few months.[1]

Rigid dilatation of the oesophagus with a variety of implements dates back to at least 1645 and self bouginage with lead-filled weights became a popular management for achalasia in the late-19th and 20th centuries. Pneumatic dilatation with an inflatable bag was little better than rigid balloon dilatation and it was not until the late 1960s when forced pneumatic dilatation with a fixed volume (Gruntzig) balloon popularised by Vantrappen became established as a viable alternative to surgical procedures.[2] This technique allowed greater pressures to be applied to the LOS with the aim of rupturing the muscle fibres. It is now recognised that balloon dilatation is better than rigid dilatation; heavy sedation or general anaesthesia is required to allow adequate

pressures (typically 2–3 atm) to stretch, or ideally rupture, the lower oesophageal muscle. Accurate placement across the LOS should be confirmed by image intensifier where waisting of the balloon by the LOS can be seen and corrected.

The results of pneumatic balloon dilatation are variable not least because of the rarity of the condition (incidence 1–2 per million in the UK) in relation to the number of gastroenterologists looking after these patients, with typically each gastroenterologist seeing only 1 or 2 patients. This has resulted in a failure to establish a standard dilatation protocol. In a survey in Nottingham performed in 1999, 12 gastroenterologists had performed 20 balloon dilatations over a 2-year period using 11 different protocols (author's unpublished data). There is a natural tendency to minimise the pressures required in dilating the LOS because of the risk of perforating the oesophagus. This results in stretching of the smooth muscle of the LOS rather than rupture. In the author's experience of > 30 cardiomyotomies following previous dilatations, only one patient had any evidence of oesophageal muscle disruption. As would be expected in a technique that predominantly involves stretching and dilatation of the muscle over a variable portion of its length, results are variable with most series reporting 'good-to-excellent' outcomes in 40–70% of patients.[3,4] The main risk of the procedure is oesophageal perforation requiring emergency surgery with its associated morbidity and mortality. Three recent publications list the incidence as 7–15%.[3] Data on outcome following oesophageal perforation remain difficult to find with most presenting > 24 h when contamination and sepsis significantly increase the mortality. Records from the US National Registry show a 2–5% mortality associated with pneumatic dilatation for achalasia.[5]

Surgery for achalasia (cardiomyotomy) aims to divide the LOS; it was first described and popularised by the German surgeon, Ernest Heller, in 1913. Heller originally described two incisions on the lower part of the oesophagus extending onto the proximal stomach wall. This procedure was later modified by Zaoijer who established that single myotomy was all that was necessary to achieve adequate swallowing, and this has become the standard procedure. The operation was carried out originally through a thoracotomy and only latterly (with the increase in gastroesophageal surgeons with a predominantly general surgical training rather than thoracic background) has the transabdominal route become more popular. The advantages of the transabdominal procedure over the thoracic approach are the avoidance of post-thoracotomy pain (present in 40% of patients) and the ability to add a fundoplication to prevent gastroesophageal reflux (GORD) which develops in 30–40% of patients post-cardiomyotomy. Antireflux procedures can be easily performed with either anterior (Dor) or posterior (Toupet) fundoplications.

Sir Alf Cuschieri first performed the laparoscopic approach in 1991.[6] The thoracoscopic approach was described in 1992 but has not been widely practised as it suffers the limitations inherent to the open thoracotomy technique, *i.e.* difficulty in assessing and extending the myotomy onto the stomach wall from the thorax, and the inability to perform an anti-reflux procedure. Perforations in the oesophageal mucosa are rare, easily visible and can be primarily sutured and patched with an anterior fundoplication. Gastroscopy can be performed intra-operatively to aid dissection or simply check adequacy of the myotomy. There are now many series of laparoscopic cardiomyotomy published with 'good-to-excellent' outcomes of 90–95% and zero mortality.

There have, to date, been no randomised trials of cardiomyotomy versus balloon dilatation nor are there likely to be so. Even the most fanatical balloon dilator must concede that a precise division of the sphincter under direct vision will achieve a more total destruction of the LOS than an intra-oesophageal balloon could hope to achieve. The question then becomes one of mortality and quality-of-life outcomes. With the advent of the laparoscopic approach, mortality rates are as low or lower than in the balloon dilatation group. In surgical series where fundoplication is not performed, reflux rates are higher than those in balloon dilation series simply because the dilation group do not have adequate LOS disruption, as recognised by the higher persistence and recurrence of dysphagia. However, the addition of an antireflux procedure prevents reflux and does not appear to inhibit the resolution of dysphagia. Could we adopt an approach of dilatation first and, if unsuccessful, proceed to surgery? Although dilatation rarely results in rupture of the oesophageal muscle, it does result in submucosal and peri-oesophageal fibrosis which makes dissection of the submucosal plane in particular more difficult and a number of studies have shown an increase in perforation in this sub-group.[7,8] Some authors have reported similar problems following BoTox injections.[8]

With truly informed consent and the increasing development of loco-regional centres offering laparoscopic cardiomyotomy, it is difficult to justify offering patients a lesser procedure. Which patient would swap the benefits of a controlled myotomy under direct vision together with the potential to combine with an antireflux procedure to reduce side-effects still with a discharge home the next day when compared with a blind dilatation of their lower oesophagus with higher failure rates, higher recurrence rates, higher risks of perforation, higher incidence of reflux and an increased mortality? With the exception of the very frail and infirm patient (who should probably receive BoTox injections into the LOS), all patients with achalasia should be offered a laparoscopic cardiomyotomy as definitive treatment.

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