Benign surgical diseases of the gastro-oesophageal junction

John S Hammond
Emily Tucker
Mark Fox
Ian J Beckingham

Abstract
This article discusses the investigation and management of gastro-oesophageal reflux disease, para-oesophageal hernias and achalasia.

Keywords Achalasia; fundoplication; gastro-oesophageal reflux; para-oesophageal hernia

Oesophageal anatomy (Figure 1)

The oesophagus is a muscular tube, approximately 25 cm long extending from the pharynx to the gastro-oesophageal junction (GOJ). It is made up of striated muscle proximally and smooth muscle (arranged in inner circular and outer longitudinal layers) distally. The oesophagus is lined by non-keratinized stratified squamous epithelium up to the Z-line where it gives way to columnar epithelium. The lower oesophageal sphincter (LOS) extends over the lower 2–4 cm of the oesophagus as it enters the stomach. Together with the diaphragmatic crura and the oblique angle of entry of the oesophagus into the stomach this provides the lower oesophageal physical barrier to reflux.

John S Hammond *PhD MRCS is a Lecturer in Gastrointestinal Surgery at Nottingham Digestive Diseases Centre, NIHR Biomedical Research Unit, Nottingham University Hospital, Queen's Medical Centre, Nottingham, UK. Conflicts of interest: none.*

Emily Tucker *MRCP is a Clinical Research Fellow in the Department of Gastroenterology, Nottingham Digestive Diseases Centre, NIHR Biomedical Research Unit, Nottingham University Hospital, Queen's Medical Centre, Nottingham, UK. Conflicts of interest: none.*

Mark Fox *MD MA MRCP is Associate Professor in the Department of Gastroenterology, Nottingham Digestive Diseases Centre, NIHR Biomedical Research Unit, Nottingham University Hospital, Queen's Medical Centre, Nottingham, UK. Conflicts of interest: none.*

Ian J Beckingham *DM FRCS Consultant Laparoscopic and Upper GI Surgeon at Nottingham Digestive Diseases Centre, NIHR Biomedical Research Unit, Nottingham University Hospital, Queen's Medical Centre, Nottingham, UK. Conflicts of interest: none.*

Anatomy of the oesophagus

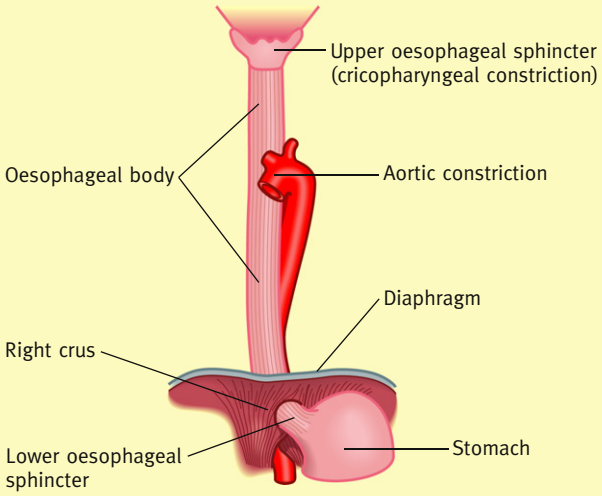

Figure 1

Gastro-oesophageal reflux disease

Gastro-oesophageal reflux is a normal physiological process that occurs in all individuals. It is defined as pathological when chronic acid reflux causes unacceptable symptoms, or pathological changes within the oesophagus (oesophagitis or Barrett's metaplasia). Gastro-oesophageal reflux disease (GORD) is the most common benign disease of the oesophagus with up to 25% of the population experiencing symptoms suggestive of GORD on a regular basis.[1,2] Box 1 lists the symptoms of GORD. The most common is heartburn, classically described as retrosternal discomfort, often after eating, and in some individuals related to body position (lying flat or bending forward). A proportion of patients have atypical symptoms including cough, pharyngitis, voice change and tooth decay. There may also be an association

Symptoms of gastro-oesophageal reflux disease

- Heartburn
- Epigastria or chest pain (masquerading as cardiac pain)
- Dysphasia
- Odynophagia (painful swallowing)
- Otalgia
- Chronic nausea
- Regurgitation of food ('vomiting')
- Poor dentition and dental caries
- Halitosis
- Cough/wheeze/aspiration

Box 1

Differential diagnosis of gastro-oesophageal reflux disease

- Carcinoma of the oesophagus
- Peptic ulcer disease
- Benign stricture of oesophagus
- Scleroderma
- Achalasia
- Non-gastrointestinal causes

Box 2

with functional gastrointestinal disorders. Establishing a clear history for GORD is therefore not always straightforward.[3]

Investigation of GORD

Upper gastrointestinal endoscopy

Upper gastrointestinal (GI) endoscopy is usually the first investigation undertaken in patients with GORD that has failed to resolve with simple measures. Endoscopy may demonstrate active oesophagitis or evidence of chronic reflux (strictures or Barrett's metaplasia) as well as identifying other pathology (evidence of dysmotility, malignancy or peptic ulcer disease) that may mimic symptoms. Box 2 lists the differential diagnosis for patients with symptoms of GORD.

Manometry and pH-impedance studies (Figure 2)

Confirmation of the diagnosis of GORD and the correlation of symptoms with reflux episodes is usual before proceeding to surgery. pH studies and manometry have been the gold standard test for confirming the diagnosis and excluding other conditions which can masquerade as GORD. High-resolution oesophageal manometry (HRM) is an advanced beyond conventional manometry.[4] It allows quicker and more intuitive analysis of oesophageal function and is more sensitive to oesophageal dysmotility and dysfunction including both functional (e.g. achalasia) and structural (e.g. stricture) outflow obstruction from the oesophagus. It can be combined with impedance measurements to allow simultaneous assessment of motility and bolus transport that can clarify the significance of subtle motility disorders.

Both tests involve the placement of a fine nasogastric probe to a point just distal to the gastro-oesophageal junction. The HRM probe has an increased number of sensors (approximately one per centimetre). Recordings of the pressures generated at different levels within the oesophagus are then measured during dry, wet and sometimes solid swallows to measure peristalsis, LOS function and response to physiological challenge. In addition, HRM facilitates accurate placement of pH probes for reflux monitoring.

After manometry has been performed a pH probe is placed 5 cm above the upper border of the LOS which detects acid reflux events and measures oesophageal acid exposure. Patients are required to retain the probe for a 24-hour period whilst maintaining their normal diet and performing their normal daily activities. A key part of this study is the reporting of reflux symptoms by patients during the monitoring period so that the association between reflux and symptoms can be established.

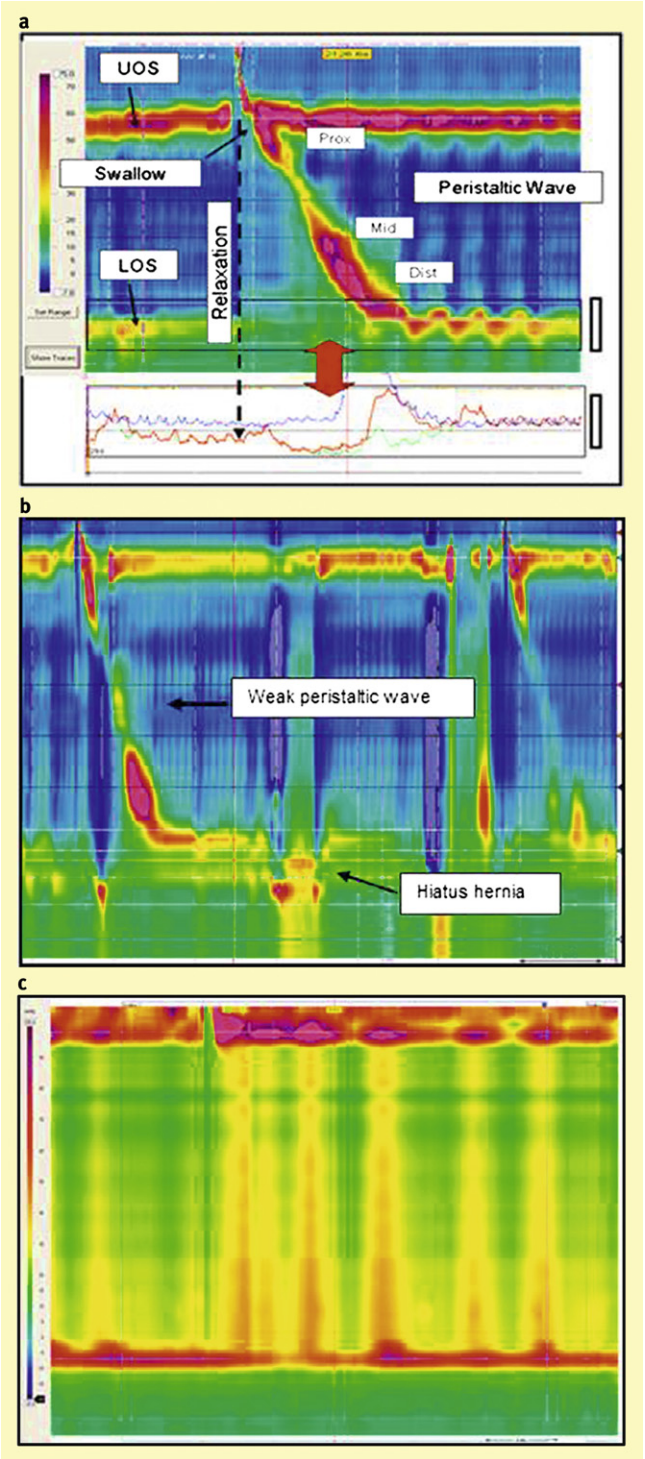

Figure 2 (**a**) Normal high-resolution manometry spatiotemporal plot: Water swallow with normal peristalsis and appropriate lower oesophageal sphincter relaxation. (**b**) Gastro-oesophageal reflux disease: Hypotensive oesophageal peristalsis on swallowing and hiatus hernia present. 24-hour pH recording confirmed prolonged acid exposure. (**c**) Achalasia (type II): no lower oesophageal sphincter relaxation on swallowing and pan-oesophageal pressurization. LOS, lower oesophageal sphincter; UOS, upper oesophageal sphincter.

Impedance monitoring is a relatively new technique that is complimentary to pH studies. It detects electrical resistance between a series of detectors within the oesophagus. These measurements differentiate between gas and liquid contents and allow the detection of weakly acid and non-acid 'volume' reflux that are not detected by pH studies. This increases the number of reflux events that can be associated with patient symptoms especially regurgitation, belching and cough after meals that often do not contain much acid. In addition, for patients tested on PPIs, this technology provides direct evidence that symptoms that persist during acid suppression are related to 'volume reflux'. Thus the combination of pH with impedance monitoring increases the sensitivity of reflux studies. Moreover recent evidence demonstrates that patients whose symptoms are related to persistent non-acid reflux are likely to improve with surgery.

In patients with GORD manometry often reveals a weak or unstable oesophago-gastric reflux barrier that facilitates reflux. Motility and clearance may be normal but is often impaired in patients with more severe GORD leading to prolonged acid exposure after reflux events. The diagnosis of GORD is established by demonstrating either increased oesophageal acid exposure or a positive association between reflux events and patient symptoms.

Contrast studies

Contrast studies provide useful information regarding the anatomy and peristaltic function of the oesophagus. However they are not of benefit in the initial assessment of patients with GORD. The main value of a contrast swallow is in the assessment of para-oesophageal hernias and for the investigation of patients with complications or recurrent symptoms following surgery.

Management of GORD

Patients with mild GORD achieve resolution of their symptoms with simple measures including antacids, reduction in alcohol intake, cessation of smoking and avoidance of spicy and irritant foods. Patients with more marked symptoms will generally require treatment with a proton pump inhibitor (PPI). Whilst many patients achieve excellent resolution of their symptoms with a PPI, their symptoms often recur upon cessation of the drug. Other patients (particularly those with volume reflux) will develop breakthrough symptoms. In these patients a lifelong PPI or dose escalation is usually required. Patients with failure of medical management or who do not wish to undergo lifelong treatment with a PPI may be considered for surgery.[5,6]

Selecting patients for anti-reflux surgery

Anti-reflux surgery may be offered to patients with complicated or uncomplicated GORD. Ideally patients should have objective evidence of GORD, that is oesophagitis on endoscopy or abnormal physiology (regular and/or prolonged reflux episodes of acid (pH <4) into the oesophagus with an overall pH <4 for >4% of the measured period). In patients with GORD with associated respiratory complications (aspiration, recurrent pneumonia) or peptic stricture formation, anti-reflux surgery should be considered provided that the patient is fit for surgery. The role of anti-reflux surgery in patients with Barrett's metaplasia is currently the subject of a number of controlled trials.

In patients with uncomplicated GORD a number of factors predict good outcomes following anti-reflux surgery. There must be good correlation of symptoms with measured acid reflux episodes (symptom index >50%). In patients with failed medical management those that had a partial or temporary response to PPIs do better than those who have had no response. Impaired oesophageal function secondary to reflux damage or the presence of a large hiatus hernia often improves following anti-reflux surgery.[7] Other predictors of good outcome following anti-reflux surgery are a high oesophageal acid exposure time and patients without evidence of functional gastrointestinal disease.

Anti-reflux surgery

The principle of anti-reflux surgery is to restore the mechanical barrier to gastro-oesophageal reflux and in doing so relieve symptoms and remove the need for long-term medical therapy. Laparoscopic fundoplication has become the standard surgical procedure for GORD. The higher morbidity of open abdominal and transthoracic surgery, together with the now widespread availability and good results from the laparoscopic procedure have resulted in these open procedures being seldom used except for revision fundoplication. Recent endoscopic innovations have poor durability and inferior outcomes compared to over laparoscopic fundoplication.[8]

Laparoscopic fundoplication is usually performed using a four- or five-port technique (Figure 3). After port placement the liver is retracted anteriorly to allow access to the gastro-oesophageal junction (GOJ). The first step involves opening the pars flaccida of the lesser omentum and identifying the right crus, taking care to avoid injury to an accessory left hepatic artery when present (10%). The phreno-oesophageal ligament (the peritoneal layer that binds the anterior surface of the oesophagus and stomach to the diaphragm) is divided up to the hiatus, down

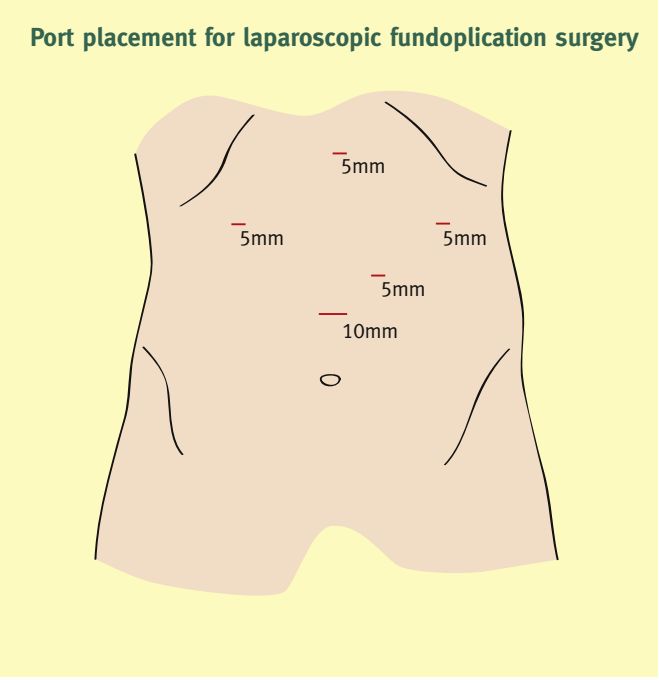

Figure 3

the left crus and across the cardia of the stomach onto the diaphragm and upper pole of the spleen. A window is made behind the oesophagus and stomach and anterior to the crura. The GOJ is now mobile and any hiatus hernia present will be evident and can be reduced by freeing up any attachments within the thoracic cavity. The crural hiatus is closed loosely around the oesophagus using non-absorbable sutures.

Having mobilized the lower oesophagus the next step is to wrap the gastric fundus around the oesophagus. This can be in the form of a partial fundoplication, either anterior or posterior, or a total fundoplication. If total fundoplication is to be performed often an oesophageal bougie (50–60 French) is used to prevent over-tightening of the wrap. The fundoplication is then fixed to the oesophagus to prevent slippage. Figure 4 illustrates three types of fundoplication. Recent meta-analyses comparing partial with complete fundoplication demonstrate that partial fundoplication is associated with reduced rates of post-operative dysphagia and other complications when compared with complete fundoplication.[9] Some centres advocate tailoring the degree of fundoplication to the results of preoperative oesophageal manometry studies to reduce the incidence of post-operative dysphagia; however this approach has not yet been supported by controlled trials.[10]

Complications of anti-reflux surgery

The complications following anti-reflux surgery can be divided into early and late complications. Conversion to an open procedure should occur infrequently (<5%). Bleeding from the major gastric vessels, liver trauma or splenic capsule tears may occur intraoperatively. Postoperatively all patients will experience a degree of dysphagia. Patients should take a liquid/semi-solid diet for the first few weeks following the operation until oesophageal function returns to normal. As long as liquids are tolerated reassurance is all that is necessary at this stage. Persistent dysphagia to solid food occurs in less than 5% of patients at 3 months after surgery. Conversely patients with oesophageal dysmotility secondary to reflux damage or outflow obstruction due to a hiatus hernia often report improved swallowing following anti-reflux surgery.[5] If severe or persistent symptoms are present then clinical assessment should be with barium swallow to look for excessive hiatal tightness or fibrosis, or malposition or slippage of the wrap. In the absence of gross structural pathology oesophageal manometry may be required. HRM may facilitate differentiation of functional and structural causes of symptoms. If symptoms are debilitating and persist, revisional correction with conversion to a partial fundoplication or undoing of the wrap altogether may be necessary. Popularization of the partial fundoplication has seen a marked reduction in these problems without a significant failure to control reflux.

Failure to control reflux symptoms or recurrence of symptoms after a period of previously good control may be associated with wrap disruption. There may be a history of vomiting or retching prior to the return of symptoms. In some cases this represents a loosening of the wrap and settles down after a period of a few weeks, in other cases there may be permanent wrap disruption requiring revisional surgery. The first line of investigation is a contrast swallow and if this is normal repeat manometry and pH/impedance studies. There is a tendency for patients to be placed back onto PPIs when they develop any upper GI symptoms following anti-reflux surgery on the assumption that it is a recurrence of the GORD. In some of these patients the transient hold-up of food at the wrap is experienced as pain ('sensitive oesophagus') which can be verified with pH studies if necessary. A liquid soothing anti-acid preparation (e.g. Gaviscon) is more appropriate in this group of patients and symptoms may settle with time.

'Gas bloat' or 'post-fundoplication' syndrome is the trapping of intra-luminal gas within the fundus of the stomach. It usually presents with epigastric pain and an inability to release gas from the upper gastrointestinal tract via the mouth. Abdominal bloating and increased rectal flatus are also common (30–50% of patients). Some (2–3%) patients experience diarrhoea. Patients should be warned of these symptoms pre-operatively particularly if other functional symptoms are present as these may be exacerbated.

Para-oesophageal hernia

A hiatus hernia is an abnormal protrusion of the stomach into the thoracic cavity through the oesophageal hiatus. They occur in approximately 10% of the population and are more common in

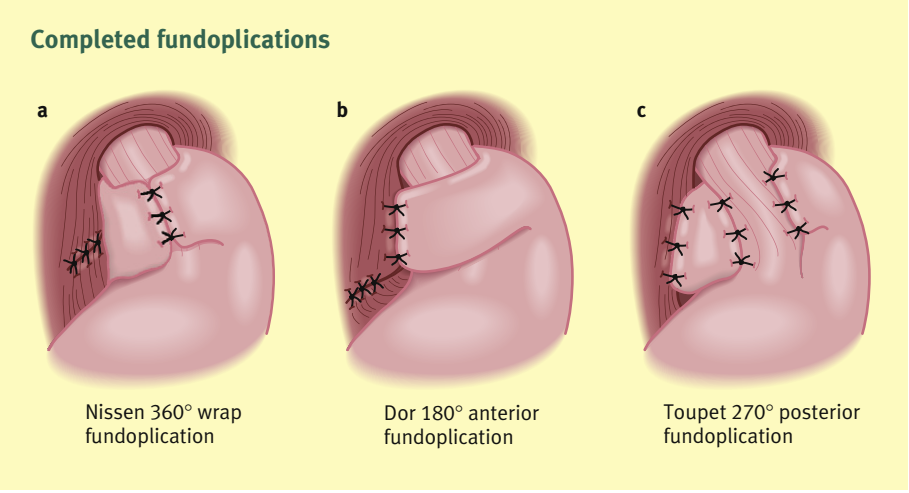

Figure 4

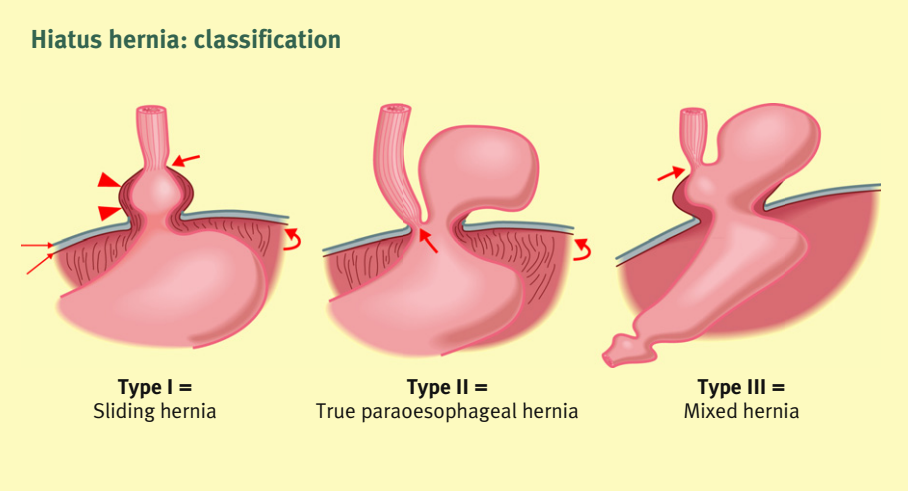

Figure 5

later life. Hiatal hernias are classified as types I–IV (Figure 5). Type I hiatal hernias (sliding) are the most common (90%). Types II (true para-oesophageal), III (mixed) and IV (where additional viscera and organs migrate into the hernia sac) are grouped as para-oesophageal hernias.

Frequently para-oesophageal hernias are asymptomatic. As they become larger patients may develop a range of symptoms (Box 3). Large parao-esophageal hernias may also present as an emergency in a critically ill patient with a history of dysphagia and proximal intestinal obstruction secondary to volvulus of the stomach.

In the elective setting endoscopy, manometry, pH-impedence studies and contrast studies can be used to characterize the para-oesophageal hernia prior to embarking on surgery. In the emergency setting diagnosis of acute gastric volvulus can be made by chest X-ray (demonstrating a mediastinal air-fluid level), supported by cross-sectional imaging as required.

The principles of para-oesophageal hernia repair are to reduce the contents of the hernia (stomach, distal oesophagus and other viscera), reduce the hernia sac fully and repair the crural defect, usually followed by an anti-reflux procedure to help prevent recurrence. Repair of even large crural defects can usually be achieved with sutures and only rarely requires reinforcement with a biological mesh. This may be undertaken as an open or laparoscopic procedure. To date there have been no prospective studies comparing outcomes following open versus laparoscopic para-oesophageal hernia repair.

In the emergency setting the initial focus of management should be to resuscitate the patient. An attempt should then be made to decompress the stomach by passage of a nasogastric tube. If this is unsuccessful the patient should proceed to theatre for emergency repair. If decompression is successful repair may be undertaken once the patient has stabilized. Principles of repair of an acute gastric volvulus of a para-oesophageal are to reduce the hernia, release the volvulus, resect non-viable tissue, repair the crural defect and fix the stomach to the diaphragm. Again this may be undertaken in conjunction with an anti-reflux procedure.

Achalasia

Achalasia is a motility disorder of the oesophagus characterized by the failure of the gastro-oesophageal junction to relax. This results in progressive dysphagia and secondary oesophageal body failure. Box 4 summarizes the symptoms and signs of achalasia. Clinical examination reveals little other than weight loss and sometimes halitosis.

Upper GI endoscopy is the first-line investigation of any patient with new onset dysphagia. In early achalasia the oesophagus

Symptoms of a para-oesophageal hernia

- Heartburn
- Epigastric/chest pain
- Post-prandial fullness
- Regurgitation
- Dysphagia
- Symptoms of anaemia secondary to gastric erosions
- Proximal intestinal obstruction

Box 3

Symptoms of achalasia

- Dysphagia
- Regurgitation of undigested food
- Halitosis
- Chest pain
- Dyspepsia
- Chronic cough/nocturnal aspiration
- Weight loss

Box 4

appears normal and whilst the lower oesophageal sphincter may appear tight it rarely occludes passage of the gastroscope. Only in the later stages will evidence of a dilated tortuous oesophagus containing food debris be present. A contrast swallow will show dilatation of the oesophagus, aperistalsis of the main oesophageal body and hold-up at the LOS with the classic 'bird's beak' appearance. Manometry provides the definitive diagnosis. Three distinct patterns of disease are recognized on HRM that correspond to treatment outcome. Classic 'type I' achalasia with no pressure activity in the oesophagus that requires aggressive treatment to relieve outflow obstruction, 'type II' in which pan-oesophageal bolus compression is present (see Figure 2c) and is associated with favourable outcome to dilatation or surgery and 'type III' in which oesophageal spasm is present and responds poorly to treatment focused on the LOS alone.

Management of achalasia

The ideal aim when treating achalasia is to reduce the LOS pressure sufficiently to relieve obstruction without permitting reflux. In practice treatment seeks to destroy the lower oesophageal sphincter to prevent hold up to the passage of oesophageal contents. The restoration of an entirely normal swallow in patients with achalasia is rarely achieved because the oesophageal body never recovers its peristalsis, and thus food boluses rely upon propulsion in the top third of the oesophagus and pharynx (striated, unaffected muscle) and gravity to enter the stomach. It is important that patients are aware of this prior to embarking on treatment.

The two mainstays of treatment for achalasia are laparoscopic cardiomyotomy with or without partial fundoplication and endoscopic therapy (pneumatic dilatation or botulinum toxin injection). Historically surgery for achalasia was restricted to patients in whom endoscopic therapy had failed, due to morbidity associated with the open technique. Following the introduction of laparoscopic cardiomyotomy, which provides all the established benefits of minimally invasive surgery (reduced post-operative pain and length of hospital stay) together with excellent long-term resolution of dysphagia, surgical treatment of achalasia has become the first-line treatment in many institutions.[11] The main consideration when undertaking laparoscopic cardiomyotomy is whether to combine it with an anti-reflux procedure. Current evidence demonstrates that with addition of a partial fundoplication (either anterior or posterior) the incidence of post-cardiomyotomy GORD is reduced without a significant elevation in ongoing dysphagia when compared with cardiomyotomy alone.

Endoscopic pneumatic balloon dilatation is the most effective non-surgical treatment for achalasia. Using a high pressure balloon, the oesophageal sphincter is stretched with the aim of inducing a permanent tear of the muscle without disruption of the mucosa. It is associated with a risk of oesophageal perforation (3–4%) and development of reflux in some patients.[12] The majority of studies show poor longer term outcomes in terms of swallowing (due to inadequate stretching of the muscle), a higher incidence of reflux and an overall higher mortality than surgery. Endoscopic botulinum toxin injection can reduce LOS pressure but does not provide good long-term resolution of dysphagia and may stimulate fibrosis at the LOS, which can interfere with future attempts at cardiomyotomy.

Laparosocopic cardiomyotomy

Using the same four- to five-port laparoscopic approach as above (see Figure 3), the GOJ is mobilized and at the point where the oesophagus meets the cardia of the stomach the circular smooth muscle fibres of the LOS a 6–8 cm myotomy is performed by dividing first the longitudinal then the circular

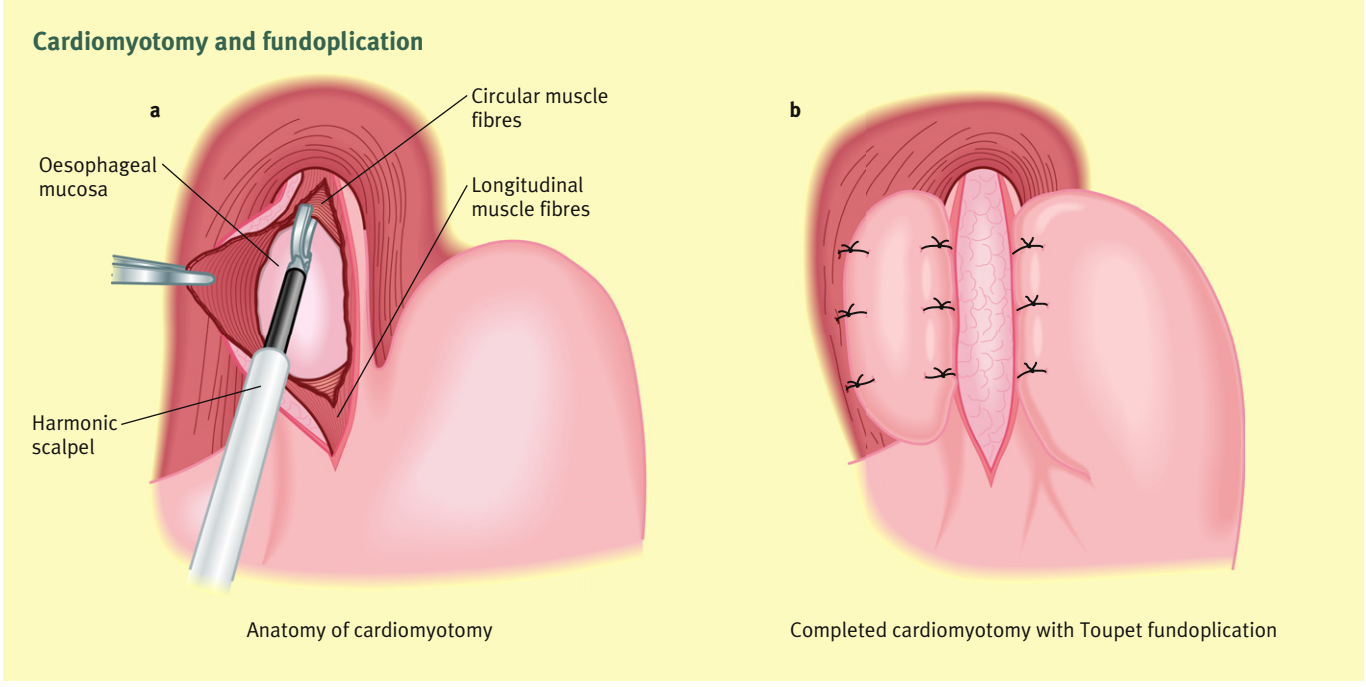

Figure 6

smooth muscle fibres in the midline, with the myotomy extended proximally on to the thoracic oesophagus and distally onto the cardia of the stomach to ensure complete division of the LOS (Figure 6). The LOS itself is not identifiable as a separate anatomical structure. Cardiomyotomy reduces the LOS pressure sufficiently to allow passage of food, but may be associated with long-term reflux. Cardiomyotomy performed in combination with partial fundoplication therefore provides the best long-term symptom relief with low morbidity for patients with achalasia.[11] ◆

REFERENCES

1 Dent J, Brun J, Fendrick AM, et al. An evidence-based appraisal of reflux disease management the Genval Workshop Report. *Gut* April 1998; **44**(suppl 2)**:** S1—16.

2 Klauser AG, Schindlbeck NE, Müller-Lissner SA. Symptoms in gastro-oesophageal reflux disease. *Lancet* 1990; **335:** 205—8.

3 Moayyedi P, Talley NJ. Gastro-oesophageal reflux disease. *Lancet* June 2006; **367:** 2086—100.

4 Fox MR, Bredenoord AJ. Oesophageal high-resolution manometry: moving from research into clinical practice. *Gut* March 2008; **57:** 405—23.

5 Dellon ES, Shaheen NJ. Persistent reflux symptoms in the proton pump inhibitor era: the changing face of gastroesophageal reflux disease. *Gastroenterology* 2010; **139:** 7—133.

6 Mahon D, Rhodes M, Decadt B, et al. Randomized clinical trial of laparoscopic Nissen fundoplication compared with proton-pump inhibitors for treatment of chronic gastro-oesophageal reflux. *Br J Surg* 2005; **92:** 695—9.

7 Beckingham IJ, Cariem AK, Bornman PC, Callanan MD, Louw JA. Oesophageal dysmotility is not associated with poor outcome after laparoscopic Nissen fundoplication. *Br J Surg* 1998; **85:** 1290—3.

8 Schiefke I, Zabel-Langhennig A, Neumann S, Feisthammel J, Moessner J, Caca K. Long term failure of endoscopic gastroplication (EndoCinch). *Gut* June 2005; **54:** 752—8.

9 Broeders JAJL, Mauritz FA, Ahmed Ali U, et al. Systematic review and meta-analysis of laparoscopic Nissen (posterior total) versus Toupet (posterior partial) fundoplication for gastro-oesophageal reflux disease. *Br J Surg* 2010; **97:** 1318—30.

10 Broeders JA, Sportel IG, Jamieson GG, et al. Impact of ineffective oesophageal motility and wrap type on dysphagia after laparoscopic fundoplication. *Br J Surg* 2011; **98:** 1414—21.

11 Campos GM, Vittinghoff E, Rabl C, et al. Endoscopic and surgical treatments for achalasia: a systematic review and meta-analysis. *Ann Surg* 2009 Jan; **249:** 45—57.

12 Beckingham IJ. Achalasia of the cardia: dilatation or division? Is pneumatic balloon dilatation justifiable anymore? *Ann R Coll Surg Engl* 2006 Jan; **88:** 11—2.

FURTHER READING

Broeders JAJL, Mauritz FA, Ahmed Ali U, et al. Systematic review and meta-analysis of laparoscopic Nissen (posterior total) versus Toupet (posterior partial) fundoplication for gastro-oesophageal reflux disease. *Br J Surg* 2010; **97:** 1318—30.

Campos GM, Vittinghoff E, Rabl C, et al. Endoscopic and surgical treatments for achalasia: a systematic review and meta-analysis. *Ann Surg* 2009 Jan; **249:** 45—57.

Moayyedi P, Talley NJ. Gastro-oesophageal reflux disease. *Lancet* June 2006; **367:** 2086—100.