GASTRO-OESOPHAGEAL REFLUX

د.محمد محمود حبش

Aetiology

Normal competence of the gastro-oesophageal junction is maintained by the LOS. *This is influenced by both its physiological function and its anatomical location relative to the diaphragm and the oesophageal hiatus*. Most episodes of physiological reflux occur during postprandial transient lower oesophageal sphincter relaxations (TLOSRs).

In the early stages of GORD, most pathological reflux occurs as a result of an increased number of TLOSRs rather than a persistent fall in overall sphincter pressure.

In more severe GORD, LOS pressure tends to be generally low, and this loss of sphincter function seems to be made worse if there is loss of an adequate length of intra-abdominal oesophagus.

The absence of an intra-abdominal length of oesophagus results in a sliding hiatus hernia. Sliding hiatus hernia is associated with GORD and may make it worse but, as long as the LOS remains competent, pathological GORD does not occur.

Many GORD sufferers do not have a hernia, and many of those with a hernia do not have GORD.

In Western societies, GORD is the most common condition affecting the upper gastrointestinal tract. This is partly due to the declining incidence of peptic ulcer as the incidence of infection with *Helicobacter pylori* has reduced as a result of improved socioeconomic conditions along with a rising incidence of GORD in the last 20–30 years. The cause of the increase is unclear, but may be due in part to increasing obesity. The strong association between GORD, obesity and the parallel rise in the incidence of adenocarcinoma of the oesophagus represents a major health challenge for most Western countries

Clinical features

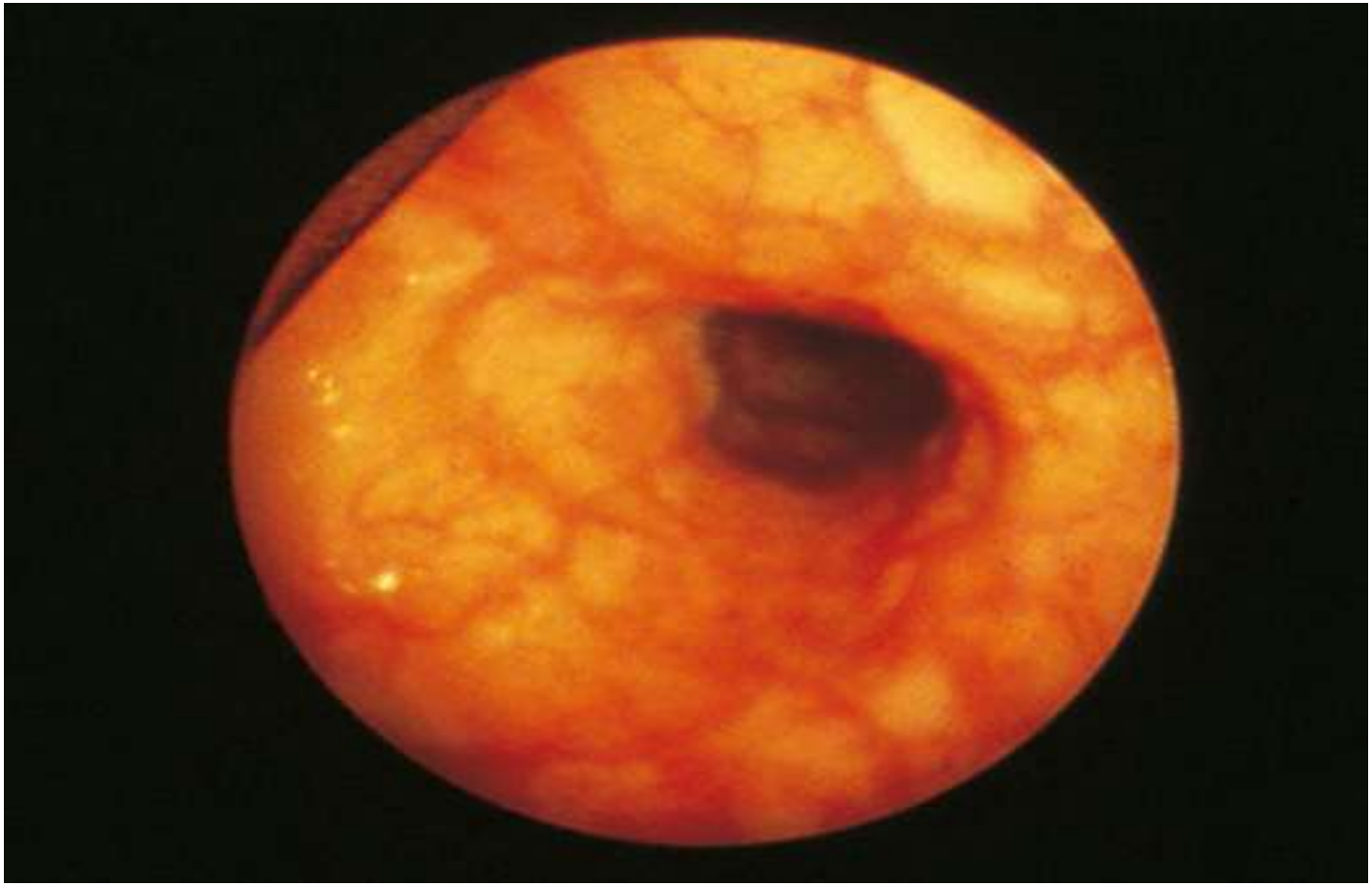
The classical triad of symptoms is retrosternal burning pain (heartburn), epigastric pain (sometimes radiating to the back) and regurgitation. *Most patients do not experience all three*. Symptoms are often provoked by food, particularly those that delay gastric emptying (e.g. fats, spicy foods). As the condition becomes more severe, gastric juice may reflux to the mouth and produce an unpleasant taste often described as ‘acid’ or ‘bitter’. Heartburn and regurgitation can be brought on by stooping or exercise.

Some patients present with less typical symptoms such as angina-like chest pain, pulmonary or laryngeal symptoms. Dysphagia is usually a sign that a stricture has occurred, but may be caused by an associated motility disorder.

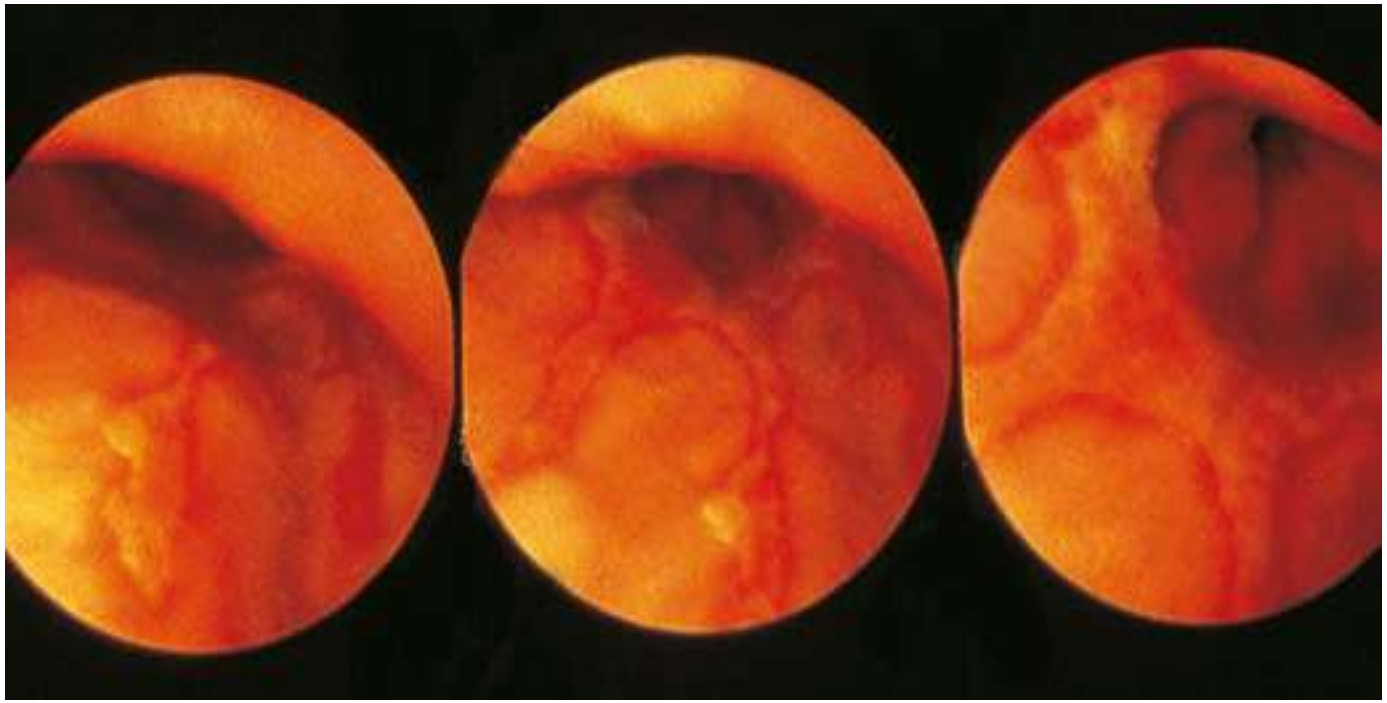
Diagnosis

In most cases, the diagnosis is assumed rather than proven, and treatment is empirical. Investigation is only required when the diagnosis is in doubt, when the patient does not respond to a proton pump inhibitor (PPI) or if dysphagia is present. The most appropriate examination is endoscopy with biopsy. In patients with atypical or persistent symptoms despite therapy, oesophageal manometry and 24-hour oesophageal pH recording may be justified to establish the diagnosis and guide management. Barium swallow and meal examination gives the best appreciation of gastro-oesophageal anatomy.

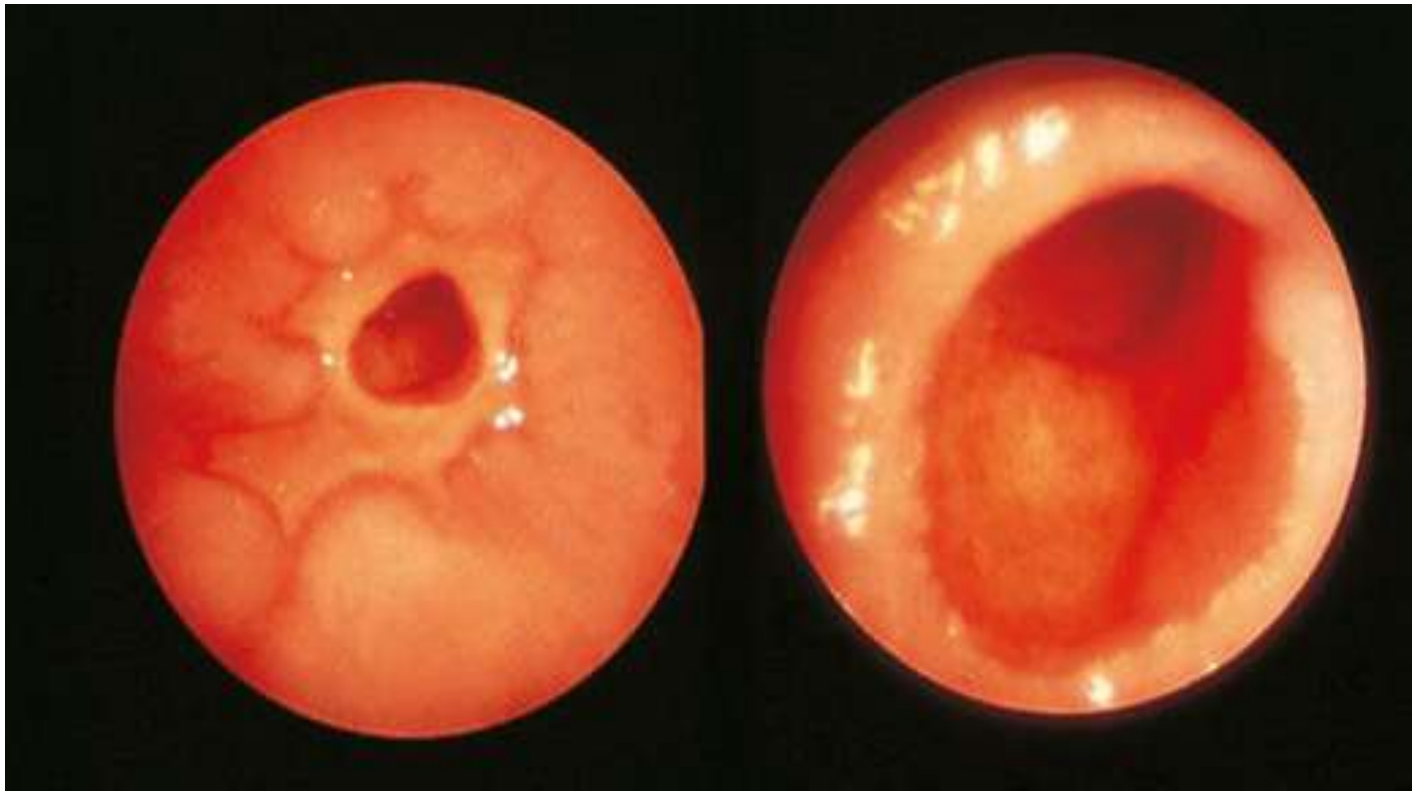
The endoscopic appearance of the normal squamous mucosa in the body of the oesophagus.



Reflux oesophagitis.



Benign stricture with active oesophagitis (left) and healed with columnar epithelium (right).



Management of uncomplicated GORD

Medical management

Simple measures that are often neglected include advice about weight loss, smoking, excessive consumption of alcohol, tea or coffee, the avoidance of large meals late at night and a modest degree of head-up tilt of the bed. *Tilting the bed has been shown to have an effect that is similar to taking an H2-receptor antagonist*. *The common practice of using additional pillows has no significant effect*.

PPIs are the most effective drug treatment for GORD. *Given an adequate dose for 8 weeks*, most patients have a rapid improvement in symptoms (within a few days), *and more than 90 per cent can expect full mucosal healing at the end of this time*.

Proton pump inhibitor therapy is also important in patients with reflux-induced strictures, resulting in significant prolongation of the intervals between endoscopic dilatation.

Surgery

Endoscopic treatments

A number of endoscopic treatments have been tried in the last ten years that attempt to augment a failing LOS.

Surgical treatments

The indication for surgery in uncomplicated GORD is essentially patient choice.

There are many operations for GORD, but they are virtually all based on the *creation of an intra-abdominal segment of oesophagus*, crural repair and some form of wrap of the upper stomach (fundoplication) around the intra-abdominal oesophagus.

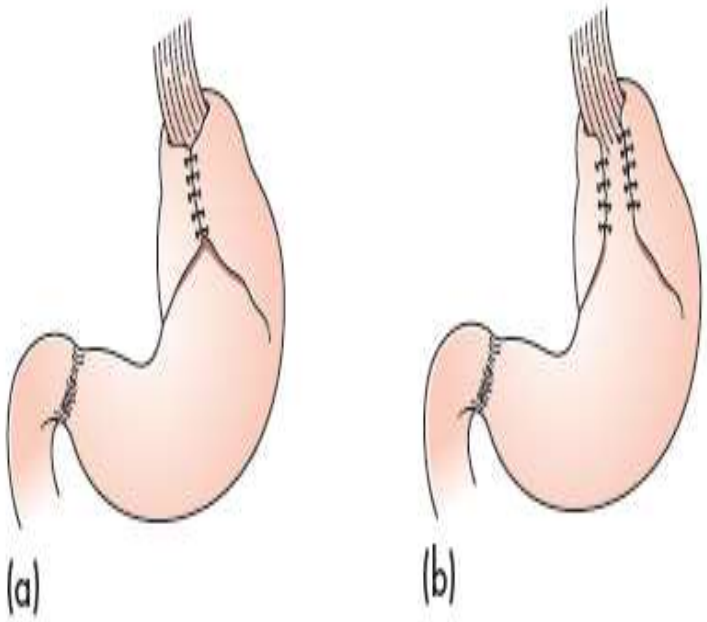


Figure 62.25 (a) Total (Nissen) fundoplication; (b) partial fundoplication (Toupet).

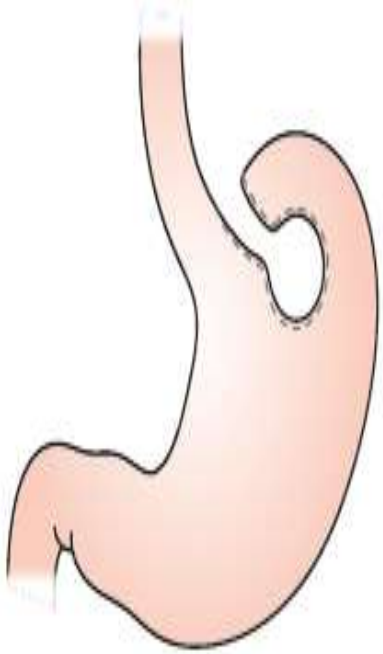


Figure 62.26 Collis gastroplasty to produce a neo-oesophagus around which a Nissen fundoplication is done. The operation may be performed by a laparoscopic as well as an open approach using circular and linear staplers.

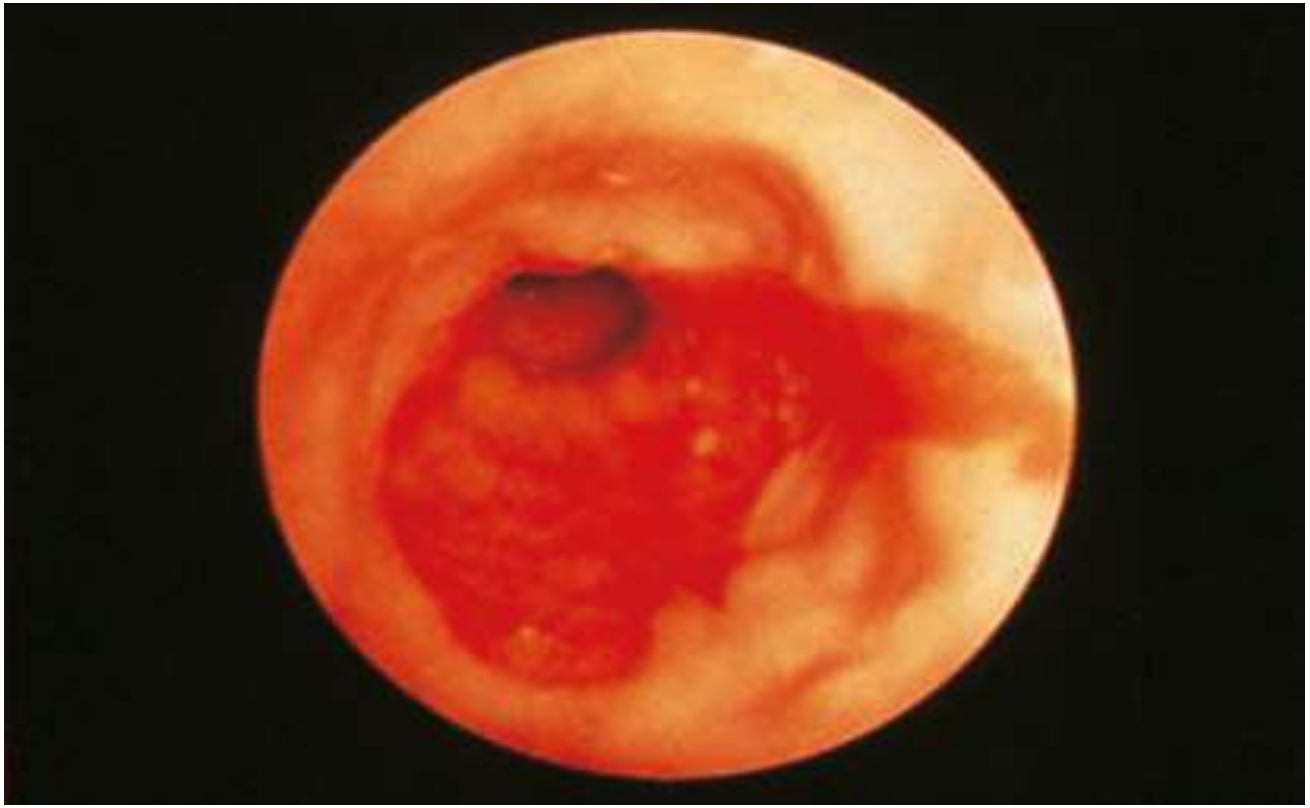
Complications of GERD

- Stricture
- Oesophageal shortening
- Barrett’s oesophagus

Barrett's oesophagus is a metaplastic change in the lining mucosa of the oesophagus in response to chronic gastro esophageal reflux.

This adaptive response involves a mosaic of cell types, probably beginning as a simple columnar epithelium that becomes 'specialised' with time. *The hallmark of 'specialised' Barrett's epithelium is the presence of mucus secreting goblet cells (intestinal metaplasia).*

Barrett's oesophagus



In Barrett's oesophagus, the junction between squamous oesophageal mucosa and gastric mucosa moves proximally. It may be difficult to distinguish a Barrett's oesophagus from a tubular, sliding hiatus hernia during endoscopy, as the two often coexist or where the visible Barrett's segment is very short. *When intestinal metaplasia occurs, there is an increased risk of adenocarcinoma of the oesophagus which is about 25 times that of the general population*. Patients who are found to have Barrett's oesophagus may be submitted to regular surveillance endoscopy with multiple biopsies in the hope of finding dysplasia or *in situ* cancer rather than allowing invasive cancer to develop and cause symptoms.

Barrett's oesophagus may be diagnosed if there is any intestinal metaplasia in the oesophagus. The relative risk of cancer probably increases with increasing length of abnormal mucosa.

The following terms are widely used:

- classic Barrett's (3 cm or more columnar epithelium);
- short-segment Barrett's (less than 3 cm of columnar epithelium);
- cardia metaplasia (intestinal metaplasia at the oesophagogastric junction without any macroscopic change at endoscopy).

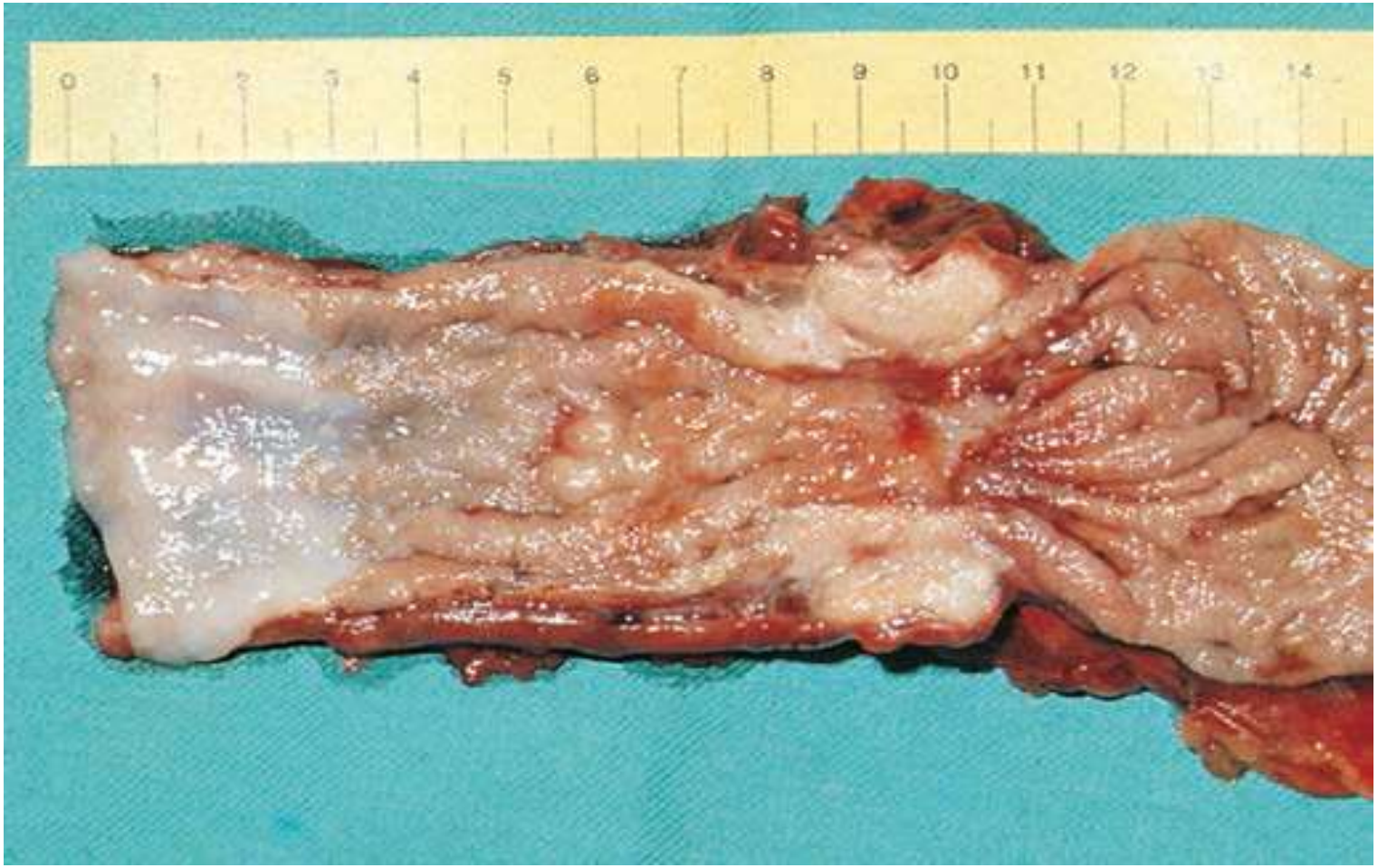
When Barrett's esophagus is discovered, the treatment is that of the underlying GORD. There has been considerable interest in recent years in endoscopic methods of ablating Barrett's mucosa in the hope of eliminating the risk of cancer development. *Laser, photodynamic therapy, argon-beam plasma coagulation and endoscopic mucosal resection (EMR) have all been used*.

In conjunction with high-dose PPI treatment or an antireflux operation, these endoscopic methods can result in aneosquamous lining.

There is no evidence yet that any of these methods is reliable in eliminating cancer risk.

damage to the oesophageal wall may cause stricturing.

The macroscopic appearances of an adenocarcinoma in Barrett's oesophagus



Achalasia

Pathology and aetiology.

Achalasia (Greek ‘failure to relax’) is uncommon, but merits prominence because it is reasonably understood and responds to treatment. It is due to loss of the ganglion cells in the myenteric (Auerbach’s) plexus, the cause of which is unknown.

In South America, chronic infection with the parasite *Trypanosoma cruzi* causes Chagas’ disease, which has marked clinical similarities to achalasia.

Achalasia differs from Hirschsprung's disease of the colon because the dilated oesophagus usually contains few ganglion cells, whereas the dilated colon contains normal ganglion cells proximal to a constricted, aganglionic segment.

Histology of muscle specimens generally shows a reduction in the number of ganglion cells (and mainly inhibitory neurones) with a variable degree of chronic inflammation. In so-called 'vigorous achalasia', which may be an early stage of the disease, there is inflammation and neural fibrosis, but normal numbers of ganglion cells.

The classic physiological abnormalities are a non-relaxing LOS and absent peristalsis in the body of the oesophagus. With time, the oesophagus dilates and contractions disappear, so that the oesophagus empties mainly by the hydrostatic pressure of its contents. This is nearly always incomplete, leaving residual food and fluid. The gas bubble in the stomach is frequently absent.

The ‘megaoesophagus’ becomes tortuous with a persistent retention oesophagitis due to fermentation of food residues and this may account for the increased incidence of carcinoma of the oesophagus.

Pseudoachalasia is an achalasia-like disorder that is usually produced by adenocarcinoma of the cardia but has also been described in relation to benign tumours at this level. It has been presumed that the inability of the sphincter to relax is linked to the loss of body peristalsis, but other cancers outside the oesophagus (bronchus, pancreas) have also been associated with pseudoachalasia.

Clinical features

•The disease is most common in middle life, but can occur at any age.

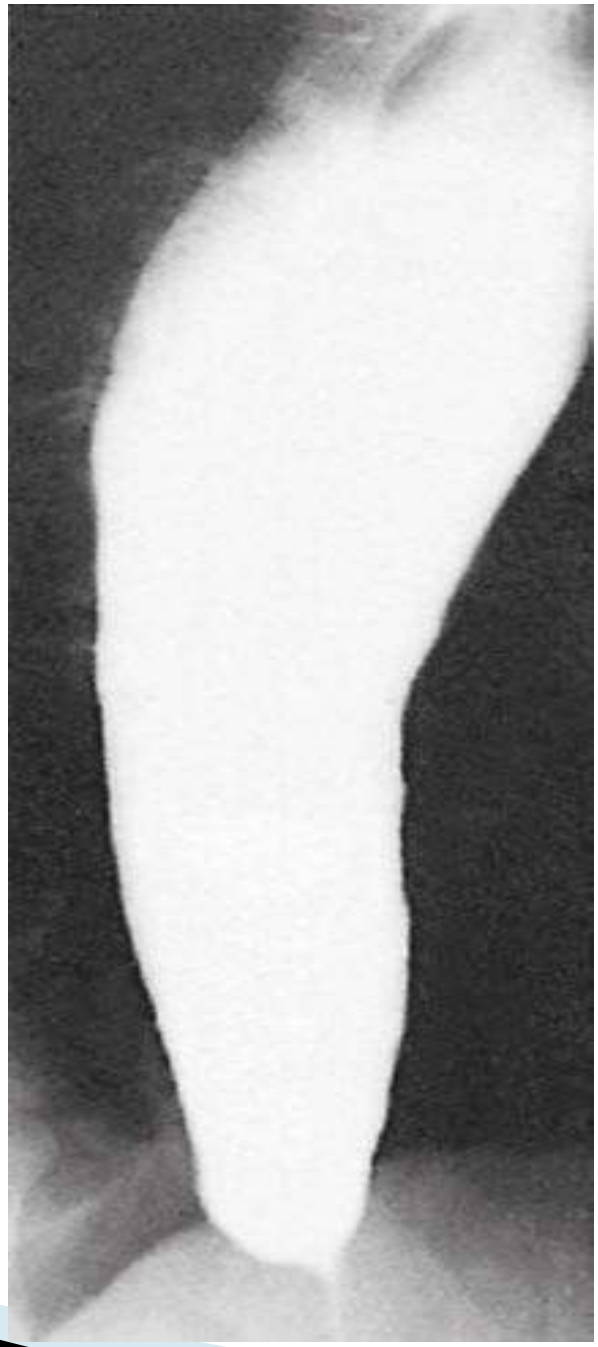
• It typically presents with dysphagia, although pain (often mistaken for reflux) is common in the early stages.

•Patients often present late and, having had relatively mild symptoms, remain untreated for many years.

•Regurgitation is frequent, and here may be overspill into the trachea, especially at night.

Diagnosis

- Achalasia may be suspected at endoscopy by finding a tight cardia and food residue in the oesophagus.
- Barium radiology may show a tapering stricture in the distal oesophagus, often described as a 'bird's beak'.
- The gastric gas bubble is usually absent.
- A firm diagnosis is established by oesophageal manometry. *Classically, the LOS does not relax completely on swallowing, there is no peristalsis and there is a raised resting pressure in the oesophagus*.



Treatment

Alone among motility disorders, achalasia responds well to treatment.

The two main methods are forceful dilatation of the cardia and Heller's myotomy.