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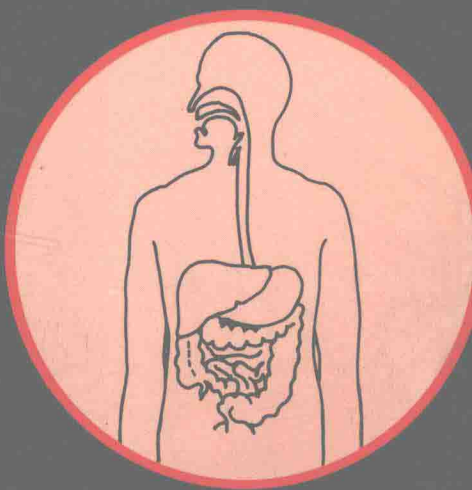
CHURCHILL LIVINGSTONE MEDICAL TEXT

A CONCISE TEXTBOOK OF GASTROENTEROLOGY

M.J.S. LANGMAN



SECOND EDITION

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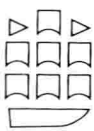

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Preface

The many changes in patterns of gastroenterological practice since the first edition of this book have meant that the text has had to be completely re-written. The book is longer, but hopefully more informative and better illustrated.

I am grateful to my secretary Mrs Janice Avery for her typing, and to Mr G. Lyth for drawing the figures. I am also grateful to the following for permission to reproduce material: *Medical Education International* and Mr J. Alexander Williams for Figure 2.7, and Messrs Edward Arnold for Figures 8.1, 11.1 and 11.3 and for Table 2.1, all taken from *Epidemiology of Chronic Digestive Disease* by myself.

Nottingham, 1982 M.J.S.L.

Contents

1

Oesophageal disorders

DYSPHAGIA

Difficulty and/or pain on swallowing are ominous symptoms which should never be ignored. By taking a careful history it is usually possible to diagnose the likely site and cause of the abnormality.

Questioning and examination should be structured to distinguish lesions of the oropharynx from those of the gullet itself and to assess the rate of progression of the lesion. Table 1.1 lists some of the commoner abnormalities and some of their distinguishing features.

Table 1.1 Causes of dysphagia

Pharyngeal	
Cough and choking on swallowing: with nasal regurgitation.	Neuromuscular disorders (Bilateral motor neurone lesions, motor neurone disease, myasthenia gravis, etc.)
without nasal regurgitation.	Pharyngeal pouch.
Difficulty in starting to swallow.	Functional, motor neurone disease.
Feeling of obstructed throat.	Functional, thyroid swelling, postcricoid carcinoma.
Oesophageal	
Solids may stick, slow progress.	Benign stricture
Solids stick, then liquids, rapid progress.	Oesophageal or high gastric carcinoma, mediastinal neoplasm.
Irregular hold up of food in chest, slow progress.	Achalasia, diffuse muscle spasm, systemic sclerosis, Chagas disease.
Chest pain on swallowing.	Oesophagitis, diffuse muscle spasm, achalasia, sometimes carcinoma.

Pharyngeal pouch

Pathology

This, the commonest variety of pharyngo-oesophageal diverticulum, tends to occur in men more often than women. Its cause is uncertain but it probably arises secondary to a rise in pharyngeal

pressure due to incoordination or spasm of the cricopharyngeal sphincter.

Clinical features
Symptoms and signs include regurgitation of food, often on bending or lying down, dysphagia for fluids rather than solids, and occasionally the patient notices a neck swelling.

Diagnosis and treatment
Barium swallow examination including lateral films is diagnostic. Treatment is by surgical excision of the pouch and sphincter section.

Other diverticula of the oesophagus
Mid or lower oesophageal diverticula can develop as a result of distortion due to juxta-oesophageal inflammatory disease, or can occur just proximal to the hiatus as epiphrenic diverticula. They are usually found incidentally and seldom cause symptoms: treatment (if needed) is by excision.

Hiatus hernia and oesophagitis
Hiatal herniae can be divided into three common types. (Fig. 1.1)

(*a*) Sliding: in which the cardia slides up into the chest, taking its attached ligaments and peritoneal reflexion with it.
(*b*) Rolling: in which a pouch of fundus of the stomach rolls up beside the cardia and into the chest.
(*c*) Mixed sliding and rolling hernia: a combination of both the first two.

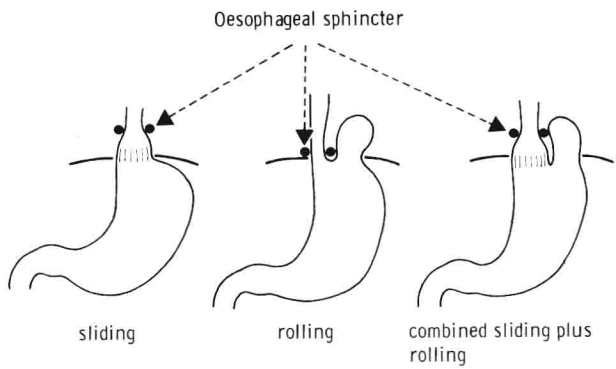

Fig. 1.1 Types of hiatal herniae

All types can become fixed in the chest, or can remain free so that the hernia is only produced by appropriate posturing.

In addition a rare variety of hiatus hernia occurs in association with a congenital short oesophagus.

All types may be associated with oesophageal reflux; but this is in general less common when the gastro-oesophageal junction remains fixed below the diaphragm.

The precise mechanisms preventing reflux are uncertain, but the following have been emphasized.

(*a*) *Physiological cardiac sphincter.* Manometric pressure studies have clearly demonstrated the presence of a sphincter zone at the bottom of the gullet which is sensitive to a variety of stimuli, for instance contracting harder in response to a rise in intra-abdominal pressure. This sphincter zone has been found to be less active in those with symptomatic oesophageal reflux, and has recently been considered to be of greater rather than, as formerly, lesser importance.

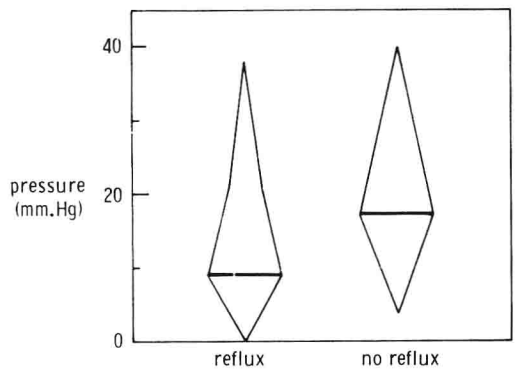

Fig. 1.2 Means and frequency distributions of oesophageal sphincter pressures in patients with and without reflux

(*b*) *Mucosal flap valve.* There is evidence suggesting that the lower oesophageal mucosa normally projects slightly into the stomach and thus forms an inert flap valve.

(*c*) *Size and activity of the diaphragmatic pinch cock.* The right crus of the diaphragm normally compresses the lower end of the gullet from side to side and alters the angle at which it enters the stomach.

Clinical features

The symptoms of hiatus hernia and oesophageal reflux are most

conveniently considered together, for they are interrelated in a complex fashion. Hiatus hernia commonly occurs without any symptoms whatsoever, particularly in elderly people with almost total gastric herniae, where herniae may be seen incidentally on chest X-ray.

By contrast there may be no demonstrable hernia but severe symptoms and easily demonstrable oesophageal reflux.

The classical symptoms associated with reflux are retrosternal burning pain together with regurgitation of bitter fluid, worse after meals and on bending and lying down and relieved by alkalis. Active oesophagitis is frequently associated with retrosternal soreness after swallowing. It is well nigh impossible to distinguish symptoms due to hernia alone, though this lesion may give rise to vague discomfort and distension after meals.

In recent years it has become increasingly clear that biliary reflux can cause oesophageal pain through reflux via the stomach.

Complications

(*a*) *Stricture*. Patients with active oesophagitis may develop a fibrosing stricture: usually in association with an oesophageal ulcer. Endoscopic confirmation to check histologically that the lesion is benign is essential.

(*b*) *Anaemia*. Iron deficiency anaemia due to slow chronic blood loss is common with hiatal herniae: but it is unwise to attribute acute blood loss simply to a hernia.

(*c*) *Ulcer*. (1) Lower oesophageal ulcer is particularly likely to develop in patients who have small areas of ectopic gastric epithelium in the gullet (Barretts oesophagus p. 6). (2) Simple chronic gastric ulcer occasionally develops at the level of the diaphragm in the mucosa of a hernia.

Assessment

Radiology. The ability of radiologists to demonstrate herniae or reflux varies greatly and it is always wise to consider carefully before attributing symptoms to these apparent abnormalities. A ragged mucosal edge or the presence of an ulcer will give such confirmation but will only be found in a minority of patients.

Endoscopy. Visual confirmation of oesophageal inflammation with or without ulceration is useful but not always necessary where there are classical symptoms. The absence of visual evidence of inflammation does not necessarily deny that reflux is the cause of pain. The mechanisms causing pain are not understood.

Acid perfusion. Oesophageal acid perfusion (see p. 234) can be a useful provocative test in difficult cases.

Treatment

The range of agents which increase or reduce lower oesophageal sphincter pressure is shown in Table 1.2. Symptoms of patients with herniae are assumed to be due to reflux and simple initial medical measures include (a) weight reduction in the obese; (b) avoiding conditions which tend to lower sphincter tone; (c) liberal alkali administration; (d) small frequent meals; (e) blocking the head of the bed at night by about six inches; (f) oral ferrous sulphate supplements for anaemia. Reflux of more than minor severity may need further drug treatment.

Table 1.2 Agents affecting lower oesophageal pressure

	Decrease	Increase
Drugs	Alcohol Caffeine Nicotine Anticholinergics (including tricyclic antidepressants)	Cholinergic agents (Bethanecol) Metoclopramide
Food	Fat	Protein
Hormones	Cholecystokinin Secretin	Gastrin
Neurotransmitters	Cholinergic (nicotinic) agents β adrenergic agents	Cholinergic (muscarinic) agents α adrenergic agents

Cimetidine. Symptoms due to acid reflux can be substantially ameliorated by hyposecretory treatment with this H_2 antagonist. 400 mg at night may be enough both to control heartburn and promote ulcer healing. Treatment does not, however, seem to affect any tendency to stricture formation.

Metoclopramide. This will increase lower oesophageal sphincter tone and increases oesophageal peristalsis. It may act by sensitising nerve endings to acetylcholine through blocking nor-adrenergic inhibitory fibres. Anti-cholinergic agents antagonise the effects of metoclopramide and should be avoided.

Alginate-antacid combinations. Combinations of these substances are believed to be particularly useful in reflux oesophagitis because the alginate tends to form a protective layer upon the gastric contents. Preliminary evidence suggests that the addition of small amounts of carbenoxolone is helpful.

Operation

Failure to respond to an adequate period (at least six months) of medical treatment suggests that operation should be considered. Confirmation of failure should include endoscopic evidence of persistent oesophagitis.

Anti-reflux operations combine repair of any hernia with a deliberate attempt to create a barrier to reflux: this is usually done by plicating the gastric fundus round the lower gullet so ensuring that there is an intra-abdominal oesophageal segment as in the Belsey, Hill and Nissen procedures. Operations may also include an anti-secretory vagotomy, and drainage. Satisfactory results should be obtained in at least four-fifths of patients.

Peptic stricture

This is usually a complication of reflux in the elderly and is a sequel to recurrent severe ulceration at the same site with fibrotic scarring. Dyspepsia is often much less of a problem than dysphagia, and if operation is thought inadvisable management can be by simple dilatation. This has become much easier and safer since fibreoptic endoscopes have been used to place guide wires over which dilating olives can be passed (Eder Puestow procedure). Repeated dilatation may be needed, but the procedure is reasonably safe and does not markedly exacerbate heartburn.

Barrett columnar lined oesophagus

Persistent oesophageal reflux may destroy the lower squamous epithelium of the gullet which is then replaced by columnar cells. Strictures commonly develop just below the upwardly misplaced columno-squamous junction. Persistent ulceration and carcinoma within the columnar lined segment are also recognized complications.

Other rarer varieties of diaphragmatic herniae

1. Anterior herniae — usually containing colon — through the foramina of Morgagni: often causing no symptoms.
2. Posterior herniae — containing stomach or colon — through the pleuroperitoneal sinuses (foramina of Bochdalek); this condition can be a cause of respiratory distress in the newborn.
3. Traumatic hernia — usually left sided.

Phrenic ampulla

Barium swallow frequently reveals a small pouch lying above the cardia which does not contain gastric mucosal folds. The appear-

ances, which are a normal pattern on oesophageal distension by a bolus, are often mistaken for a true hernia.

Acute oesophagitis

Apart from inflammation due to reflux of acidic gastric contents or, after gastric surgery, of alkaline duodenal juice containing bile, oesophagitis may be caused by corrosive poisons and by infections.

(*a*) Due to the ingestion of corrosive poisons, e.g. sodium hydroxide. Necrosis may be mucosal or full thickness leading to mediastinitis. Oral feeding should cease immediately (and gastrostomy may be necessary). Infections need treatment with broad spectrum antibiotics. Steroid therapy may perhaps delay or prevent the later development of stricture but the evidence is unconvincing. Once strictures have developed, they may require dilatation or resection with colonic replacement.

(b) In association with tuberculosis, syphilis (tertiary disease), fungus infections (e.g. monilia), and cytomegalovirus infections. Appropriate specific treatment is available for the first three of these.

Oesophageal stricture

Strictures arise as a consequence of inflammatory damage due to reflux, to the swallowing of corrosives and following radiotherapy of the gullet. Dysphagia due to stricture is usually only slowly progressive and for solids rather than liquids.

Treatment

Fibrotic strictures due to corrosives and radiotherapy need cautious dilatation at oesophagoscopy. The treatment of stricture associated with reflux is considered on page 6.

Oesophageal web

Patients — usually female — with severe chronic iron deficiency, may develop diffuse epithelial changes such as glossitis and atrophic gastritis as well as anaemia and koilonychia. If there are oesophageal changes they take the form of a thin fibrous web in the upper gullet. This is usually seen on simple barium swallow or with cine radiology. It has also recently been suggested that oesophageal webs can develop independent of any iron deficiency.

Prognosis

Ferrous sulphate supplements and oesophagoscopic destruction of the web are required.

Treatment
The condition does not tend to recur, provided iron deficiency is rectified, but there is an increased liability to postcricoid carcinoma.

Extrinsic oesophageal disease
Dysphagia may be due occasionally to:

(a) Extrinsic pressure from enlarged hilar glands, for instance secondary to bronchial carcinoma, or from an aortic aneurysm.

(b) Gastric carcinoma infiltrating the cardia.

(c) Aberrant right subclavian artery passing behind the oesophagus (dysphagia lusoria). If symptoms are severe, then the artery should be divided.

Disorders of motility

Achalasia of the cardia

Pathology
There is a general disturbance of oesophageal motility with loss of the normal peristaltic wave (due to degeneration of intra-mural ganglion cells) and failure of the cardia to relax on the arrival of a food bolus. The cause is unknown.

Clinical features
Early stages. Features include pain which is retrosternal in site, worse on taking food; liable to relapses and remissions and associated with sticking of food (solid and liquid) in the gullet and occasional prompt regurgitation of the undigested food together with ropes of sticky oesophageal mucus and swallowed saliva. The dysphagia may be relieved by taking a cold or fizzy drink or by the Valsalva manoeuvre.

Late stages. Dysphagia tends to remit and be replaced by dull retrosternal discomfort and regurgitation of sour fluid on stooping and lying down, even long after meals.

Diagnosis
Early stages. There may be no dilatation and barium sulphate may appear to pass (by gravity) easily into the stomach or only be held up slightly. Bread soaked in barium may, however, be held up and an antigravity swallow will show inability to push the contrast medium uphill. The absence of a peristaltic wave can be confirmed, for completeness rather than necessity, by manometric studies with miniature balloons placed sequentially in the gullet. Diffuse motor

disorder can also be demonstrated by hypersensitivity to mecholine. (A classical physiological response to denervation.)

Late stages. Gross oesophageal dilation is obvious (often even on chest X-ray) and there is much retained food residue in the gullet. The lower end of the oesophagus shows a smooth conical narrowing and the gastric air bubble is lost — due to the oesophageal water seal.

Complications

a. Pulmonary spillover disease. Recurrent pneumonia and bronchiectasis are late complications of gross stasis.
b. Carcinoma of the midoesophagus. Presumably due to chronic irritation in a stagnant gullet.

Treatment

Bouginage has been discarded as ineffective. Medical treatment with glyceryl trinitrate, and amyl or octyl nitrite may give temporary relief of symptoms but the only logical measures are forcible dilatation of the cardia or Heller's procedure (oesophageal cardiomyotomy).

Dilatation with a pneumatic or metal dilator is designed to rupture muscle fibres but not the mucosa: it is not a procedure for the occasional performer.

Heller's operation is the preferred procedure giving good results in most hands, provided that gross dilatation has not occurred — though even then it is worth attempting. The length of the gastro-oesophageal incision must be sufficient to split the cardia properly, but if overextensive, may predispose to oesophageal reflux.

Oesophageal spasm

Diffuse oesophageal muscular spasm, a condition in which there are irregular contractile (tertiary) waves, is an uncommon condition of unknown cause. The associated retrosternal discomfort may mimic that of cardiac infarction, though there is usually a relationship to eating. Barium swallow reveals the characteristic corkscrew appearance due to the irregular tertiary contractions.

Treatment

Anticholinergic drugs such as propantheline may be helpful in relieving pain.

Prognosis

In some patients there is progression to achalasia of the cardia, but in many the condition remains static.

Chagas' disease of the oesophagus

Infection with *Trypanosoma cruzei*, a relatively common disease in Brazil and other areas of South America, is followed in some individuals by abnormalities of the gut and heart. The gastrointestinal changes are due to degeneration of the intramural nerve plexus, presumably directly consequent upon the parasitic disease. The oesophageal changes mimic those of achalasia of the cardia (and treatment is similar). Elsewhere the disease produces megaduodenum and megacolon by an identical pathological process.

Progressive systemic sclerosis (scleroderma)

Dysphagia is a common complication of this generalized connective tissue disease. It is due to loss of the peristaltic wave as the gullet musculature becomes progressively replaced by fibrous tissue.

Treatment

Corticosteroids and para-aminobenzoate have been recommended, but there is no good evidence to support their use.

Oesophageal atresia

Pathology

This congenital anomaly is found in one in every 3000 live births. The main varieties are: (a) with distal tracheo-oesophageal fistula (the majority), (b) without any tracheal fistula, (c) with proximal and distal tracheo-oesophageal fistula, (d) with proximal tracheo-oesophageal fistula only.

It should be suspected in infants of low birth weight or in pregnancy complicated by hydramnios (because this is often associated with upper intestinal obstruction). Other anomalies (commonly duodenal and anorectal malformations) are found in up to a half of the cases.

Clinical features

Respiratory distress after birth due to inhaled secretions manifests itself by episodes of cyanosis and choking, made worse by feeding.

Diagnosis

An oesophageal tube passed through the mouth will be stopped at about 10 cm. The diagnosis can be confirmed radiologically be showing the site of arrest of a radio-opaque tube. If air is detected in the intestine, then the condition must be complicated by a distal