Drug	*Mechanism of Action*	*Effect on Oesophagus*
Cholinergics	Stimulate cholinergic receptors	Increase sphincteric pressures and amplitudes of oesophageal contractions
Anticholinesterases	Increase local concentrations of acetylcholine	ditto
Metoclopramide	Sensitizes tissue to acetylcholine	ditto
Anticholinergics	Block the actions of acetylcholine	Lower sphincteric pressures and decrease amplitudes of oesophageal contractions
β-Adrenergic blockers	Block inhibitory β-receptors in the cardiac sphincter	Raise sphincteric pressures after swallowing
β-Adrenergic stimulators	Stimulate β-receptors	Decrease sphincteric pressures
Antacids	Release gastrin from antral G cells, neutralize acid	Increase sphincteric pressures

Table *Summary of effects of drugs*

Section 5 The physician's problem

J. R. BENNETT

Physician, Gastrointestinal Unit, Hull Royal Infirmary

The physician's problem is not (or should not be) the diagnosis and treatment of characteristic reflux symptoms, with postural and postcibal heartburn, perhaps occasional dysphagia and regurgitation of bitter fluid, and no other significant symptoms. Provided he trusts his clinical assessment and expects nothing of radiology save the demonstration of possible associated disease, then diagnosis in this large group is easy. So, usually, is therapy, for most patients need nothing more than a simple and cheap antacid. Indeed, my experience of therapeutic trials in reflux oesophagitis has been frustrating simply because of the excellent response of control groups given a supposedly placebo antacid.

The physician's diagnostic problems are picking up diseases which masquerade as oesophagitis, and picking out the oesophagitis masquerading as another disease; and his therapeutic problem is coping with the symptoms which do not easily resolve and, if possible, anticipating complications.

Diseases Simulating Simple Gastrooesophageal Reflux

These are achalasia/diffuse spasm, carcinoma cardia, pyloric stenosis, and gastric ulcer and duodenal ulcer.

In achalasia the pain is possibly due to abnormal, spontaneous spasm in the oesophageal wall and regurgitation is from the oesophagus, not the stomach. The other conditions listed are not truly masquerading; the patient's symptoms do include reflux, but there is a cause for the reflux other than weakness of the lower oesophageal sphincter. Particularly beware of carcinoma of the cardia which may have an insidious onset and be difficult to detect radiologically.

Conditions Simulated by Gastrooesophageal Reflux

This group is more important, and errors can lead to serious mistakes in therapy and prognosis.

PEPTIC ULCER (THE REFLUXING HYPERSECRETOR

Some patients with epigastric and lower chest discomfort may be found to have an 'irritable duodenal cap' on radiographs, which is a radiological phenomenon and not a symptom, and may be shown to have gastric hypersecretion, which is a physiological phenomenon. Yet their symptoms are due to gastrooesophageal reflux, and if this is not recognized they may get the wrong treatment for years, and may even have 'ulcer surgery'—which can be disastrous.

ANGINA

Because of similarities in their character, distribution, and precipitating factors, reflux symptoms may be confused with cardiac pain (Bennett, 1966a).

'SIMPLE' VOMITING

A few patients with reflux complain mainly of 'vomiting', almost as though an episode of reflux led to a siphonage effect. Correct therapy depends on recognizing the primary mechanism of the trouble which is regurgitation and not true vomiting, although the regurgitate may be so nauseating that vomiting follows (see section 1).

Diagnosis

In differentiating gastrooesophageal reflux from abdominal troubles, the important characteristic of oesophageal pain is much more its situation than its type. The epigastric pain which is high epigastric and lower chest should turn one's mind away from the stomach and duodenum to the gullet. Sometimes discomfort from the lower oesophagus may be felt largely in the throat.

Discomfort felt as hot fluids or spirits are swallowed, sometimes called 'odynophagia', is especially characteristic. If the pain wakes the patient at night, or if she is conscious of it particularly on waking up, one should search for other features which suggest postural precipitation—gardening is a common cause of trouble.

OESOPHAGEAL PERFUSION

If one is at all puzzled by pain, then one can readily give a patient oesophageal pain to compare with that which he usually experiences. Dripping decinormal hydrochloric acid quickly provokes recognizable discomfort in most patients who have reflux symptoms. (Bernstein, 1967; Bennett and Atkinson, 1966b). It is not a test for oesophagitis; it is a test to identify the site of origin of a pain by the comparison of an induced pain with the naturally occurring pain.

Dysphagia is not a prominent feature of simple reflux. If it is frequent or at all severe it suggests a complication or an alternative diagnosis.

However, iron-deficiency anaemia or even overt gastrointestinal haemorrhage may be the presenting feature, the patients having tolerated their mild reflux symptoms for years without complaint.

'WHAT TO DO WITH A RADIOLOGICAL REPORT'

The radiological contribution to making a positive diagnosis of simple gastrooesophageal reflux is small. If a hiatus hernia is present it supports—but does not prove—the diagnosis (see sections 1 and 3). The absence of a hernia is immaterial, and the radiological assessment of reflux is subject to so many vagaries of behaviour by both patient and radiologist as to be meaningless (see section 3). The real contribution of radiology is to pick out the complications and the associated diseases mentioned above.

I do not think that radiologically observed spasm of the oesophagus on drinking acidified barium (Donner, Silbiger, and Hookman, 1966) is sufficiently discriminatory to be a diagnostic help. Endoscopy is really not essential in making the diagnosis in most patients who present with pain, for the histological changes in the mucosa may be invisible to the naked eye (see section 2), and biopsies

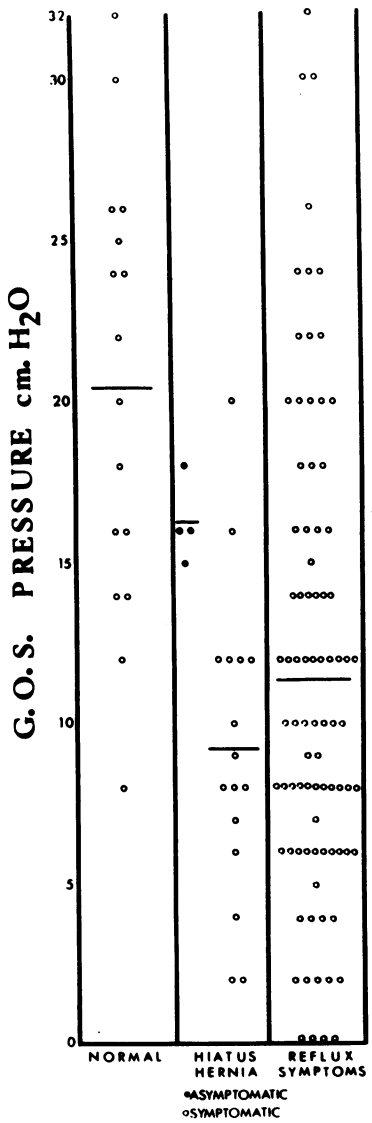

Fig. 9

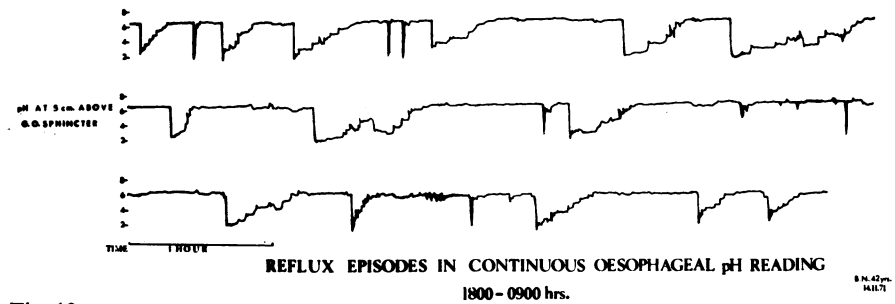

Fig. 10

are often difficult to interpret when taken and assessed by conventional methods (see section 2). Its only effect on management, as far as I am concerned, is that if I see marked 'oesophagitis' I keep a more careful eye on the patients, and exhort them the more vigorously to adhere to their treatment. Oesophagoscopy is obligatory, though, if continuous discomfort worsened by eating or drinking, or if dysphagia, bleeding, or anaemia are present.

MANOMETRY AND PH MEASUREMENTS

Measuring the pressure of the lower oesophageal sphincter cannot tell one if the patient has reflux symptoms or not. Despite enthusiastic claims from Boston (Winans and Harris, 1967) most of us still find overlap between the pressures recorded in the sphincter in symptomatic and asymptomatic subjects, even using modern techniques (fig. 9).

pH measurements can be useful. It is possible, by monitoring oesophageal pH with quite simple instruments, to measure quantitatively and accurately the frequency of acid reflux, and somewhat less accurately the quantity of reflux. Where there is diagnostic difficulty or where the response to therapy is unsatisfactory these measurements can be a sheet-anchor—at least for me (see section 1). I cannot say 'no home should be without one' but I do believe these measurements should be more readily available than they are at present (fig. 10).

Medical Treatment

The cornerstone of all medical treatment is antacid, and the majority of patients respond quickly and well to frequent doses—initially hourly, and then reducing. Gaviscon seems to work as judged by intraluminal pH measurement. Silicone-containing antacid preparations may help too; however, Gaviscon and silicone should not be used simultaneously, as the 'floating raft' of the former is destroyed by the anti-foaming effect of the latter.

Of course patients should stop smoking anyway, but smoking a cigarette reduces cardiac sphincter squeeze and many admit that they get more heartburn after smoking (Dennish and Castell, 1971; Stanciu and Bennett, 1972). Fat patients should lose weight. Posture can be very important, and many patients who have failed to derive benefit from antacids, at whatever frequency, rapidly lose their symptoms when this is explained. Obviously the avoidance of stooping by day is commonsense, but it is the night time which makes the difference. Sitting propped up in bed may even increase the frequency of reflux if the stomach is thereby squashed and, in any case, the patient always slithers down flat if he sleeps soundly. On the other hand, propping the bed-head up eight inches usually reduces reflux markedly. Metoclopramide as a 10 mg tablet by mouth is short acting (25-90 min) (Dilawari and Misiewicz, 1972), but it can sometimes be a helpful adjunct taken immediately before and after meals, for example. And sometimes, as with peptic ulcers, admission to hospital can induce a remission, perhaps by persuading the patient that the therapeutic advice they had followed only half-heartedly really does work.

Medical treatment does not always succeed, though it usually does in uncomplicated reflux. Where there is a stricture, or ulcer, or oesophagitis so severe as to cause significant bleeding, then it probably will not, and if the patient is young enough, fit enough, and thin enough, surgery is the answer. But even with these complications, in patients whom one holds back from surgery because of age, one is often surprised how well they do with conservative treatment.

I regard these as the indications for referral to a surgeon.

FAILURE OF SYMPTOMS TO RESPOND TO MEDICAL TREATMENT

If the patient and his doctor are dissatisfied with the response, and if the patient understands and accepts what surgery will involve.

INSUPPORTABLE COST OF MEDICAL TREATMENT

Some patients can keep their symptoms well controlled by medicine, or careful avoidance of challenging the antireflux mechanism, but find that it is incompatible with a satisfactory normal life—usually this means they cannot do their job satisfactorily, eg, market gardening. If they relapse soon after reducing treatment, they require surgery.

TROUBLESOME CONCOMITANT DISEASE

If a peptic ulcer or gallstone requires surgery, and the patient also has reflux, it is sensible to deal with both simultaneously.

COMPLICATIONS

These are stricture or significant bleeding uncontrolled by medical means. The presence of a hiatal hernia alone is in no circumstances an indication for surgery.

Section 6 Surgical treatment

BRUCE TORRANCE

Surgeon, Department of Gastroenterology, Manchester Royal Infirmary

Indications for Surgery

Over the years, surgeons interested in the treatment of hiatal hernia have become more selective in their recommendations to operate and in the majority of patients the usual conservative measures are invoked. In particular, the patient is invited, if obese, to lose at least 1 stone in weight, and if successful this measure does seem to relieve symptoms in many patients. If the patient is still disabled by reflux symptoms after a reasonable trial of medical treatment then we operate.

I, too, have been impressed with the poor correlation between the degree of endoscopic 'oesophagitis', the histological changes in the oesophagus, and the severity of symptoms, but patients who complain of a scalding retrosternal pain on drinking hot fluids do tend to have quite severe endoscopic 'oesophagitis'. Endoscopic assessment of the degree of 'oesophagitis' was difficult and inaccurate with the primitive instruments of the past but the fiberoptic flexible oesophagoscope makes it much easier to examine the lower oesophagus in detail and to assess the degree of 'inflammation' present.

A barium swallow will only demonstrate oesophagitis if this is particularly severe, and all I personally expect from my radiological colleagues is the demonstration of the presence of the hiatal hernia, stating whether this is reducible or fixed, and giving some idea of the freedom of reflux of gastric contents into the oesophagus. Broadly speaking, clinical assessment takes pride of place over both radiological examination and oesophagoscopy but all three investigations are important in deciding at what stage severe and potentially irreversible changes are occurring, so that by earlier surgical intervention the onset of an irreversible stricture may be prevented. Patients with progressive dysphagia should be operated upon without delay.

Not uncommonly a gastric ulcer develops in the herniated stomach and sits astride the posterior limb of the oesophageal hiatus. Occult or torrential bleeding tends to occur from these ulcers and they should always be treated surgically at an early stage. The majority of ulcers will heal spontaneously after a simple repair of the hernia but, if the repair is through the abdominal approach, it is reasonable to add a vagotomy and pyloroplasty, following a four quadrant biopsy. When the herniation is paraoesophageal and the symptoms are minimal, the decision to recommend surgical treatment may be difficult. In younger patients with a large hernia and minimal risk of surgery a prophylactic operation seems reasonable.

About 25% of patients with hiatal hernia whom I see have some associated upper abdominal pathology, usually gallstones or duodenal ulceration. Since these conditions may influence the clinical symptoms a careful assessment of their contribution must be made before deciding to repair the hernia. In addition the associated abdominal pathology may well influence the approach to the repair.