Chapter 23.1

Peptic ulcer—stomach and duodenum

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Historical perspective

The first description of gastric ulcer is attributed to the Italian physician Marcello Donati in 1586, and the first case of perforated gastric ulcer was recorded by Christopher Rawlinson in England in 1727. Duodenal ulcer was first described by Georg Hamberger in Germany in 1746, and Jacopo Penada from Italy recorded a perforation in 1793. In the second part of the nineteenth century, duodenal ulcers were still rare, but benign gastric ulcer was increasingly recognized. Once general anaesthesia (1846) and antisepsis (1867) had made surgery acceptable and safe, surgeons became involved in the treatment of peptic ulcer. In 1881, Rydigier performed a gastric resection for a benign gastric ulcer and the patient survived. However, he received wide criticism, and the Editor who published the paper, which was entitled ‘The first gastric resection for gastric ulcer’, added the footnote ‘and hopefully the last!’ In 1892, Ludwig Heusner in Germany successfully sutured a perforated gastric ulcer. It was probably not until 1893 that the first operation for duodenal ulcer was performed, a gastrojejunostomy by Codivilla. This rapidly became the treatment of choice for both duodenal and gastric ulcers. On the other hand, 20 years later, von Haberer and Finsterer were championing extensive gastrectomies and their view prevailed when an increasing number of gastrojejunal ulcers were seen after simple gastroenterostomy.

Resection held sway until the 1940s, when Dragstedt re-introduced vagotomy, which had, in fact, been used by Latarjet in 1922 and Skiassi in 1925; but Dragstedt had the experimental evidence that placed the operation on a sound physiological footing. Over the following 40 years, vagotomy became refined and prospective randomized trials were performed to determine the best operation. However, until H₂-receptor antagonists became available in 1976, surgery was the only way to alter the natural history of the disease, and even then the antagonists, and the later proton-pump inhibitors, were only effective while they were being taken. The major change in approach to the treatment of peptic ulcer was the recognition in 1982 that Campylobacter (Helicobacter) pylori was an important factor in the pathogenesis, and that the natural history of the disease could be altered by eliminating this micro-organism.

So surgery was the definitive, elective treatment of peptic ulcer for exactly 100 years. Today the surgeon’s major contribution is in the treatment of life-threatening complications.

Worldwide prevalence and incidence

It is difficult to establish the true prevalence of peptic ulcer, because no one has
gastroscoped a large, representative sample of the population with and without dyspeptic symptoms. Probably about 10 per cent of adults in the United States of America and Europe suffer from an ulcer at some time in their lives. Among individuals presenting to open-access endoscopy clinics with symptoms, between 10 and 19 per cent are found to have peptic ulcer. The ratio of duodenal ulcer to gastric ulcer is 4:1. However, there is evidence that the incidence and severity of the disease is decreasing in the West, and that this decrease started before the introduction of powerful acid-suppressing drugs. In Sweden, elective hospital admissions for peptic ulcer diminished from 64 per 100 000 of the population in 1950 to 11 per 100 000 in 1986. In contrast, for the 10 years after the introduction of \( \text{H}_2 \)-receptor antagonists in the United Kingdom, whereas the elective admissions diminished considerably, emergency admissions for bleeding or perforation remained the same overall but actually increased in males and females over 65 years of age (Fig. 1). In the early 1990s, there were still 40 000 admissions and 4700 deaths annually in the United Kingdom. The reduction in pyloric stenosis is notable and may be due to earlier detection and effective treatment, by whatever means. However, when fibreoptic endoscopy replaced the barium meal as the main method of diagnosis, many small and acute ulcers were seen that were not recognized before.

In Eastern and African countries the pattern varies and is changing. In Singapore, for example, the changes vary in different racial groups. In Japan, where gastric cancer is so common, benign peptic ulcer has a lower prevalence than in the West. In some African countries there are high local prevalences due to specific dietary habits, and migration to the towns in many developing countries has been accompanied by an increase, or increased diagnosis, of peptic ulceration.

**Fig. 1.** Admission rates for (a) men and (b) women with bleeding duodenal ulcer, subdivided by age, in the Trent Regional Health Authority, 1972–84. The total volume of \( \text{H}_2 \)-receptor antagonists (\( \text{H}_2 \text{RA} \)) used is shown only in the graph on the right, and is not subdivided by sex. — • > 65 years; … 35–64 years; – < 35 years; — all ages.
Classification of peptic ulcers

Ulcers are classified either by their state, acute or chronic, or by their site (Table 1). Acute ulcers are those that have not had time to produce any fibrotic reaction around them and they may be very superficial. When they are multiple and widespread they are often referred to as ‘erosions’. A useful classification of gastric ulcers is based on that of Johnson (Table 2). Nearly all chronic duodenal ulcers occur in the bulb (or ‘cap’), and those occupying a more distal site raise the suspicion of Zollinger–Ellison syndrome (or an ulcerating carcinoma of the pancreas). Acute ulcers tend to perforate easily because there is no fibrotic reaction, and they may bleed from superficial mucosal vessels rather than from the major vessels, such as the splenic or pancreaticoduodenal arteries, that are eroded by chronic ulcers.

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<th>Classification of gastroduodenal peptic ulcer by site</th>
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<td>Gastric</td>
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<td>Zollinger–Ellison syndrome</td>
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Table 1 Classification of gastroduodenal peptic ulcer by site
The classification is important because ulcers differ in their pathogenesis and their management. The management of a patient with multiple, recurrent, aggressive chronic ulcers from Zollinger–Ellison syndrome is very different from that of a patient with acute gastric erosions from non-steroidal anti-inflammatory drugs.

### Pathophysiology of peptic ulceration

‘No acid, no ulcer’ is an old adage, but modern drug therapy and surgery have shown it is true. When acid secretion is effectively suppressed, nearly all ulcers heal and do not recur so long as the suppression continues. It was William Prout in 1824 who discovered that the acid in the stomach was hydrochloric.

### Physiology of gastric secretion

#### Acid secretion

Acid is secreted from parietal cells, which are found in the fundus and body of the stomach, whereas the antrum, the muscular ‘pump’, contains gastrin- and mucus-secreting cells. It is this anatomical demarcation that makes parietal-cell (proximal gastric) vagotomy without drainage possible. The secretion of acid is by stimuli originating from (a) the brain, (b) mechanical (distension), and (c) chemical stimulation of the stomach. Secretion from these cells is regulated by neural–hormonal and paracrine pathways that involve (a) direct cholinergic stimulation of acid secretion and (b) cholinergic inhibition of somatostatin secretion, (c) direct gastrin stimulation of parietal cells, and (d) gastrin-induced stimulation of histamine release from enterochromaffin-like cells. In the antrum, cholinergic nerves and gastrin-releasing peptide stimulate gastrin secretion, which is accompanied by cholinergic inhibition of somatostatin. These mechanisms together both stimulate and remove inhibitory influences on gastrin release and thus parietal-cell activity. There are also calcium sensory receptors on gastrin cells, which may explain the increased gastric acid secretion in...
patients with a high serum calcium (hyperparathyroidism). Figure 2 summarizes the main mechanisms.

**Fig. 2.** Model illustrating the neural, paracrine, and hormonal regulation of acid secretion in the fundus and gastrin secretion in the antrum of the stomach. ACh, acetylcholine; GRP, gastrin-releasing peptide; VIP, vasoactive intestinal peptide; SST, somatostatin; ECL, enterochromaffin-like cell.

Drugs act by blocking either the H₂ receptors on the parietal cells (H₂-receptor antagonists) or the secretory mechanism within the cells (proton-pump inhibitors). Vagotomy blocks the cholinergic stimulation of the parietal cells, which desensitizes them to the other stimuli.

If the antrum is also vagotomized, the secretion of somatostatin is increased, which blocks gastrin release and so reduces acid secretion by a second mechanism.

**Pepsinogen secretion**

Pepsinogen, the precursor of pepsin, is secreted via the gastric chief cells and is stimulated by two pathways, one involving hormones such as vasoactive intestinal peptide (secretin) and prostaglandins (E₂), the other involving carbachol, cholecystokinin, and other peptide hormones. It is most active in protein digestion between pH 2 and 4.

**Mucosal defence**

Defence involves resistance and repair, and if small ulcers occur frequently, the rapid repair mechanism is crucial in preventing them becoming chronic, perforating, or bleeding. The gastric mucosa is highly efficient at protecting itself against its luminal acid. Growth factors such as epidermal and platelet-derived promote ulcer healing. The stomach also has the ability to adapt to harmful stimuli whereby initial exposure to a damaging agent,
such as aspirin, makes the mucosa more resistant to subsequent doses. Possibly transferring growth factor-α plays a part in this process.

The duodenum has efficient neutralizing mechanisms if acid from the stomach is delivered to it at a reasonable rate. However, if gastric emptying is particularly rapid, gastric secretion particularly high, or duodenal mixing of bile and pancreatic juice defective, then the defence can be overcome: hence most duodenal ulcers are associated with an increased secretion of gastric acid. The proton-pump inhibitors omeprazole and lansoprazole enhance duodenal bicarbonate secretion as well as reducing gastric acid.

**Mucosal blood flow**

Increase in mucosal blood flow protects the mucosa, provides oxygen and nutrients to the epithelium, and dilutes and neutralizes back diffusion of acid and toxins. If there is acidaemia centrally, then this neutralizing mechanism may fail.

**Non-steroidal anti-inflammatory drugs**

Ulceration by non-steroidal anti-inflammatory drugs remains a problem. In the United States of America, 70 million prescriptions for non-steroidals are given annually, and there are 76 000 related hospital admissions and 7600 deaths. The discovery of two isoforms of cyclo-oxygenase has led to the hope that non-steroidals that only inhibit cyclo-oxygenase 2 will be ‘gastrointestinal safe’. At present, patients with a history of peptic ulcers should not be started on these drugs without added protection, and they should be stopped if ulcers develop.

**The role of Helicobacter in the pathogenesis of peptic ulcer**

**How does Helicobacter pylori influence acid secretion?**

The precise pathway is not known, but one possible mechanism is that it decreases antral somatostatin secretion (which inhibits gastrin release), leading to prolonged hypergastrinaemia. It may also act by synthesizing a selective H₃-receptor agonist.

However, patients with duodenal ulcer have a greater sensitivity to gastrin than *H. pylori*-affected controls and also an increased maximum acid secretory capacity.

**H. pylori and mucosal defence: microcirculation**

Extracts of *H. pylori* from human infection produce platelet activation and aggregation in the mucosal microcirculation of rats, with leakage of albumin. These changes would predispose to ulceration and delay healing. It is not yet clear in what other ways, if any, *H. pylori* inhibits defence mechanisms. The publication of the entire genome of *H. pylori* in 1997 has opened new possibilities for understanding and combating the infection.

**Duodenal juice and gastric mucosal defence**

For surgeons the effect of duodenal juice on the gastric mucosa is important. Not only does this action occur before surgery in certain conditions, but gastric operations such as
gastroenterostomy, partial gastrectomy, and vagotomy and pyloroplasty all increase the duodenal reflux into the stomach. For many years, duodenal juice has been implicated in damaging gastric mucosa in such a way as to make it more susceptible to acid damage. The evidence comes from clinical studies, which show a higher concentration of bile and pancreatic juice in the stomach of patients with gastric ulcer. Exposure time is obviously important and delayed gastric emptying compounds the problem of duodenal gastric reflux; there are a number of different factors leading to the final ulceration.

The risk factors for gastric ulceration include smoking, excessive alcohol, non-steroidal anti-inflammatory drugs, and corticosteroids. Additional risk factors for duodenal ulcer are liver cirrhosis, atheroma (particularly coronary arterial disease), renal transplantation, and other states of immune suppression.

The natural history of untreated peptic ulcer

In the days when the only treatment for ulcers was to try to neutralize the acid after it had been secreted, the natural history of the disease could be observed. Peptic ulcer is a cyclical disease with a sequence of healing and exacerbation over the years. In some countries, ulcers tend to become reactivated in the winter months, but chronic ulcer can be present for years. The concept of a ‘burnt out’ ulcer is attractive, but even an ulcer that has been quiescent for many years can be reactivated at a time of stress, such as a surgical operation or new drug therapy, and it is important that prophylaxis be started when a patient with a past history of ulceration is undergoing such stress. It was always thought that acid secretion diminished with age, and the most recent work has confirmed this view, once other diseases have been excluded. However, the important message is that any untreated ulcer is potentially lethal from perforation or bleeding, and it is impossible to predict in any individual patient when this might happen. The only safe policy is to keep ulcers healed.

Before the importance of *Helicobacter* was appreciated, clinicians had noted that bismuth-containing ulcer treatments reduced the recurrence rate after healing. During the years when $H_2$-receptor antagonists were the mainstay of treatment, recurrence was high after stopping treatment (80 per cent within 18 months) and did not depend on how long the treatment had been used. The only two treatments that altered the natural history were continuous, full-dose, $H_2$-receptor antagonists or surgery (e.g. vagotomy).

Symptoms and signs of peptic ulcer

Many ulcers are symptomless and a severe complication may be the first sign of their presence. It used to be thought that peptic ulcers gave very characteristic symptoms, and that duodenal and gastric ulcers could be distinguished by the timing of the pain in relation to food, duodenal ulcer being characterized by pain when starving, relieved by meals, whereas gastric ulcer pain was brought on by food, so that the patient avoided meals and lost weight. There is some truth in these assertions, but symptoms are often not so clear-cut. When all the many symptoms attributed to duodenal ulcer are carefully analysed, the most discriminating (but by no means 100 per cent) is the patient being woken with...
localized epigastric pain in the middle of the night and it being relieved by taking antacids or milk. Various forms of dyspepsia, such as fullness and slight nausea, are common, but vomiting usually only occurs if there is obstruction. In gastric ulcer the symptoms occur sooner after food, and vomiting and weight loss are more common, whereas they are rare with uncomplicated duodenal ulcer. Continuing pain radiating to the back suggests a penetrating ulcer, but it must be differentiated from carcinoma of the pancreas. A stomal ulcer at a gastrojejunostomy will give a similar type of pain felt in the periumbilical region rather than the epigastrium, because the ulcer affects the mid- rather than the foregut.

Symptoms may be confusing because of the coexistence of other digestive conditions such as oesophageal reflux, with or without hiatus hernia, and gallstones. Anaemia is an important symptom because both types of ulcers can bleed slowly. The other, most likely cause of anaemia without rectal bleeding or melaena is carcinoma of the caecum or right colon. The most important differential diagnosis of peptic ulcer is carcinoma of the stomach. Patients over the age of 50 years with new dyspepsia must be investigated by endoscopy as soon as possible. Perhaps the most useful, practical guide in younger patients is that a man with intermittent upper abdominal pain is likely to have a duodenal ulcer, whereas a woman is likely to have gallstones. In older people, the sex difference for peptic ulcer is not so marked.

Physical examination is useful for confirmation if there is localized tenderness in the epigastrium, and to exclude other conditions and detect clinical anaemia. It does not, however, make the diagnosis.

**Diagnosis**

Fibreoptic endoscopy is the principal method of diagnosis, as it enables biopsies to be taken to exclude malignancy in gastric ulcers, and to do a rapid urease test or histological examination for *Helicobacter*. The $^{14}$C breath test is used to confirm eradication to avoid a further gastroscopy.

A double-contrast barium meal is rarely used as a primary means of diagnosis, but is useful in patients with a morbid fear of endoscopy. On open-access endoscopy for ‘dyspepsia’, about 12 per cent of patients have duodenal ulcer and 3 per cent gastric ulcer, but the numbers and ratios vary with age. Every gastric ulcer should be suspected of being malignant until proved benign by multiple biopsies and complete endoscopic healing.

A frequent clinical problem is the patient who is referred for gastroscopy having been started on acid-inhibitory drugs several weeks before. If no ulcer is seen, it is often impossible to know whether there was an acute ulcer that has healed, or whether the diagnosis was not peptic ulceration. In these circumstances, it is best to stop the treatment and arrange to endoscope the patient as soon as the pain recurs. Ideally, patients should not be put on powerful acid-reducing drugs, which heal ulcers rapidly, before a diagnosis is made: otherwise they may be condemned to long periods of drug therapy or to *Helicobacter* elimination for the wrong reasons.

**Gastric acid secretion tests**

These have no place in the routine diagnosis of ulcers, but a serum gastrin of several
hundred pmol (normal < 40 pmol/l) is strongly suggestive of a gastrinoma in Zollinger–Ellison syndrome, provided that the patient is not achlorhydric with pernicious anaemia (in which case any ulcer would be malignant), is not on acid-inhibiting drugs, nor has had a vagotomy. H₂-receptor antagonists should be stopped for 48 h and proton-pump inhibitors for at least 1 week before a sample for serum gastrin is taken.

**Treatment of uncomplicated peptic ulcers**

The aim of treatment is (i) to relieve symptoms; (ii) to heal the ulcer as quickly as possible; (iii) to prevent recurrence; (iv) to prevent complications.

A full dose of H₂-receptor antagonists will heal about 90 per cent of duodenal ulcers in 8 weeks, and proton-pump inhibitors will have the same effect in about 4 weeks. Pain relief occurs in a few days, there being little difference between the two groups of drugs at 2 weeks. Rapid pain relief has led to patients not completing the full healing course, or using powerful acid-suppressing drugs as ‘indigestion tablets’, taking them when they have an attack of pain. It is important to stress that the risk of complications is not over until the ulcer is completely healed and stays healed. Proton-pump inhibitors are the most effective drugs in healing benign gastric ulcers, with a healing rate of 70 to 80 per cent at 4 weeks and 80 to 90 per cent at 8 weeks.

**Elimination of H. pylori**

It is important to eliminate, not just suppress, *H. pylori*, and many regimens have been used and compared. The classic triple therapy of ‘bismuth’, metronidazole, and amoxycillin (amoxicillin) for 2 weeks eliminates *H. pylori* in 90 per cent of patients. However, this was not very palatable and new regimens have superseded it, based on ranitidine–bismuth citrate or proton-pump inhibitors with an antibiotic. A proton-pump inhibitor, with metronidazole 400 mg and amoxycillin 500 mg three times a day, results in eradication rates of 85 to 95 per cent in 1 week. If patients are allergic to amoxycillin, clarithromycin may be substituted; this antibiotic combined with ranitidine–bismuth citrate heals 93 per cent of patients. For the 5 to 15 per cent that do not respond to the proton-pump inhibitor, bismuth should be added and the course extended for 2 weeks. Antibiotic resistance is an increasing problem, both primary resistance to metronidazole (41 per cent) and secondary resistance to clarithromycin (in 25 per cent of patients whose ulcer failed to heal on dual therapy). The elimination of *H. pylori* should be limited to patients with peptic ulceration: there is no evidence that universal testing and elimination is justified. Elimination of *H. pylori* may make gastro-oesophageal acid reflux worse.

**Prevention of ‘stress’ ulcers**

Acute peptic ulcers are a complication of any serious trauma, particularly if the patient is in an intensive care unit. Prophylaxis with H₂-receptor antagonists (e.g. ranitidine) intravenously was associated with only 1.7 per cent clinically significant bleeds, but acid suppression leads to bacterial colonization of the stomach, and many intensive care units prefer mucosal protection with sucralfate. It is also important to avoid a systemic acidosis by careful monitoring. For those patients who cannot come off non-steroidal anti-
inflammatory drugs, concurrent treatment with the prostaglandin E\textsubscript{1} analogue misoprostol, or with effective acid-suppressive drugs, helps to prevent ulcers and treat their complications. If a patient has presented with a bleed or perforation, these protective measures are essential. Eradication of \textit{Helicobacter} before treatment with non-steroidals reduces the incidence of gastroduodenal ulceration, but this is not possible acutely.

\textbf{Should a patient be given treatment for suspected peptic ulcer disease without a diagnosis being made?}

The answer to this question is 'no', because of the danger of masking a carcinoma in the older patient, and of submitting a younger patient to intensive drug treatment without knowing the diagnosis. However, young patients who relapse with exactly the same symptoms as when the diagnosis was first made may reasonably be given a second course of acid-suppressing drugs. In most circumstances, \textit{H. pylori} would now have been treated to prevent recurrence.

\textbf{Indications for elective surgery}

With the introduction of H\textsubscript{2}-receptor antagonists in 1976, the frequency of elective hospital admissions for peptic ulcer started to decline, but overall the frequency of emergency surgery stayed the same, and, as mentioned above, actually increased in elderly men and women over the following 7 years (Fig. 1). Elective surgery was the alternative to lifelong drug treatment or for the 10 per cent or so of patients whose ulcers failed to heal. With very few treatment failures with proton-pump inhibitors, and the ability to eliminate \textit{H. pylori} in most cases, the need for elective surgery is even less. The present indications can be summarized as follows:

- The occasional patient whose ulcer fails to heal with medical treatment or who relapses frequently and is \textit{H. pylori} negative.
- Prevention of recurrent complications when the patient is at high risk.
- Possibly as a protection in patients who cannot be without non-steroidal anti-inflammatory drugs.
- For financial reasons, either from the patient's or supplier's viewpoint: in some countries the patient has to pay for drugs but the operation is free, and, in many countries, surgery is considerably cheaper than long-term drug therapy.

\textbf{Which elective operation for duodenal ulcer?}

There is a variety of operations, all of which aim to produce a permanent reduction in gastric acid secretion: truncal or proximal gastric vagotomy, partial gastrectomy, or truncal vagotomy and antrectomy. The classic, Leeds–York, prospective, randomized trial for duodenal ulcer compared three of these, truncal vagotomy and drainage, subtotal gastrectomy, and vagotomy and antrectomy. It found that the recurrence rate was zero with the most radical operation, vagotomy and antrectomy, but this had significant side-effects,
whereas the vagotomy and gastroenterostomy had the highest recurrence (4.2 per cent). However, the side-effects of dumping and diarrhoea were still significant, although in different proportions for the different operations, and surgeons realized that it was as much the drainage procedure as the vagotomy that produced these. The next logical step was to do a selective and then highly selective or proximal gastric vagotomy where just the parietal mass was denervated leaving the antral muscular pump to empty the stomach. Therefore, a drainage procedure was not required, as gastric emptying, particularly of solids, would be relatively normal. Prospective, randomized trials comparing this new procedure with truncal vagotomy and pyloroplasty for duodenal ulcers showed a similar recurrence rate (9 per cent) but significantly fewer side-effects after proximal gastric vagotomy. Surgeons have been obsessed, over the years, with the recurrence rate of operations, whereas a transient ulcer recurrence may be of no consequence. Much more important is persistence of ulcer and the life-threatening complications. If the recurrence of ulcers is classified in a modified Visik grading, then proximal gastric vagotomy has been extremely effective in the elective treatment of duodenal ulcer, with very few complications.

When proximal gastric vagotomy was a standard, long-term treatment for duodenal ulcer, it was being done before there was an effective medical treatment or as an alternative to long-term, full-dose H₂-receptor blockers. Although it was suggested that it was not so effective in patients whose ulcer had not healed with H₂-receptor blockers, further studies did not confirm this. Nowadays, operation is only being considered for a few patients with very resistant ulcers and it is not known whether this selected subgroup will respond as well to proximal gastric vagotomy. In addition, the new generation of gastric surgeons have little experience with proximal gastric vagotomy, which is an operation that is technically demanding if the vagotomy is to be complete. Therefore, unless the surgeon has had much experience in the past of proximal gastric vagotomy, the right operation is probably truncal vagotomy and antrectomy because, in the selected patients, it is absolutely essential that the ulcer is healed and remains so. Surgeon and patient would have to accept the small but definite incidence of side-effects (see below).

**Elective operations for gastric ulcer**

The great difference between gastric and duodenal ulcers is the gastric ulcer's malignant potential. Therefore, whichever operation is contemplated, excision of the ulcer is always advisable. A Billroth I partial gastrectomy has stood the test of time as a standard operation for benign gastric ulcer. Proximal gastric vagotomy with ulcer excision did have a vogue and was tested in trials; overall, it was found not to be as satisfactory. In addition, it is technically difficult if there is a lot of scarring around the lesser-curve gastric ulcer. Emergency surgery for bleeding gastric ulcer is a different issue and will be discussed below. The recurrence rate of gastric ulcer after Billroth I in different studies varies between 0 and 16 per cent.

**Complications of peptic ulcer**

Complications of peptic ulcer account for 4500 deaths a year in the United Kingdom. The surgeon, nowadays, is mainly concerned with the complications of peptic ulcer rather than
elective treatment. Unfortunately, so many of the patients are elderly with other serious conditions, such as atrial fibrillation treated with anticoagulants, which makes emergency surgery more hazardous.

**Perforation**

Acute perforation may be the first sign of peptic ulcer, and fatality in elderly people can be as high as 20 per cent. The classic symptoms and signs of severe epigastric pain, board-like rigidity, with air under the diaphragm on the chest radiograph, often make the diagnosis easy in 80 per cent of patients. But perforation is not always so straightforward. A gastric ulcer perforated into the lesser sac (Fig. 3) can give misleading symptoms and be confused with pancreatitis (the serum amylase may be slightly raised because of the absorption of pancreatic juice from the peritoneal cavity). Alternatively, the fluid from a perforated duodenal ulcer may track down the right paracolic gutter and present with tenderness in the right iliac fossa, mimicking acute appendicitis. More than one surgeon has made a small appendectomy incision only to find bile-stained fluid emerging from the peritoneal cavity! Perforation is particularly difficult to diagnose in the patient who is on a high dose of steroids, because the symptoms and signs (although not the radiograph) are masked. The mortality has a direct relation to the delay before treatment. The common differential diagnosis of peritonitis with air under the diaphragm is a perforated diverticulum of the colon.
Management

The first stage of management is restoration of fluids and correction of electrolyte abnormalities, and the start of nasogastric suction. The aspirate commonly contains small flecks of fresh or altered blood. There is a long-standing tradition in surgical management of the acute stage that no analgesia should be given until a firm diagnosis has been made. This is unkind and unnecessary, especially for a patient who has to travel some distance with every bump in the road exacerbating the peritoneal pain. A short-acting analgesic such as pethidine may be given as long as the receiving room in the hospital is made aware that this has been done.

Is there a place for non-operative treatment?

Although surgery is normally the correct treatment for perforated duodenal ulcer, the whole
patient and the comorbidity need to be taken into account. Perforations may seal themselves by adherence to liver, gallbladder, or omentum. If an elderly, unfit patient presents with a 3-day history suggesting perforation but does not show the signs of generalized peritonitis, and is otherwise a very poor operative risk, then non-operative treatment is entirely reasonable. This would mean nasogastric suction, intravenous fluids and antibiotics, and may be undertaken despite the presence of air under the diaphragm on the radiograph. A careful watch must be kept to ensure the patient does not deteriorate further. Abscesses caused by the perforation can often be drained percutaneously later.

A water-soluble contrast radiograph can help to confirm that the perforation is, in fact, sealed, but gastroscopy must be done with great care, with little gastric and duodenal distension, because a lightly sealed omental patch can be blown off by enthusiastic insufflation! Mortality rates of less than 10 per cent are claimed for non-surgical treatment, but these figures have little meaning unless the selection criteria are carefully scrutinized. A follow-up gastroscopy is required to ensure any ulcer has healed and to biopsy a gastric ulcer.

**Operative techniques—simple suture or patch?**

An acute, perforated duodenal ulcer may be closed by simple suturing and reinforced with an omental patch over the top. However, it is impossible to close a large, chronic ulcer when there is fibrosis, which holds the edges apart. In this case, an (attached) omental patch is sutured into the defect, with sutures passing across the perforation over the patch. If they are tied too tight, the omentum will become infarcted. Long-lasting but absorbable sutures are ideal for this, whereas non-absorbable sutures remain in the duodenal wall and can act as the site for further ulcerations.

Perforated gastric ulcers may be excised, the new edges brought together with interrupted sutures, and reinforced by an overlapping layer or some omentum. Normally there is plenty of ‘spare stomach’ on the lesser curve or posterior surface to allow the edges to come together without tension. The excised ulcer must be sent for histological examination to exclude malignancy. In patients on steroids, special care and extra reinforcement are necessary because of slow healing. The peritoneal cavity is washed out thoroughly with saline and a drain inserted only if there was a collection of infected material that might re-accumulate.

Laparoscopic repair of perforated ulcer has been done, the aim being to reduce surgical trauma. However, washing out gastric contents, which is an important part of treatment, is not so easy through a laparoscope, and peas and pieces of chipped potatoes may be left behind! A well-conducted, randomized, controlled trial compared open and laparoscopic suture and glue; it was concluded that the laparoscopic approach had no advantage over the open procedure and took longer to perform, which is not surprising, considering that an open repair of perforation and wash-out is a quick and simple procedure. The use of glue is more controversial and has little advantage in the open procedure, although it is attractive if it is being done laparoscopically. But more work needs to be done before this can be recommended as a routine.
Should an ulcer-curing operation be added?

An important decision at operation is whether or not to add a vagotomy to simple ulcer closure (or perform a partial gastrectomy for a perforated gastric ulcer). Before the days of powerful acid inhibitors or the recognition of H. pylori, surgeons based their decisions on whether or not there was chronic ulcer disease and on the risks of prolonging the operation. Now that the natural history of peptic ulcer can be altered, either by continuous drug therapy or elimination of Helicobacter, the need to do a definitive procedure has greatly diminished. The surgeon must aim to close the perforation as quickly and effectively as possible, especially in poor-risk patients. The exception may be when a patient is likely to have to stay on non-steroidal anti-inflammatory drugs or steroids and may already be on a protective dose of acid-reducing drugs. Although vagotomy does not protect completely from ulceration by these drugs, it acts synergistically with misoprostol or long-term H₂-receptor antagonists. For gastric ulcer, there is still debate whether Billroth I for perforation is safer in terms of making sure the ulcer actually heals. Again, this judgement has to be made against the experience of the surgeon and the general condition of the patient. If a drainage procedure is used to accompany a truncal vagotomy, then a gastrojejunostomy is preferable to a pyloroplasty across the perforation for duodenal ulcer. This is because it can be closed later, should dumping or diarrhoea be a problem (see below). A gastrojejunostomy may also be required when the repair of a very scarred duodenal ulcer might produce some gastric-outlet obstruction.

Haemorrhage

All haemorrhage from peptic ulcers is potentially lethal and temporary cessation of bleeding is no cause for complacency. Bleeding peptic ulcer accounts for 70 per cent of all upper gastrointestinal bleeds, and the general mortality is 5 to 10 per cent. Thirty per cent of patients who bleed from peptic ulcers have no previous definite history or previous diagnosis of ulcer disease. The mortality among those who need surgery is higher, around 20 per cent, because their bleeding is the most severe. Sadly, the mortality has not improved over the last 50 years because, despite fibreoptic endoscopy and other advances, the patients who bleed are increasingly elderly with many concurrent illnesses. Important points in their history include previous peptic ulcer, alcohol abuse, drugs (particularly non-steroidals), and anticoagulants, and cardiovascular and pulmonary disease. Important signs are the stigmata of liver disease, which point to oesophageal varices as the cause of the bleeding, but do not, it must be emphasized, exclude peptic ulcer disease.

The management of haemorrhage consists of five phases: (i) resuscitation; (ii) diagnosis; (iii) immediate control of bleeding; (iv) prevention of re-bleeding; (v) prevention of recurrent bleeding in the future. Only when an ulcer is healed and remains healed has the risk of haemorrhage passed. There is increasing world-wide evidence that patients with upper gastrointestinal haemorrhage are best treated in a dedicated ‘bleeding unit’ jointly staffed by surgeons and gastroenterologists, so that they can be closely observed and agreed protocols adhered to so that intervention can be prompt. In such units, a mortality of less than 5 per cent can be achieved. Bleeding presents as haematemesis, melaena, or both. Massive bleeding can lead to only slightly altered blood being passed per rectum, but
in this situation the patients are severely shocked because the bleeding is so severe.

**Resuscitation**

This consists of an intravenous line, cross-matching of blood, and a full blood count. The haemoglobin measured immediately afterwards does not give any indication of the severity of the bleed as the concentration will not drop until there has been dilution with replacement fluids. A low haemoglobin at the time of the bleed indicates previous chronic bleeding. There is continued transatlantic debate about whether crystalloids or colloids should be given while awaiting the blood.

**Diagnosis**

If there has been a significant bleed with the blood pressure low and other signs of shock, patients should be gastroscoped as soon as they are stable. The purpose of the gastroscopy is to determine the site of bleeding and judge the risk of recurrent haemorrhage; it does not often help in determining when the surgeon should operate. The most important differential diagnoses are the results of portal hypertension—oesophageal or gastric varices and congestive gastropathy (see Chapter 32.1). The stigmata of recent haemorrhage are shown in Table 3 and have some value in predicting re-bleeding. An intravenous injection of metaclopramide before endoscopy helps to clear the stomach of blood and, incidentally, control temporarily the bleeding from oesophageal varices.

<table>
<thead>
<tr>
<th>Sign</th>
<th>Risk of rebleed* (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulsatile bleeding or oozing</td>
<td>85</td>
</tr>
<tr>
<td>Fresh clot in ulcer crater</td>
<td>85</td>
</tr>
<tr>
<td>Adherent clot on ulcer</td>
<td>50</td>
</tr>
<tr>
<td>Visible vessel in base: i.e., dark spot, red spot, elevated tubular structure, defect in base of ulcer</td>
<td>40</td>
</tr>
</tbody>
</table>

*Or of continued bleeding

**Table 3** Endoscopic signs of recent haemorrhage

**Immediate control of bleeding**

The simplest method of controlling bleeding is the injection of 0.5 to 1 ml volumes of 1 in 100000 adrenaline (epinephrine) into the ulcer base around the bleeding point. Other methods include neodymium–yttrium aluminium garnet lasers and bipolar...
electrocoagulation, as well as the injection of sclerosants with and without adrenaline. Prospective, randomized trials support simple injection techniques. Indications for immediate surgery are as follows:

1. very severe bleeding in the absence of signs of liver disease when endoscopy would cause delay;
2. the failure to be able to see the bleeding point in someone who is still bleeding (oesophageal varices having been excluded);
3. failure to control a spurting vessel by injection;
4. rare lesions, such as an aortoenteric fistula, that invariably re-bleed.

**Prevention of re-bleeding**

Re-bleeding in hospital is the major cause of death, and so it is very important that the patient should not be allowed to have a third bleed. Most elderly patients stand an operation better than a recurrence of severe bleeding. Continued oozing, although not dramatic, can be an unrecognized danger. The signs of re-bleeding are:

1. fresh haematemesis or melaena in a stabilized patient (old melaena may be present for some time after the initial bleed);
2. a fall in blood pressure;
3. a steady rise in pulse rate in a stabilized patient.

Indications for surgery are as follows:

1. one severe re-bleed;
2. transfusion requirements of 4 units in 24 h in a patient over 50 years or 6 units in a patient under 50 years of age;
3. shortage of whole blood.

It is normal to start acid-suppressing drugs, but it is naive to think that they would prevent re-bleeding in a large ulcer with an exposed vessel. It takes many days, if not weeks, for the ulcer to heal to the extent that eliminates the risk of further bleeding.

**Prevention of recurrent ulcer**

The secondary aim of treatment is to prevent recurrent ulcer in the future, but this must be considered as part of the overall treatment plan. It would determine the type of surgery.
Operation

As with a perforated ulcer, the decision is whether to do definitive surgery with an ulcer-curing operation, such as Billroth I partial gastrectomy for a gastric ulcer, or truncal vagotomy and pyloroplasty for duodenal ulcer, or whether to use medical treatment to prevent future ulcers. Theoretically, undersewing of the bleeding vessel together with medical treatment, such as the elimination of \textit{H. pylori}, is an option. Unfortunately, the international randomized trial designed to answer this question did not control for surgical techniques and had to be stopped because of a high mortality after minimal surgery. The decision depends on the patient's general condition and the experience of the surgeon. Figure 4 summarizes the management of bleeding peptic ulcer.
**Stenosis**

Stenosis of the body of the stomach, creating two chambers like an hourglass, is rare nowadays because ulcers are treated effectively before they reach this stage. Pyloric (or, more accurately, duodenal) stenosis still occurs because fibrosis is the result of healing. However, duodenal oedema is more common than true fibrosis and may clear with medical treatment. When a benign gastric ulcer is secondary to pyloric stenosis (type 2), the stenosis needs treating. Occasionally, a benign gastric ulcer can be secondary to malignant gastric-outlet obstruction. A non-operative option for established stenosis is balloon dilatation at endoscopy, and this may be successful when the main problem is oedema rather than true stenosis. However, effective dilatation of a stenosis can lead to rupture of the duodenal wall, or to re-stenosis rather soon afterwards. For established fibrosis, a vagotomy and pyloroplasty or gastroenterostomy is necessary, with excision of a gastric ulcer if present. The advantage of a gastroenterostomy is that, if the stenosis does improve and there are side-effects from the drainage procedure (such as dumping or bilious vomiting), the gastroenterostomy may be closed later. If the stenosis is sufficiently distal to the pylorus, a duodenoplasty may be used, thus preserving the pyloric function. Operative dilatation of the pylorus followed by a proximal gastric vagotomy, although an attractive option, was not always successful, because of leakage and re-stenosis.

**Fistulas**

**Gastrocolic**

A benign gastric ulcer of the greater curve may occasionally cause a gastrocolic fistula, although this is more common (10–20 per cent of cases) when a jejunal ulcer occurs at the anastomosis of a retrocolic gastroenterostomy; these should strictly be called gastrojejunoscolic fistulas. The unpleasant side-effects are diarrhoea, weight loss, and faecal vomiting. Surgeons used to recommend a proximal colostomy first, before doing a resection of the ulcer, closing the colon, and adding an antrectomy or truncal vagotomy depending on the previous operation. Nowadays a one-stage operation is feasible after the patient's nutrition and electrolyte disturbances have been corrected. Duodenocolic fistulas are rare and can result from a duodenal ulcer or carcinoma of the hepatic flexure.

**Duodenobiliary**

A fistula may form between the duodenum and the gallbladder or the common bile duct. Cholecystoduodenal fistulas are usually due to gallbladder disease and may lead to gallstone 'ileus' when a large gallstone passes through the fistula and becomes impacted in the terminal ileum. Fistulas are particularly common when there is xanthogranulomatous
cholecystitis. Choledocoduodenal fistulas are due to posterior penetration of a duodenal ulcer. If there is a narrow opening or other cause of biliary stenosis, then cholangitis results. The fistula may heal when the ulcer does, but if not it is excised, and the duodenum and bile duct closed over a T-tube if stenosis is a risk. Occasionally a choledocejunostomy is required if there is severe narrowing of the bile duct.

**Malignant change in peptic ulcers**

See Chapter 23.2.

**Surgical techniques**

**Billroth I partial gastrectomy**

This is the standard operation for gastric but not for duodenal ulcer. The distal stomach and pylorus, including the ulcer, is excised and an end-to-end gastroduodenal anastomosis is made to restore continuity (Fig. 5). The benign ulcers are commonly on the lesser curve and therefore the resection line may be taken high. A new lesser curve is made to narrow the stoma of the gastric remnant to the size of the duodenum. A specially angled clamp makes this easier (Fig. 5(a)); stapling devices are quicker, but far more expensive than sutures. Dividing the lateral peritoneal attachments of the duodenum (Kocherization) allows the duodenum to be mobilized medially to prevent any tension on the anastomosis. The physiological advantage is that the food progresses in the normal direction.
Pylorus-preserving gastrectomy (Maki)

This variant was devised to retain pyloric function and therefore prevent the rapid gastric emptying and dumping that may occur when the pylorus is excised. About 1.5 cm proximal

Fig. 5. Three stages in a Billroth I partial gastrectomy.

http://gateway.ut.ovid.com/gw2/ovidweb.cgi
to the pylorus is preserved with the pyloric branches of the vagus nerve that come down from the liver (Fig. 6). This may lead to initial delayed emptying compared with Billroth I, but both clinical assessment and isotope emptying studies show that it retains a more normal emptying pattern long term and therefore may be appropriate for benign disease.

**Billroth II (Polya) partial gastrectomy**

This can be used for both duodenal and gastric ulcers, and involves resecting two-thirds of the stomach, closing the duodenum distal to the pylorus (usually beyond the ulcer), and an end-to-side gastrojejunal anastomosis. In the past there was much discussion of whether the jejunum should be retrocolic or antecolic and whether the loop should be isoperistaltic to the stomach or retroperistaltic (Fig. 7). For a benign disease, this probably does not matter, but for malignant disease the jejunum is brought in front of the colon so that it is less likely to be involved in any local recurrence. Closing off the duodenum takes the ulcer-bearing area out of the acid stream, but the physiology of digestion is more disturbed than after a Billroth I.

Fig. 6. Pylorus-preserving gastrectomy (Maki).
The closing of the duodenum can be very difficult if there is much scarring. It may have to be closed distal to a posterior penetrating ulcer, and this may involve a longitudinal rather than transverse suture line (Fig. 8). Sometimes the ulcer can be excluded and the duodenum closed, leaving the ulcer posteriorly on to the pancreas. With very large, fibrosed ulcers, as a last resort, a drainage tube may be put into the duodenum and the duodenum sutured around it to provide a formal duodenal fistula, but this is not ideal. Another technique is to anastomose a jejunal Roux limb over the duodenum instead of trying to close the duodenum when there is only a little tissue available. It is the leakage from the duodenal stump that provides the main risk of Polya partial gastrectomy, and that is why vagotomy and drainage is a safer operation.
Vagotomy and antrectomy

If resection of the antrum (gastrin-secreting area) is used in addition to a truncal vagotomy, the resected area is smaller, about 6 cm proximal to the pylorus on the lesser curve. The re-anastomosis may be gastroduodenal or gastrojejunal, the gastroduodenal being preferable if the duodenum is not too scarred (Fig. 9).

Fig. 8. Longitudinal closure of duodenum distal to a posterior penetrating duodenal ulcer.
Truncal vagotomy and pyloroplasty or gastrojejunostomy

Truncal vagotomy is carried out as high as possible on the abdominal oesophagus above the area where the vagi start to branch. It is really a perhiatal procedure. The peritoneum over the anterior surface of the oesophagus is divided and the phreno-oesophageal ligament identified and elevated, exposing the anterior vagal trunk. The posterior trunk lies on the right posterior aspect of the oesophagus, often well away from the oesophageal wall, and the encircling finger around the oesophagus may pass medial to it and therefore it may be missed. A 1-cm piece is taken out of each of the trunks and the surgeon must carefully search the oesophageal wall for small branches. A 4 cm-long segment of the oesophagus should be denuded of its attachments. The habit of sending a piece of suspected vagus nerve for histological examination does not help completeness because the trunks may branch high and small branches be left. Some surgeons recommend re-anastomosing the peritoneum over the oesophagus, but it does not seem that this is important. When a patient has perforated and the peritoneal cavity is contaminated, there is a theoretical possibility of a mediastinitis but this is not a problem in practice.

Pyloroplasty

These are of several different types, for example Heineke–Mikulicz, Finney (gastroduodenostomy), and ulcer-excluding (Fig. 10). They all bypass or destroy the pyloric mechanism and so affect gastric emptying and duodenogastric reflux; once this has been destroyed, a good-sized pyloroplasty is probably better than a very small one, which could delay emptying. Pyloromyotomy, as is sometimes used in conjunction with oesophagectomy, is not usually appropriate when there is a marked duodenal ulcer.
Proximal gastric vagotomy

This is also referred to as parietal-cell or highly selective vagotomy, and is a modification of vagotomy that leaves the antrum innervated and thus avoids the need for a drainage procedure by leaving gastric emptying as normal as possible. It is more demanding technically because only small nerve branches are divided close to the lesser curve of the stomach. The only places that residual innervation can be left are at the lower and upper limits of dissection. Unless special intraoperative tests are used, the distal (antral) dissection should be less than 6 cm proximal to the pylorus on the lesser curve, leaving only the most distal branch from the nerve of Latarjet. The proximal dissection is carried 6 cm up the oesophagus and 6 cm out along the fundus towards the spleen. This may or may not entail dividing the first short gastric vessel (Fig. 11).

Fig. 10. Pyloroplasties: (a) Heineke–Mikulicz, (b) Finney, (c) ulcer-excluding.
Anterior seromyotomy and posterior truncal vagotomy

A modification in order to speed the operation was to cut the muscle of the lesser curve just in from the edge, and divide the nerve branches as they were just plunging in through the muscle. As this is difficult to do on the posterior surface, a posterior truncal vagotomy was performed. The operation is based on the principle that only the anterior nerve to the antrum need be preserved to ensure antral function. However, more recent anatomical dissections have found that, in about 3 per cent of patients, the only supply to the antrum is from the posterior nerves, such that they would get delayed gastric emptying. This operation was effective in the hands of those who were enthusiasts but did not gain general acceptance; although it is quicker to perform, it probably is not as sure in dividing all the branches as the more tedious standard dissection that divides all structures entering the lesser curve (Fig. 12).

Fig. 11. Completion of proximal gastric vagotomy showing denervation 6 cm up oesophagus, 6 cm out along fundus and 6 cm proximal to pylorus.
Laparoscopic procedures

Truncal vagotomy and gastroenterostomy has been performed laparoscopically and several small series have been reported of the more selective procedures. Seromyotomy is probably the most suitable for the laparoscopic approach. It is still an open question how much the method of access alters the metabolic response and there are not enough elective operations being done to mount a randomized, controlled trial. However, pre- and postoperative acid-secretion studies should be used to assess completeness of vagotomy, as this is essential to prevent recurrence.

Postoperative management

Apart from the general management of any patient after a major abdominal operation, the question the patients wish to have answered is when they can start to eat and drink! All gastric operations cause some degree of initial gastric stasis and it used to be the practice to keep a nasogastric tube in for some days afterwards. There is good evidence that this does no good and, in fact, probably does some harm. Very occasionally, acute dilatation of the stomach occurs after both vagotomy and resection, and presents as shock and effortless bilious vomiting. This must be recognized and a nasogastric tube passed immediately, while fluids are replaced intravenously.

After proximal gastric vagotomy, gastric emptying is nearly normal as there is no antral denervation, so the patients may drink in a few hours and take a soft diet the following day. All other gastric operations involve a suture line and after a gastrojejunal anastomosis the stomach tends to fill with bile. Sometimes a nasogastric tube is helpful in relieving nausea during this period, but otherwise the patient can start to increase fluid gradually from 48 h. When a significant proportion of the stomach has been removed, meals have initially to be little and often until the gastric remnant adapts, but no longer should patients be put on

Fig. 12. Lesser-curve seromyotomy: (a) extent of seromyotomy; (b) completed overlap repair.
strict 'gastric diets'; they should be encouraged to try a full, mixed diet in small quantities and just to avoid any particular food that upsets them.

**Alcohol**

After all operations for peptic ulcer except proximal gastric vagotomy, the more rapid emptying of liquids means that alcohol will be absorbed very rapidly into the circulation; therefore, even a small amount of alcohol quite soon gives high blood concentrations and the patient must be warned, ideally in writing, about the risk of driving or working machinery.

**Postvagotomy and postgastrectomy syndromes**

**Dumping**

This is associated with rapid emptying from the stomach. It is traditionally divided into early or late, but often the differentiation is not so clear-cut. It consists of a group of cardiovascular and gastrointestinal symptoms, such as faintness, sweating, tachycardia, bloating, nausea, and cramping abdominal pain.

**Early dumping**

Gastric emptying is normally regulated by duodenal osmoreceptors, but if the pylorus is divided or bypassed, hypertonic fluids can be 'dumped' into the upper small intestine. This leads to an outpouring of fluid into the small intestine to dilute the bowel contents, thereby reducing the blood volume. Whether or not a particular patient experiences cardiovascular symptoms may depend on how sensitive he/she is to slight changes in plasma volume. Gastrointestinal symptoms are due to the sudden release of gastrointestinal peptides such as cholecystokinin and motilin. Symptoms severe enough to interfere with normal activity occur in about 5 per cent of patients after vagotomy and drainage or partial gastrectomy, but another 10 per cent have milder symptoms. Symptoms tend to improve with the time from the operation as the upper gastrointestinal tract adapts.

Treatment is initially dietary by avoiding high-osmotic foods and separating drinking and eating. In severe cases, somatostatin injected subcutaneously just before meals may give dramatic release by blocking the output of peptides or hormones. Although this can be a nuisance and is expensive, it is no more troublesome than that experienced by diabetics who have regularly to self-administer insulin. Long-acting forms of somatostatin analogue are now available.

**Late dumping**

This is due to hypoglycaemia occurring about 2 h after a meal because of a large initial secretion of insulin in response to the high sugar load. The insulin and glucose concentrations can be measured and the phenomenon confirmed. The syndrome is less common than early dumping, and the dietary changes should be the same. However, the patient can also carry a glucose sweet, which can be taken as soon as the symptoms start,
to prevent a severe hypoglycaemia.

**Surgical treatment**

If the patient has a gastroenterostomy and a patent, intact pylorus, then just taking down
the gastroenterostomy will probably solve the problem. (One year after a truncal vagotomy
the drainage procedure is no longer needed because the vagal reflexes within the stomach
have adapted.) Repair of a pyloroplasty is possible, and in over 60 per cent of cases
improves the symptoms sufficiently for them not to interfere with the patient's normal
activity. The operation must be precise over an 10 to 12 mm bougie (Fig. 13) (12 mm is the
maximum resting diameter of the normal pylorus). The insertion of a reversed jejunal
segment between the stomach and jejunum may give temporary relief, but after a time the
antiperistaltic activity appears to cease.

![Gastroscope view of reconstituted pylorus.](image)

**Diarrhoea**

'Postvagotomy' diarrhoea is the most common postoperative symptom. Some change in
bowel habit (often for the better!) occurs in 70 per cent of patients, but severe diarrhoea
may affect 10 per cent of patients after truncal vagotomy and drainage, but only 1 per cent
after proximal gastric vagotomy. It is partly due to the removal of the parasympathetic
influence on the function of the fore- and midgut, but the drainage procedures are also
important. The increase in small-bowel fluid load and rapid release of gastrointestinal
peptides affecting the colon also play their part (exaggerated duodenocolic reflex). In addition, the hypoacidity of the stomach removes the barrier to bacterial entry into the bowel, which can cause enteritis. The role of bile salts is important, and if a patient has had a cholecystectomy and a truncal vagotomy and drainage at the same time, or separately, the incidence of diarrhoea is 40 per cent.

**Treatment**

Fortunately, we have good antidiarrhoeal drugs such as codeine phosphate or loperamide, but, despite this, 1 per cent of patients find the condition disabling. Bacterial overgrowth should be looked for using the breath test and treated if present. Cholestyramine, which binds bile salts, does have an effect on diarrhoea, although not on reflux symptoms. Surgery is occasionally needed and many types of reversed jejunal loops have been tried. Perhaps the most successful is the distal reversed ileal onlay graft (Cuschieri).

**Bile ‘reflux’**

The pylorus not only controls emptying but also prevents reflux of duodenal juice. When it is removed, destroyed or bypassed, there is reflux into the stomach (remnant); although bilirubin is the obvious culprit because of its colour, pancreatic juice is also important. When bile and pancreatic juice mix, lysolecithin is produced from lecithin. This, together with the bile salts, is damaging to the gastric mucosa. The symptoms are nausea, burning epigastric pain, bilious vomiting, and weight loss. As with the other syndromes, many people have the physiological changes without the symptoms and it is not known why some are ‘sensitive’ to duodenal juice in the stomach and others are not. Persistent reflux can damage the gastric mucosa causing foveolar hyperplasia, even in the absence of *H. pylori*. Duodenal juice can also reflux into the oesophagus and may be particularly dangerous there: it is probably linked with the intestinal metaplasia of Barrett's oesophagus, with its malignant potential. Nowadays, bile in the stomach and oesophagus can be verified by a semiquantitative bile probe (Bilitech) or it can be inferred from the sodium concentrations, detected by a sodium-sensitive probe. There is no direct correlation between symptoms and the degree of reflux, but mucosal damage can occur in the absence of symptoms.

**Treatment**

Cholestyramine seems to have little effect in binding bile salts in the stomach (as opposed to the colon) and this may relate to pH. However, prokinetic drugs such as metoclopramide or cisapride may help to clear the stomach and oesophagus, particularly if the antrum is still present.

**Revisional surgery**

This should only be done once the presence of significant reflux has been established, either by a history of repeated bilious vomiting or direct measurement. Pyloric reconstruction or the closure of a gastrojejunostomy are the first surgical measures if there has been no resection. After a Polya (Billroth II) gastrectomy, a Roux-en-Y reconstruction or Tanner Roux 19 is relatively easy to do without refashioning the anastomosis (Fig. 14). If
the Roux limb is at least 40 cm, reflux of duodenal juice is eliminated from the stomach, but sometimes the Roux limb empties poorly and patients must be warned that all symptoms may not go.

The dilemma for the surgeon is whether to add a vagotomy to prevent recurrent ulceration of the stoma in the unprotected Roux limb, or whether to rely on long-term, low-dose, acid-reducing drugs. Adding a vagotomy to the gastric remnant can make any delayed emptying worse. As the vagotomy can be technically difficult after a previous operation, drug treatment, particularly in the elderly patient, is probably best.

**Malignant change**

Twenty years after a gastric resection for benign disease, a patient has a 3.7-fold increased risk of developing carcinoma of the gastric remnant. When the operation is for gastric ulceration, the risk is 5.5, rising to 8.6 if the original procedure was a Billroth II (Polya). The frequency does not justify regular endoscopic follow-up, but any new symptom or anaemia long after a gastrectomy should be taken very seriously. The risk to the stomach and other organs may relate to the production of nitrosamines by bacteria in the relatively hypoacidic stomach remnant or due to long-term bile damage to the gastric mucosa leading to metaplasia and dysplasia.

**Delayed gastric emptying**

Delayed gastric emptying of solids can coexist with rapid emptying of liquids and persists in a few patients long after the early postoperative period. Solid emptying depends on antral contractions, whereas the early liquid emptying is produced by fundal pressure. After
vagotomy, especially if there has been some obstruction of the antral outlet, emptying may take many months to resolve. Patients, therefore, are advised to keep their meals as dry as possible and drink between meals, and to bite their meals up well.

**Treatment**

Further surgery does not help unless there is actual narrowing or obstruction at the outlet. Adding a gastrojejunostomy to a pyloroplasty merely adds more bile reflux to the poorly emptying stomach. Prokinetic drugs are helpful, for example cisapride, metoclopramide or erythromycin, and have even been found to give some benefit on the gastric remnant when the antrum has been removed.

**Stomal ulcer**

Ten years after vagotomy and gastroenterostomy or pyloroplasty, there is a 10 per cent chance of stomal ulcer, and after Polya (Billroth II) gastrectomy about 5 per cent where a vagotomy has not been added. After a vagotomy and antrectomy, recurrent ulcer is very rare. The persistent use of non-steroidal anti-inflammatory drugs correlates with this risk, whereas *Helicobacter* disappears after a drainage procedure when there is increased bile reflux, e.g. Polya partial gastrectomy.

**Nutritional problems**

The classic two-thirds partial gastrectomy leads to loss of weight in populations where the food is bulky and not high in calories because there is not enough space for sufficient food. Vagotomy, which left the stomach intact, was a significant improvement.

Late problems include iron, folate and vitamin B\textsubscript{12} deficiency as well as hypocalcaemia. Vitamin B\textsubscript{12} is usually absorbed if a fair gastric remnant has been left. It is important to check that the patients are eating a balanced diet. Persistent diarrhoea as steatorrhoea can lead to hypocalcaemia and malabsorption of fat and fat-soluble vitamins, especially when the duodenum is bypassed and the mixing of food with bile and pancreatic secretion is poor. It is prudent to check the calcium several years after peptic ulcer surgery.

**Conclusion**

For 100 years, surgery was the definitive treatment for chronic peptic ulcer. Over those years, operations were refined to make them more physiological and improvements in perioperative care made them increasingly safe and successful. But surgeons were often treating the secondary cause, that is, hypersecretion of acid, rather than the primary cause.

Today, gastric surgeons have the challenge of difficult decisions and difficult operations in the treatment of life-threatening emergencies affecting an increasingly elderly population. Surgeons must also be alert to early gastric malignancy associated with peptic ulcer, or its previous surgical treatment, as well as to the long-term complications of peptic ulcer surgery, which was performed frequently up to 10 to 15 years ago.

However, preliminary reports in the United States of America suggest that, now, 50 per
cent of gastroduodenal ulcers are not associated with \textit{H. pylori}. This and emerging antibiotic resistance by \textit{Helicobacter} is a warning to the surgical community not to lose the experience and skills so painfully learnt in the long years before \textit{H. pylori} was recognized.

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