

Schwartz's Surgery

Dempsey

PEPTIC ULCER DISEASE

Introduction

Peptic ulcers are focal defects in the gastric or duodenal mucosa which extend into the submucosa or deeper (Fig. 25-30). They may be acute or chronic, and ultimately are caused by an imbalance between the action of peptic acid and mucosal defenses (Fig. 25-31). Peptic ulcer remains a common outpatient diagnosis, but the number of physician visits, hospital admissions, and elective operations for peptic ulcer disease have decreased steadily and dramatically over the past 3 decades (Fig. 25-32). These trends all predated the advent of fiberoptic endoscopy, highly selective vagotomy, and the use of H₂-blockers. However, the incidence of emergency surgery and the death rate associated with peptic ulcers are fairly stable (

Fig. 25-33). These epidemiologic trends probably represent the net effect of several factors, including decreasing prevalence of *H. pylori* infection, better medical therapy, increases in outpatient management, and the use of NSAIDs and aspirin (with and without ulcer prophylaxis).

These epidemiologic facts notwithstanding, it is important to reiterate that peptic ulcer is a common disease in the U.S. In 2000, the total direct costs (hospital, physicians, and drugs) of peptic ulcer disease was about \$3.3 billion, with indirect costs (lost work and productivity) of over \$6 billion. The prevalence of peptic ulcer in the U.S. is about 2%, and the lifetime risk is about 10%. In 1998, the crude mortality rate for peptic ulcer was 1.7 per 100,000 individuals. Gastric ulcer has a higher mortality than duodenal ulcer because of its increased prevalence in the elderly. Recent studies have shown an increase in the rates of hospitalization and mortality in elderly patients for the peptic ulcer complications of bleeding and perforation. Presumably this is due to the increasingly common use of NSAIDs and aspirin in this elderly cohort, many of whom have *H. pylori* infection.

FIG. 25-30. Diagram of gastric erosions and ulcers. [*Reproduced with permission from Ashley SW, Evoy D, Daly JM: Stomach, in Schwartz SI (ed): Principles of Surgery, 7th ed. New York: McGraw-Hill, 1999, p 1181.*]

FIG. 25-31. Balance of acid/peptic aggressive factors and mucosal defensive factors in the gastric mucosa. (*Reproduced with permission from Mertz HR, Walsh JH: Peptic ulcer pathophysiology. Med Clin North Am 75:799, 1991.*)

FIG. 25-32. Physician visits for peptic ulcer. (*Reproduced with permission from Munnangi S, Sonnenberg A: Time trends of physician visits and treatment patterns of peptic ulcer disease in the United States. Arch Intern Med 157:1489, 1997.*)

FIG. 25-33. Death rate for peptic ulcer. (*Reproduced with permission from Donahue PE: Arch Surg 134:1373, 1999.*)

Pathophysiology and Etiology

A variety of factors may contribute to the development of peptic ulcer disease. Although it is now recognized that the large majority of duodenal and gastric ulcers are caused by *H. pylori* infection and/or NSAID use ([Fig. 25-34](#)), the final common pathway to ulcer formation is peptic acid injury of the gastroduodenal mucosal barrier. Thus the adage "no acid, no ulcer" remains true even today. Acid suppression, either with medication or surgery, remains a mainstay in healing both duodenal and gastric ulcers and in preventing recurrence. It generally is thought that *H. pylori* predisposes to ulceration, both by acid hypersecretion, and by compromise of mucosal defense mechanisms. NSAID use is thought to lead to peptic ulcer disease predominantly by compromise of mucosal defenses. Duodenal ulcer has typically been thought of as a disease of increased peptic acid action on the duodenal mucosa, whereas gastric ulcer has been viewed as a disease of weakened mucosal defenses in the face of relatively normal action of peptic acid. However, increased understanding of the pathophysiology of peptic ulcer has blurred this distinction. Clearly, weakened mucosal defenses play a role in many duodenal and most gastric ulcers (e.g., duodenal ulcer in an *H. pylori*-negative patient on NSAIDs or a patient with a typical type I gastric ulcer with acid hyposecretion), whereas increased aggressive activity of peptic acid may result in a duodenal or gastric ulcer in the setting of normal mucosal defenses (e.g., a duodenal ulcer in a patient with Zollinger-Ellison syndrome, or a gastric ulcer in a patient with gastric outlet obstruction, antral stasis, and acid hypersecretion).

Elimination of *H. pylori* infection or NSAID use is important for optimal ulcer healing, and perhaps is even more important in preventing ulcer recurrence and/or complications. A variety of other diseases are known to cause peptic ulcer, including Zollinger-Ellison syndrome (gastrinoma), antral G-cell hyperfunction and/or hyperplasia, systemic mastocytosis, trauma, burns, and major physiologic stress. Other causative agents include drugs (all NSAIDs, aspirin, and cocaine), smoking, alcohol, and psychologic stress. In the U.S., probably more than 90% of serious peptic ulcer complications can be attributed to *H. pylori* infection, NSAID use, or cigarette smoking.

Helicobacter pylori Infection

Helicobacter pylori is uniquely equipped for survival in the hostile environment of the stomach. It possesses the enzyme urease, which converts urea into ammonia and bicarbonate, thus creating an environment around the bacteria that buffers the acid secreted by the stomach. Mutant strains of *H. pylori* that do not produce urease are unable to colonize the stomach. The organism lives in the mucus layer atop the gastric surface epithelial cells, and some attach to these cells. There are a variety of possible mechanisms whereby *H. pylori* may produce mucosal injury ([Table 25-6](#)). One fundamental mechanism appears to be a disturbance in acid secretion. This is due at least in part to the inhibitory effect that *H. pylori* exerts on antral D cells that secrete somatostatin, a potent inhibitor of antral G-cell gastrin production. *H. pylori* infection is associated with decreased levels of somatostatin, decreased somatostatin messenger ribonucleic acid production, and fewer somatostatin-producing D cells. These effects are probably

mediated by *H. pylori*-induced local alkalization of the antrum (antral acidification is the most potent antagonist to antral gastrin secretion), and *H. pylori*-mediated increases in other local mediators and cytokines, among other means. The end result is hypergastrinemia and acid hypersecretion (Fig. 25-35). This hypergastrinemia presumably leads to the parietal cell hyperplasia seen in many patients with duodenal ulcer. The acid hypersecretion and the antral gastritis are thought to lead to antral epithelial metaplasia in the postpyloric duodenum. This duodenal metaplasia allows *H. pylori* to colonize the duodenal mucosa, and in these patients the risk of developing a duodenal ulcer increases 50-fold. When *H. pylori* colonizes the duodenum, there is a significant decrease in acid-stimulated duodenal bicarbonate release. When *H. pylori* infection is successfully treated, acid secretory physiology tends to normalize (Fig. 25-36). Other mechanisms whereby *H. pylori* can induce gastroduodenal mucosal injury include the production of toxins (*vacA* and *cagA*), local elaboration of cytokines (particularly interleukin 8) by infected antral mucosa, recruitment of inflammatory cells and release of inflammatory mediators, and production of immunoglobulins. It is likely that *H. pylori* predisposes to gastric ulcer at least in part by weakening mucosal defenses.

The evidence supporting the central role of *H. pylori* in the pathophysiology of peptic ulcer disease is strong. Patients with *H. pylori* infection and antral gastritis are three and a half times more likely to develop peptic ulcer disease than patients without *H. pylori* infection. Up to 90% of patients with duodenal ulcers, and 70 to 90% of patients with gastric ulcers, have *H. pylori* infection. It is clear from multiple randomized prospective studies that curing *H. pylori* infection dramatically alters the natural history of peptic ulcer disease, decreasing the recurrent ulcer rate from over 75% in patients treated with a course of acid-suppressive therapy alone (in whom *H. pylori* is not eradicated) to less than 20% in patients treated with a course of antibacterial therapy (Fig. 25-37).

Obviously, other factors are involved in the etiology of peptic ulcer disease, since everyone who has *H. pylori* (up to 50% of the adult population in some areas of the U.S.) does not get peptic ulcer disease. Only about 15 to 20% of patients colonized with *H. pylori* will develop peptic ulcer disease over their lifetime. Many patients on aspirin and NSAIDs develop peptic ulcer disease without *H. pylori* infection. These observations notwithstanding, it is clear from a variety of well-designed laboratory, clinical, and epidemiologic studies that *H. pylori* is indubitably an important factor in the development and recurrence of peptic ulcer disease. *H. pylori* also plays an etiologic role in gastric cancer and lymphoma (see below).

Acid Secretion and Peptic Ulcer

A variety of abnormalities related to mucosal acid exposure have been described in patients with duodenal ulcer (Fig. 25-38). As a rule, duodenal ulcer patients secrete more acid than patients with gastric ulcer. It has long been recognized that duodenal ulcer patients as a group have a higher mean BAO and also a higher mean MAO compared to normal controls (Fig. 25-39). Nocturnal acid secretion is more commonly elevated than daytime secretion. However, many duodenal ulcer patients have basal and peak acid outputs in the normal range, and there is no correlation between acid secretion and the severity of the ulcer disease. Duodenal ulcer patients produce more acid than normal

controls in response to any known secretory stimulus for gastric acid output. Although duodenal ulcer patients usually have normal fasting gastrin concentrations, they often produce more gastric acid at any given dose of gastrin than controls. Considering that many duodenal ulcer patients do produce excessive gastric acid, it has been argued that a "normal" fasting gastrin level in these patients is inappropriately high, and that there is an impaired feedback mechanism, especially in light of the apparently increased sensitivity of the parietal cell mass to gastrin. Many of these long-standing observations now seem reasonable in light of recently gained understanding of the perturbations in acid and gastrin secretion associated with *H. pylori* infection (described above). Some patients with duodenal ulcer also have increased rates of gastric emptying that delivers an increased acid load per unit of time to the duodenum. Finally, the buffering capacity of the duodenum in many patients with duodenal ulcer is compromised due to decreased duodenal bicarbonate secretion.

In patients with gastric ulcer, acid secretion is variable. Generally, four types of gastric ulcer are described. The most common, Johnson type I gastric ulcer, is typically located near the angularis incisura on the lesser curvature, close to the border between the antrum and the body of the stomach. These patients usually have normal or decreased acid secretion. Type II gastric ulcer is associated with active or quiescent duodenal ulcer disease, and type III gastric ulcer is prepyloric. Both type II and type III gastric ulcers are associated with normal or increased gastric acid secretion. Type IV gastric ulcers occur near the gastroesophageal junction, and acid secretion is normal or below normal. Patients with type I or IV gastric ulcers may have weak mucosal defenses that permit an abnormal amount of injurious acid back-diffusion into the mucosa. Duodenogastric reflux may play a role in weakening the gastric mucosal defenses in patients with gastric ulcer. A variety of components in duodenal juice, including bile, lysolecithin, and pancreatic juice, have been shown to cause injury and inflammation in the gastric mucosa. NSAIDs and aspirin have similar effects. Although chronic gastric ulcer is usually associated with surrounding gastritis, it is unproven that the latter leads to the former.

Nonsteroidal Anti-Inflammatory Drugs in Peptic Ulcer Disease. NSAIDs (including aspirin) are inextricably linked to peptic ulcer disease. Patients with rheumatoid arthritis and osteoarthritis who take NSAIDs have a 15 to 20% annual incidence of peptic ulcer, and the prevalence of peptic ulcer in chronic NSAID users is about 25% (15% gastric and 10% duodenal). Complications of peptic ulcer disease (specifically hemorrhage and perforation) are much more common in patients taking NSAIDs. More than half of patients who present with peptic ulcer hemorrhage or perforation report the recent use of NSAIDs, including aspirin. Many of these patients remain asymptomatic until they develop these life-threatening complications.

The overall risk of significant serious adverse GI events in patients taking NSAIDs is more than three times that of controls ([Table 25-7](#)). This risk increases to five times in patients over age 60. In elderly patients taking NSAIDs, the likelihood that they will require an operation related to a GI complication is 10 times that of the control group, and the risk that they will die from a GI cause is about four and a half times higher. This problem is put into perspective when one realizes that approximately 20 million patients in the United States take NSAIDs on a regular basis; perhaps as many regularly take

aspirin. Persons who take NSAIDs also have a higher hospitalization rate for serious GI events than those who do not.

Factors that clearly put patients at increased risk for NSAID-induced GI complications include age over 60, prior GI event, high NSAID dose, concurrent steroid intake, and concurrent anticoagulant intake. *Any patient taking NSAIDs or aspirin who has one or more of these risk factors should receive concomitant acid suppressive medication or misoprostol at a therapeutic dose, or should be considered for alternative treatment with cyclooxygenase 2 (COX-2) inhibitors (Table 25-8).*

Smoking, Stress, and Other Factors

Epidemiologic studies suggest that smokers are about twice as likely to develop peptic ulcer disease as nonsmokers. Smoking increases gastric acid secretion and duodenogastric reflux. Smoking decreases both gastroduodenal prostaglandin production and pancreaticoduodenal bicarbonate production. These observations may be related, and any or all could explain the observed association between smoking and peptic ulcer disease.

Although difficult to measure, both physiologic and psychologic stress undoubtedly play a role in the development of peptic ulcer in some patients. In 1842, Curling described duodenal ulcer and/or duodenitis in burn patients. Decades later, Cushing described the appearance of acute peptic ulceration in patients with head trauma (Cushing ulcer). Even the ancients recognized the undeniable links between peptic ulcer disease and stress. Patients still present with ulcer complications (bleeding, perforation, and obstruction) that are seemingly exacerbated by stressful life events. Recently, the use of crack cocaine has been linked to juxtapyloric peptic ulcers with a propensity to perforate. Alcohol is commonly mentioned as a risk factor for peptic ulcer disease, but confirmatory data are lacking.

FIG. 25-34. Causes of peptic ulcer disease. [*Reproduced with permission from Spechler SJ: Peptic ulcer disease and its complications in Feldman M (ed): Sleisinger and Fordtran's Gastrointestinal and Liver Disease, 7th ed. Philadelphia: Saunders, 2002, p 747.*]

FIG. 25-35. Model of *H. pylori*-induced effects on duodenal ulcer pathogenesis. (*Reproduced with permission from Peek RM Jr., Blaser MJ: Pathophysiology of Helicobacter pylori-induced gastritis and peptic ulcer disease. Am J Med 102:200, 1997.*)

FIG. 25-36. Basal acid output in normal controls and in patients with duodenal ulcer, before and after the eradication of *H. pylori* infection.

FIG. 25-37. Treatment of *H. pylori* dramatically decreases the recurrence rate of duodenal and gastric ulcer. [*Reproduced with permission from Graham DY, Lew GM, Klein PD, et al: Effect of treatment of Helicobacter pylori infection on the long-term recurrence of gastric or duodenal ulcer; a randomized controlled study. Ann Intern Med 116:705, 1992.*]

FIG. 25-38. Frequency of physiologic abnormalities in patients with duodenal ulcer. [Reproduced with permission from Del Valle J, Chey WD, Scheiman JM: *Acid peptic disorders*, in Yamada T et al (eds): *Textbook of Gastroenterology*, 4th ed., Philadelphia: Lippincott Williams & Wilkins, 2003, p 1321.]

FIG. 25-39. Basal and peak acid output in normal (open circles) and duodenal ulcer (closed circles) patients. (Reproduced with permission from Blair AJ, et al: *JCI* 79:582, 1987.)

Clinical Manifestations

Over 90% of patients with peptic ulcer disease complain of abdominal pain. The pain is typically nonradiating, burning in quality, and located in the epigastrium. The mechanism of the pain is unclear. Patients with duodenal ulcer usually experience pain 2 to 3 hours after a meal and at night. Two thirds of patients with duodenal ulcers will complain of pain that awakens them from sleep. The pain of gastric ulcer more commonly occurs with eating and is less likely to awaken the patient at night. A history of peptic ulcer disease, use of NSAIDs, over-the-counter antacids, or antisecretory drugs, is suggestive of the diagnosis. Other signs and symptoms include nausea, bloating, weight loss, stool positive for occult blood, and anemia. Duodenal ulcer is about twice as common in men compared to women, but the incidence of gastric ulcer is similar in men and women. On average, gastric ulcer patients are 10 years older than duodenal ulcer patients, and the incidence is increasing in the elderly, probably because of increasing NSAID use in this cohort with a high incidence of *H. pylori* infection.

Diagnosis

In the young patient with dyspepsia and/or epigastric pain, it may be appropriate to initiate empiric therapy for peptic ulcer disease without confirmatory testing. All patients over 45 with the above symptoms should have an upper endoscopy, and all patients, regardless of age, should have this study if any alarm symptoms (see [Table 25-4](#)) are present. A double contrast upper GI x-ray study may be useful. All gastric ulcers should be adequately biopsied, and any sites of gastritis should be biopsied to rule out *H. pylori*, and for histologic evaluation. Additional testing for *H. pylori* may be indicated. Although somewhat controversial, it is not unreasonable to test all peptic ulcer patients for *H. pylori*. A baseline serum gastrin level is appropriate to rule out gastrinoma.

Complications

The three most common complications of peptic ulcer disease, in decreasing order of frequency, are bleeding, perforation, and obstruction. Most peptic ulcer-related deaths in the U.S. are due to bleeding. Bleeding peptic ulcers account for about half of the clinically significant cases of upper GI bleeding at most medical centers ([Table 25-9](#)). Patients with a bleeding peptic ulcer typically present with melena and/or hematemesis. Nasogastric aspiration is usually confirmatory of the upper GI bleeding. Abdominal pain is quite uncommon. Shock may be present, necessitating aggressive resuscitation and blood transfusion. Early endoscopy is important to diagnose the cause of the bleeding and to assess the need for hemostatic therapy.

Three quarters of the patients who come to the hospital with bleeding peptic ulcer will stop bleeding if given acid suppression and kept NPO (non per os, meaning nothing by mouth). However, one fourth will continue to bleed or will rebleed after an initial quiescent period, and virtually all the mortalities (and all the operations for bleeding) occur in this group. This group can be fairly well delineated based on clinical factors related to the magnitude of the hemorrhage and endoscopic findings ([Table 25-10](#)). Shock, hematemesis, transfusion requirement exceeding four units in 24 hours, and high-risk endoscopic stigmata (active bleeding or visible vessel) define this high-risk group. These patients benefit from endoscopic therapy to stop the bleeding. The most common endoscopic hemostatic modalities used are injection with epinephrine, and electrocautery. Persistent bleeding or rebleeding after endoscopic therapy is an indication for operation, although repeat endoscopic treatment has been successful in treating rebleeding. Elderly and high-risk patients do not tolerate repeated episodes of hemodynamically significant hemorrhage, and may benefit from early elective operation after initially successful endoscopic treatment, especially if they have one or more of the risk factors mentioned above or a high-risk ulcer. Planned surgery under controlled circumstances often yields better outcomes than emergent surgery performed in the middle of the night. Deep bleeding ulcers on the posterior duodenal bulb or lesser gastric curvature are high-risk lesions, because they often erode large arteries not amenable to nonoperative treatment, and early operation should be considered.

Perforated peptic ulcer usually presents as an acute abdomen. The patient can often give the exact time of onset of the excruciating abdominal pain. Initially, a chemical peritonitis develops from the gastric and/or duodenal secretions, but within hours a bacterial peritonitis supervenes. Fluid sequestration into the third space of the inflamed peritoneum can be impressive, and fluid resuscitation is mandatory. The patient is in obvious distress, and the abdominal exam shows peritoneal signs. Usually marked involuntary guarding and rebound tenderness is evoked by a gentle examination. Upright chest x-ray shows free air in about 80% of patients ([Fig. 25-40](#)). Once the diagnosis has been made, the patient is given analgesia and antibiotics, resuscitated with isotonic fluid, and taken to the operating room. Rarely, the perforation has sealed spontaneously by the time of presentation, and surgery can be avoided. Nonoperative management is appropriate only if there is objective evidence that the leak has sealed (i.e., radiologic contrast study), and in the absence of clinical peritonitis.

Gastric outlet obstruction occurs in no more than 5% of patients with peptic ulcer disease. It is usually due to duodenal or prepyloric ulcer disease, and may be acute (from inflammatory swelling and peristaltic dysfunction) or chronic (from cicatrix). Patients typically present with nonbilious vomiting and may have a profound hypokalemic hypochloremic metabolic alkalosis. Pain or discomfort is common. Weight loss may be prominent, depending on the duration of symptoms. Initial treatment is nasogastric suction, intravenous hydration and electrolyte repletion, and antisecretory medication. The diagnosis is confirmed by endoscopy. Cancer must be ruled out. Currently, most patients admitted to the hospital with obstructing ulcer disease require intervention, either balloon dilation or operation.

FIG. 25-40. Pneumoperitoneum on upright chest x-ray in patient with perforated ulcer.

Medical Treatment

Patients with peptic ulcer disease should stop smoking and avoid alcohol and NSAIDs (including aspirin). If *H. pylori* infection is documented, it should be treated with one of several acceptable regimens ([Table 25-11](#)). Infectious disease consultation may be helpful in the compliant, symptomatic patient with persistent *H. pylori* infection following treatment; or another regimen could be tried (e.g., quadruple therapy). If initial *H. pylori* testing is negative, the ulcer patient may be treated with H₂-receptor blockers or proton pump inhibitors. Sucralfate or misoprostol may also be effective. If ulcer symptoms persist, an empiric trial of anti-*H. pylori* therapy is reasonable (false-negative *H. pylori* tests are common). Generally, antisecretory therapy can be stopped after 3 months if the ulcerogenic stimulus (usually *H. pylori*, NSAIDs, or aspirin) has been removed.

However, long-term maintenance therapy for peptic ulcer should be considered in all patients admitted to hospital with an ulcer complication, all high-risk patients on NSAIDs or aspirin (the elderly or debilitated), and all patients with a history of recurrent ulcer or bleeding. Consideration should be given to maintenance therapy in refractory smokers with a history of peptic ulcer. Misoprostol, sucralfate, and acid suppression may be quite comparable in many of these groups, but misoprostol may cause diarrhea and cramps, and cannot be used in women of childbearing age because of its abortifacient properties.

Surgical Treatment

The indications for surgery in peptic ulcer disease are bleeding, perforation, obstruction, and intractability or nonhealing. Gastric cancer must always be considered in gastric ulcer, whereas malignancy is almost never an issue in duodenal ulcer. Fundamentally, the vast majority of peptic ulcers are adequately treated by a variant of one of the three basic operations: highly selective vagotomy, vagotomy and drainage, and vagotomy and distal gastrectomy.

Highly Selective Vagotomy

Highly selective vagotomy (HSV), also called parietal cell vagotomy or proximal gastric vagotomy, is safe (mortality risk <0.5%) and causes minimal side effects. The operation severs the vagal nerve supply to the proximal two thirds of the stomach, where essentially all the parietal cells are located. It preserves the vagal innervation to the antrum and pylorus, and the remaining abdominal viscera ([Fig. 25-41](#)). Inadequate denervation of the proximal stomach due to technical error may lead to inadequate acid suppression and an unacceptably high incidence of ulcer recurrence. HSV decreases total gastric acid secretion by about 65 to 75%, which is quite comparable to the reduction seen with truncal vagotomy and acid-suppressive medication. Gastric emptying of solids is typically normal in patients after parietal cell vagotomy; liquid emptying may be normal or increased due to decreased compliance associated with loss of receptive relaxation and

accommodation. When applied to uncomplicated duodenal ulcer, the recurrence rate is higher with HSV than with vagotomy and antrectomy. However, our increased understanding of the pathophysiologic role of *H. pylori* and NSAIDs in the development of recurrent ulcer may mitigate this concern. HSV has not performed particularly well as a treatment for type II (gastric and duodenal) and III (prepyloric) gastric ulcer, perhaps because of hypergastrinemia caused by gastric outlet obstruction and persistent antral stasis.

HSV was accepted into the surgical armamentarium largely as a treatment for uncomplicated, intractable duodenal ulcer. Although the operation has been shown to be effective in treating selected patients with complicated peptic ulcer, its usefulness in this regard remains suspect for two reasons. First, many surgeons feel that complicated ulcer disease may call for a more radical operation than uncomplicated disease (a hypothesis that has not been proven). Second, HSV was conceived as an ulcer operation that preserves the pylorus and does not involve opening the GI tract. Most patients with complicated peptic ulcer disease need an ancillary procedure that invalidates these two technical advantages of HSV (e.g., pyloroduodenotomy to oversew a bleeding duodenal ulcer, or gastrojejunostomy to bypass an obstruction).

The Taylor procedure (anterior seromyotomy and posterior truncal vagotomy) is an attractive and simple alternative to HSV. This operation appears to have a similarly low incidence of side effects, and an acceptably low incidence of recurrent ulceration. Effective acid suppression is achieved and normal gastric emptying is maintained. Posterior truncal and anterior HSV are comparable. Although formal HSV is readily accomplished as a laparoscopic procedure, these shortcut operations are particularly attractive to the laparoscopic surgeon, and merit consideration.

Vagotomy and Drainage

Truncal vagotomy and pyloroplasty, and truncal vagotomy and gastrojejunostomy are the paradigmatic vagotomy and drainage (V+D) procedures. However, selective vagotomy and drainage, and HSV and gastrojejunostomy may be useful ulcer operations in selected patients. The advantage of V+D is that it can be performed safely and quickly by the experienced surgeon. The main disadvantages are the side effect profile (10% of patients have significant dumping and/or diarrhea), and a 10% recurrent ulcer rate. Whether the incidence of these postoperative problems (heretofore determined by studies predominantly involving patients with intractable uncomplicated duodenal ulcer) will be different in the current era, with our improved knowledge of complicated ulcer, *H. pylori*, and NSAIDs, is unknown. A serious attempt should be made to perform a complete truncal vagotomy ([Fig. 25-42](#)), keeping in mind that in many patients there are more than two vagal trunks at the esophageal hiatus. During truncal vagotomy, care must be taken not to perforate the esophagus, a potentially lethal complication. Intraoperative frozen section confirmation of at least two vagal trunks is prudent. Unlike HSV, V+D is widely accepted as a successful operation for complicated peptic ulcer disease. It has been described as a useful part of the operative treatment for bleeding duodenal and gastric ulcer, perforated duodenal and gastric ulcer, and obstructing duodenal and gastric (type II and III) ulcer. When applied to gastric ulcer, the ulcer should be excised or biopsied.

Truncal vagotomy denervates the antropyloric mechanism, and therefore some sort of procedure is necessary to ablate or bypass the pylorus; otherwise gastric stasis often results. Gastrojejunostomy is a good choice in patients with gastric outlet obstruction or a severely diseased proximal duodenum. The anastomosis is done between the proximal jejunum and the most dependent portion of the greater gastric curvature, in either an antecolic ([Fig. 25-43](#)) or retrocolic fashion ([Fig. 25-44](#)). On the other hand, pyloroplasty is useful in some patients who require a pyloroduodenotomy to deal with the ulcer complication (e.g., posterior bleeding duodenal ulcer), in those with limited or focal scarring in the pyloric region, or when gastrojejunostomy is technically difficult. The most commonly performed pyloroplasty is the Heineke-Mikulicz type, which closes a longitudinal transpyloric incision in a transverse fashion ([Fig. 25-45](#)). Other occasionally useful techniques include the Finney ([Fig. 25-46](#)) and the Jaboulay pyloroplasties ([Fig. 25-47](#)). These more extensive pyloroplasty techniques may make subsequent distal gastric resection more difficult and/or hazardous.

Vagotomy and Antrectomy

The advantages of vagotomy and antrectomy (V+A) are the extremely low ulcer recurrence rate and the applicability of the operation to many patients with complicated peptic ulcer disease (e.g., bleeding duodenal and gastric ulcer, obstructing peptic ulcer, nonhealing gastric ulcer, and recurrent ulcer). When applied to gastric ulcer disease, the resection is usually extended far enough proximally to include the ulcer. The disadvantage of V+A is the somewhat higher operative mortality rate when compared with HSV or V+D. Following antrectomy, gastrointestinal continuity may be re-established, either via a Billroth I gastroduodenostomy ([Fig. 25-48](#)) or a Billroth II loop gastrojejunostomy ([Fig. 25-49](#)). Since antrectomy routinely leaves a 60 to 70% gastric remnant, reconstruction as a Roux-en-Y gastrojejunostomy should be avoided ([Fig. 25-50](#)). The Roux-en-Y operation is an excellent procedure for keeping duodenal contents out of the stomach and esophagus. However, in the presence of a large gastric remnant, this reconstruction will predispose to marginal ulceration and/or gastric stasis.

Although it is *not* clear that the long-term morbidity with vagotomy and antrectomy is greater than with truncal vagotomy and drainage, it *is* clear that the operative mortality rate is somewhat higher with the resectional procedure. Antrectomy should thus be avoided in hemodynamically unstable patients. It should also be avoided in patients with extensive inflammation and/or scarring of the proximal duodenum, since secure anastomosis (Billroth I) or duodenal closure (Billroth II) may be compromised.

Distal Gastrectomy

Distal gastrectomy without vagotomy (usually about a 50% gastrectomy to include the ulcer) has traditionally been the procedure of choice for type I gastric ulcer. Reconstruction may be done as a Billroth I (preferable) or Billroth II. Truncal vagotomy is added for type II and III gastric ulcers, or if the patient is believed to be at increased risk for recurrent ulcer, and should be considered if Billroth II reconstruction is contemplated. Though not routinely used today in the surgical treatment of peptic ulcer, subtotal gastrectomy (75% distal gastrectomy) without vagotomy may be an appealing

choice for an occasional ulcer patient. Periesophageal dissection is avoided (vagotomy is unnecessary if 75% gastrectomy is performed), and extensive periduodenal dissection is minimized (Billroth II is the reconstruction of choice). Finally, concomitant gastric ulcers (type II or III) are resected. However, subtotal gastrectomy is rarely the first operation of choice for any patient with duodenal ulcer, since it leaves an inadequate gastric reservoir, and since vagotomy and antrectomy has a lower recurrent ulcer rate, is at least as safe, and has a similar side effect profile.

Choice of Operation for Peptic Ulcer

The choice of operation for the individual patient with peptic ulcer disease depends on a variety of factors, including the type of ulcer (duodenal, gastric, recurrent, or marginal), the indication for operation, and the condition of the patient, among others. Other important considerations are intra-abdominal factors (duodenal scarring/inflammation, adhesions, or difficult exposure), the ulcer diathesis status of the patient, the surgeon's experience and personal preference, whether *H. pylori* infection is present, the need for NSAID therapy, previous treatment, and the likelihood of future compliance with treatment. [Table 25-12](#) shows the surgical options for managing various aspects of peptic ulcer disease. In general, resective procedures have a lower ulcer recurrence rate, but a higher operative morbidity and mortality rate ([Table 25-13](#)) when compared to nonresective ulcer operations. Because ulcer recurrence is often related to *H. pylori* and/or NSAIDs, it is usually managed adequately without reoperation. Thus, gastric resection to minimize recurrence in duodenal ulcer disease is often not justified today; resection for gastric ulcer remains the standard because of the risk of cancer.

FIG. 25-41. Highly selective vagotomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-42. Truncal vagotomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-43. Antecolic gastrojejunostomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-44. Retrocolic gastrojejunostomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-45. Heineke-Mikulicz pyloroplasty. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-46. Finney pyloroplasty. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-47. Jaboulay pyloroplasty. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-48. Billroth I gastroduodenostomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-49. Billroth II antecolic gastrojejunostomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

FIG. 25-50. Roux-en-Y gastrojejunostomy. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

Bleeding

Bleeding is the most common cause of ulcer-related death. Recently, the total number of operations for bleeding peptic ulcer in the U.S. appears to have increased, as has the ratio of operations for bleeding ulcer to total ulcer operations. It is not known if or how endoscopic therapy, *H. pylori* treatment, and/or antisecretory drug use will affect this trend, which is no doubt due in part to the aging of the population and the epidemic of NSAID use. Although the surgeon treating patients with bleeding peptic ulcer disease needs to know when to operate and what operation to perform, the success of nonsurgical therapy in treating and preventing bleeding peptic ulcer disease has resulted in the selection of a subgroup of high-risk patients for today's surgeon. It is certainly likely that patients currently coming to operation for bleeding peptic ulcer disease are at higher risk, and thus more likely to have a poor surgical result than ever before. The surgical options for treating bleeding peptic ulcer disease include suture ligation of the bleeder (and biopsy for gastric ulcer); suture ligation and definitive nonresective ulcer operation (HSV or V+D); and gastric resection (usually including vagotomy and ulcer excision).

Bleeding Duodenal Ulcer

Indications for operation for bleeding duodenal ulcers are massive hemorrhage that is unresponsive to endoscopic control, and transfusion requirement of more than 4 to 6 units of blood, despite attempts at endoscopic control. Lack of availability of a therapeutic endoscopist, recurrent hemorrhage after one or more attempts at endoscopic control, lack of availability of blood for transfusion, repeat hospitalization for bleeding duodenal ulcer,

and concurrent indications for surgery such as perforation or obstruction, also are indications for surgery. The mortality rate for surgery for bleeding duodenal ulcer is 10 to 20%. Early operation should be considered in patients over 60 years of age, those presenting in shock, those requiring more than 4 units of blood in 24 hours or 8 units of blood in 48 hours, those with rebleeding, and those with ulcers greater than 2 cm in diameter or strategically located as described above.

The two operations most commonly used for bleeding duodenal ulcer are V+D combined with oversewing of the ulcer, or V+A. The trade-off appears to be an increased risk of rebleeding with V+D, compared to the increased operative mortality of V+A. When the mortality for reoperation for rebleeding is considered, the overall mortality is probably comparable for the two approaches. Patients who are in shock or medically unstable should not have gastric resection.

An initial pyloromyotomy incision allows access to the bleeding posterior duodenal ulcer, and an expeditious Kocher maneuver allows the surgeon to control the hemorrhage with the left hand if necessary. Heavy suture material on a stout needle is used to place figure-of-eight sutures or a U-stitch in order to secure the bleeding vessel at the base of the posterior duodenal ulcer. Multiple sutures are usually necessary. Once the surgeon is unequivocally convinced that hemostasis is secure, a pyloroplasty can be performed. A truncal vagotomy completes the operation. If V+A is selected, smaller ulcers are resected with the specimen, but larger bleeding duodenal ulcers must often be left behind in the duodenal stump. If this is the case, suture hemostasis must be attained and a secure duodenal closure accomplished. The anterior wall of the open duodenum can be sutured to either the proximal or distal lip of the posterior ulcer once the bleeding vessel has been sutured. The duodenal closure can be buttressed with omentum and the duodenum should be decompressed, either with a lateral duodenostomy or retrograde duodenostomy tube via the proximal jejunum. Use of a feeding jejunostomy is also considered. A Billroth II anastomosis is preferred because it avoids the extensive mobilization necessary for Billroth I, and because it keeps the ulcer out of the acid stream if it is left behind.

Bleeding Gastric Ulcer

Bleeding gastric ulcers tend to occur in older and/or medically complicated patients, and this fact tends to increase the operative risk. Although this has been used by some as an excuse not to operate early on these patients, experience shows that planned surgery in a resuscitated patient results in a better operative survival rate than emergent operation in a patient who has rebled and is in shock. Patients with gastric ulcer bleeding who are most likely to require surgery have bled more than 6 units and have presented in shock. Endoscopically, their ulcers tend to be on the lesser curvature with the usual stigmata of recent hemorrhage. Distal gastric resection to include the bleeding ulcer is the procedure of choice for bleeding gastric ulcer. Second best is V+D with oversewing and biopsy of the ulcer. Oversewing of the bleeder followed by long-term acid suppression is a reasonable alternative in extremely high-risk patients. The specter of cancer is ever present in the patient with gastric ulcer, whether it is bleeding or not.

Perforation

Perforation is the second most common complication of peptic ulcer. As with bleeding peptic ulcer disease, NSAID use has also been inextricably linked with perforated peptic ulcer disease, especially in the elderly population. Well over 20% of patients over the age of 60 presenting with a perforated ulcer are taking NSAIDs at the time of perforation. The mortality rate for perforated gastric ulcer is higher than that for duodenal ulcer, because the group with the former malady tend to be older and sicker. Surgery is almost always indicated, although occasionally nonsurgical treatment can be used in the stable patient without peritonitis, and in whom radiologic studies document a sealed perforation. Patients with acute perforation and GI blood loss (either chronic or acute) should be suspected of having a posterior "kissing" ulcer, and the appropriate definitive operation should then be performed.

The options for surgical treatment of perforated duodenal ulcer are simple patch closure, patch closure and HSV, or patch closure and V+D. Simple patch closure alone should be done in patients with hemodynamic instability and/or exudative peritonitis signifying a perforation over 24 hours old. In all other patients the addition of a definitive ulcer operation (HSV or V+D) should be considered. Numerous studies have reported a negligible mortality with this approach. Although in the U.S. and Western Europe there is clearly a trend away from definitive operation for perforated duodenal ulcer, it still seems prudent to add HSV to most stable patients with a perforated duodenal ulcer, especially in those with a chronic history, and in those who are unlikely to be compliant with *H. pylori* treatment or who require NSAIDs. In the pre-*H. pylori* era, it was shown that only 30% of patients with perforated duodenal ulcer treated by simple closure had good long-term results. Early data now suggest that simple closure of perforated duodenal ulcer may achieve satisfactory long-term results when *H. pylori* infection (present in 50 to 75% of patients with perforated duodenal ulcer) is eliminated. However, it sometimes is difficult to determine the *H. pylori* status of the patient having emergent operation for perforated ulcer. Furthermore it is doubtful whether many of these patients will comply with the medication regimen required to eradicate *H. pylori*. Therefore, using possible *H. pylori* infection as an excuse not to do a definitive ulcer operation in any patient with perforated duodenal ulcer is irrational. While it must be acknowledged that laparoscopic patch repair for perforated ulcer is commonly done without definitive surgery, perhaps laparoscopic HSV or some variant thereof can be recommended for those patients who have persistent or recurrent ulcer symptoms.

Perforated gastric ulcer carries a higher mortality rate than perforated duodenal ulcer (10 to 40%). This is generally thought to be due to the gastric ulcer patients' more advanced age, increased medical comorbidities, delay in seeking medical attention, and the larger size of gastric ulcers. Perforated gastric ulcers are associated with NSAID and tobacco use, and often present without prior symptoms. All perforated gastric ulcers are best treated by distal gastric resection with or without truncal vagotomy, depending upon ulcer type. Vagotomy is usually performed for type II and III gastric ulcers. Patch closure with biopsy; or local excision and closure; or biopsy, closure, truncal vagotomy, and drainage are alternative operations. All perforated gastric ulcers, even those in the prepyloric position, should be biopsied if they are not removed at surgery. HSV should generally not be used for perforated gastric ulcer. Simple patch closure has been shown to have a higher short-term complication rate (20 vs. 5%) and a higher ulcer recurrence rate

(25 vs. 10%) than distal gastrectomy. The latter often eradicates *H. pylori*, which could partially explain this finding; other authors have cited lower ulcer recurrence rates following simple patch closure of perforated gastric ulcer with effective eradication of *H. pylori*. However, it should be recognized that many patients with perforated gastric ulcer are *H. pylori* negative.

Obstruction

Currently, gastric outlet obstruction is the least common indication for operation in peptic ulcer disease. Acute ulcers associated with obstruction due to edema and/or motor dysfunction may respond to intensive antisecretory therapy and nasogastric suction. But most patients with significant obstruction from chronic ulceration will require some sort of more substantial intervention. Endoscopic balloon dilation can often transiently improve obstructive symptoms, but many of these patients ultimately fail and come to operation. Even in patients who have a successful initial dilation, at least half will require subsequent surgery, usually for recurrent obstruction, although interval bleeding and perforation is also a possibility. Some investigators have speculated that effective treatment of *H. pylori* in patients with obstructing peptic ulcer disease will improve the results of balloon dilation and decrease the ulcer recurrence rate. But again, a significant percentage of patients with ulcer-related gastric outlet obstruction do not have demonstrable *H. pylori* infection.

The most common operations for obstructing peptic ulcer disease are V+A and V+D. HSV and drainage is comparable to vagotomy and antrectomy in this setting. HSV and gastrojejunostomy is an appealing operation for obstruction, both because it can be done as a laparoscopically assisted procedure, and because it does not complicate future resection should this be needed. All gastric ulcers associated with obstruction should be adequately biopsied if not resected.

Intractability or Nonhealing

This should indeed be a rare indication for surgery performed today. Arguably, the patient referred for surgical evaluation because of intractable peptic ulcer disease should raise red flags for the surgeon. Acid secretion can be totally blocked and *H. pylori* eradicated with modern medication; therefore, the question remains: "Why does the patient have a persistent ulcer diathesis?" The surgeon should review the differential diagnosis of nonhealing ulcer prior to any consideration of operative treatment ([Table 25-14](#)).

Surgical treatment should be considered in patients with nonhealing or intractable peptic ulcer disease who have multiple recurrences, large ulcers (> 2 cm), complications (obstruction, perforation, or hemorrhage), or suspected gastric cancer. Surgery should be approached most cautiously in the thin or marginally nourished individual.

It is important that the surgeon not fall into the trap of performing a large, irreversible operation on these patients, based on the unproven theory that if all other methods have failed, a larger operation is required. Today's patients are different than those of 3 or 4

decades ago. One might argue that modern medical care has healed the minor ulcer, and that patients presenting with true intractability or nonhealing will be more difficult to treat and are likely to have chronic problems after a major ulcer operation. If surgery is necessary, less is often better. It is the practice of this author never to perform a gastrectomy as the initial elective operation for intractable duodenal ulcer in the thin or asthenic patient. Instead, the preferred operation for this group of patients is HSV. In patients with nonhealing gastric ulcer, wedge resection with HSV should be considered in thin or frail patients. Otherwise distal gastrectomy (to include the ulcer) is recommended. It is unnecessary to add a vagotomy in patients with type I gastric ulcer. Juxtaesophageal gastric ulcers (type IV) are pathophysiologically akin to type I gastric ulcers (i.e., associated with gastric acid hyposecretion), but are often difficult to resect as part of a distal gastrectomy. A variety of techniques have been used to treat these ulcers surgically, including the Csendes operation, the Pauchet gastrectomy, and the Kelling-Madlener procedure ([Fig. 25-51](#)).

FIG. 25-51. Operations for gastric ulcer. [*Reproduced with permission from Farrell TM, Hunter JG: Laparoscopic surgery of the stomach and duodenum, in Zuidema GD, Yeo CJ (eds): Shackelford's Surgery of the Alimentary Tract, 5th ed., Vol. II. Philadelphia: Saunders, 2002, p 202.*]

Zollinger-Ellison Syndrome

The Zollinger-Ellison syndrome (ZES) is caused by the uncontrolled secretion of abnormal amounts of gastrin by a pancreatic or duodenal neuroendocrine tumor (i.e., gastrinoma). Most cases (80%) are sporadic, but 20% are inherited. The inherited or familial form of gastrinoma is associated with multiple endocrine neoplasia type I (MEN I), which consists of parathyroid, pituitary, and pancreatic (or duodenal) tumors. Gastrinoma is the most common pancreatic tumor in patients with MEN I. Patients with MEN I usually have multiple gastrinoma tumors, and surgical cure is unusual. Sporadic gastrinomas are more often solitary and amenable to surgical cure. Currently, about 50% of gastrinomas are malignant, with lymph node, liver, or other distant metastases at presentation. Five-year survival in patients presenting with metastatic disease is approximately 40%.

The most common symptoms of ZES are epigastric pain, GERD, and diarrhea. The average age of presentation is 50 years, and over 90% of patients with gastrinoma have peptic ulcer. Most ulcers are in the typical location (proximal duodenum), but atypical ulcer location (distal duodenum, jejunum, or multiple ulcers) should prompt an evaluation for gastrinoma. Gastrinoma also should be considered in the differential diagnosis of recurrent or refractory peptic ulcer, secretory diarrhea, gastric rugal hypertrophy, esophagitis with stricture, bleeding or perforated ulcer, familial ulcer, and ulcer in the setting of hypercalcemia.

Hypergastrinemia in the presence of elevated BAO suggests gastrinoma. Despite this relatively simple guideline, most patients with ZES have been symptomatic for several years prior to diagnosis. In patients on antisecretory therapy, medication should be stopped for several days prior to checking the serum gastrin level, since acid suppression

may falsely elevate gastrin levels. Causes of hypergastrinemia can be divided into those associated with hyperacidity and those associated with hypoacidity ([Table 25-15](#)). The diagnosis of ZES is confirmed by the secretin stimulation test. An intravenous bolus of secretin (2 U/kg) is given and gastrin levels are checked before and after injection. An increase in serum gastrin of 200 pg/mL or greater suggests the presence of gastrinoma. Other provocative tests such as calcium stimulation or standard meal are usually unnecessary. Patients with gastrinoma should have serum calcium and parathyroid hormone levels determined to rule out MEN I.

Eighty percent of primary tumors are found in the gastrinoma triangle ([Fig. 25-52](#)) and many tumors are small (< 1 cm), making preoperative localization difficult. Transabdominal ultrasound is quite specific, but not very sensitive. CT will detect most lesions over 2 cm in size and magnetic resonance imaging (MRI) is comparable. Endoscopic ultrasound is more sensitive than these other noninvasive imaging tests, but it still misses many of the smaller lesions, and may confuse normal lymph nodes for gastrinomas. Currently, the imaging study of choice for gastrinoma is somatostatin receptor scintigraphy (SRS, the octreotide scan). When the pretest probability of gastrinoma is high, the sensitivity and specificity of this modality approach 100%. Gastrinoma cells contain type 2 somatostatin receptors which bind the indium-labeled somatostatin analogue (octreotide) with high affinity, making imaging with a gamma camera possible ([Fig. 25-53](#)). Currently, angiographic localization studies are infrequently performed for gastrinoma. Both diagnostic angiography and transhepatic selective venous sampling of the portal system have been supplanted by selective secretin infusion, which helps to localize the tumor as inside or outside the gastrinoma triangle. In this test, an arterial catheter is selectively placed in a named vessel supplying the pancreas (e.g., gastroduodenal or splenic), and a venous catheter is placed in a hepatic vein. Secretin is injected into the visceral artery and gastrin is sampled in the hepatic vein. A significant elevation in hepatic venous gastrin indicates that the tumor is supplied by the injected artery. Probably the most important means of locating gastrinomas is intraoperative exploration.

All patients with sporadic (nonfamilial) gastrinoma should be considered for surgical resection and possible cure. The lesions should be located in 90% of patients, and 60% are cured by extirpation of the gastrinoma(s). A thorough intraoperative exploration of the gastrinoma triangle and pancreas is essential, but other sites (i.e., liver, stomach, small bowel, mesentery, and pelvis) should be evaluated as part of a thorough intra-abdominal evaluation to find the primary tumor, which is usually solitary. The duodenum and pancreas should be extensively mobilized and intraoperative ultrasound should be used. Intraoperative EGD with transillumination should be considered. If the tumor cannot be located, generous longitudinal duodenotomy with inspection and palpation should be considered. Lymph nodes from the portal, peripancreatic, and celiac drainage basins should be sampled. Ablation or resection of hepatic metastases should be considered. The management of gastrinoma in patients with MEN I is controversial because the patients are rarely cured by operation, and the tumors tend to be small and multiple. If tumor can be imaged preoperatively, operation by an experienced gastrinoma surgeon is reasonable.

Acid hypersecretion in patients with gastrinoma can always be managed with high-dose proton pump inhibitors. Highly selective vagotomy may make management easier in some patients and should be considered in those with surgically untreatable or unresectable gastrinoma.

FIG. 25-52. Gastrinoma triangle. [*Reproduced with permission from Ritchie WP Jr.: Benign diseases of the stomach and duodenum, in Ritchie WP, Steele G, Dean RH (eds): General Surgery. Philadelphia: Lippincott, 1995, p 117.*]

FIG. 25-53. Positive octreotide scan in patient with gastrinoma (*arrow*).