DIAGNOSIS AND MANAGEMENT OF BENIGN GASTRIC AND DUODENAL DISEASE

Gentian Kristo, MD, and Thomas E. Clancy, MD*

The treatment of benign gastric and duodenal disease has changed in recent decades, reflecting improved medical management of peptic ulcer disease (PUD) and a decreased need for surgical intervention. Although surgical therapy was the only effective option for PUD in the past, elective surgery is increasingly rare in recent decades. Operative therapy for PUD is largely reserved for urgent management of complications such as hemorrhage, bleeding, or perforation or the management of obstruction from intractable disease. The shift away from acid-reducing operations to damage control procedures reflects the remarkable success of medical management.

In this chapter, we focus primarily on the management of benign gastroduodenal disease, with a focus on the workup and management of PUD and consideration of relevant surgical techniques. The management of duodenal diverticular disease is briefly addressed. Additional gastroduodenal disease and procedures are considered elsewhere in the publication, including bariatric surgery, transduodenal approaches to the bile duct, cystgastrostomy for intractable pancreatic pseudocysts, and duodenal diverticulization for trauma.

Peptic Ulcer Disease

Epidemiology

PUD has an overall point prevalence in the US population of 1.8%, with a lifetime incidence of 10%. The presence of Helicobacter pylori increases the incidence of ulcer to about 1% per year, a rate that is six- to 10-fold higher than for noninfected subjects.

Advances in the medical management of PUD, including the use of effective acid-suppressing medications (e.g., histamine receptor antagonists and proton pump inhibitors [PPIs]) and the treatment of H. pylori, have led to a decrease in the incidence of PUD, the rates of hospitalization, and mortality. Despite these trends, PUD remains a significant cause of morbidity and health care costs, with estimates of annual expenditures (excluding medication costs) of $5.65 billion in the United States. Although the need for elective surgical management of uncomplicated duodenal and gastric ulcers has steadily decreased in recent decades, the rate of emergent surgery for complicated PUD has remained unchanged, at 7% of hospitalized patients.

Pathophysiology

The relationship between gastroduodenal ulcers and gastric acid secretion has been recognized for decades and is expressed in the dictum “no acid, no ulcer.” A contemporary viewpoint suggests that PUD results from an imbalance between factors damaging the gastroduodenal mucosa and those protecting the mucosa [see Table 1].

The majority of gastric and duodenal ulcers are caused by three factors: H. pylori infection, nonsteroidal antiinflammatory drug (NSAID) use, and acid hypersecretory states (e.g., Zollinger-Ellison syndrome [ZES]). Most patients with gastroduodenal ulcers will be colonized with H. pylori, and recurrence of ulcers is common without treating the H. pylori infection. H. pylori, a gram-negative spiral organism, is present in about half the world population, particularly in developing countries, where up to 20% of healthy volunteers will demonstrate infection. Infection of gastric epithelium is followed by gastric inflammation, and the relationship between H. pylori and ulcer formation has been widely demonstrated. The presence of ulcers in just a small fraction of individuals with infection suggests the action of other etiologic factors causing ulceration.

NSAIDs cause injury via suppression of prostaglandin synthesis, which leads to impaired mucosal defense. NSAID use can result in a range of lesions, from superficial erosions to deeper ulcerations. Mucosal injury caused by NSAIDs is more common in the stomach than the duodenum. Cigarette smoking may impair ulcer healing and may increase the risk of H. pylori–related ulceration.

Presentation/clinical manifestations

Most patients with PUD present with mild pain, burning discomfort, tenderness in the epigastrium, or nausea. Classically, the pain from a duodenal ulcer occurs 2 to 3 hours after a meal, is relieved temporarily with food or antacids, and at times awakens patients at night between about 11 pm and 2 am, when the circadian stimulation of acid secretion

Table 1 Factors Damaging and Protecting the Gastroduodenal Mucosa

<table>
<thead>
<tr>
<th>Damaging factors</th>
<th>Protective factors</th>
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<tbody>
<tr>
<td>Gastric acid</td>
<td>Mucus</td>
</tr>
<tr>
<td>Pepsin</td>
<td>Bicarbonate</td>
</tr>
<tr>
<td>Bile salts</td>
<td>Endogenous prostaglandins</td>
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<tr>
<td>Helicobacter pylori infection</td>
<td>Growth factors</td>
</tr>
<tr>
<td>Nonsteroidal antiinflammatory drugs (NSAIDs)</td>
<td>Cell regeneration</td>
</tr>
<tr>
<td>Smoking</td>
<td>Mucosal blood flow</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
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<tr>
<td>Stress</td>
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is maximal. Gastric ulcer pain is often aggravated by meals. However, the classic symptoms described are neither sensitive nor specific and are present in only a small number of patients. Furthermore, the nature of presenting symptoms alone does not differentiate between benign ulcerations and gastric malignancy.

**DIAGNOSIS**

Given the low predictive value of the signs and symptoms of PUD, the diagnosis of uncomplicated peptic ulcers is difficult to make solely on a clinical basis.

The diagnosis of PUD is typically made by endoscopy and upper gastrointestinal (UGI) radiography. Endoscopy is the most accurate method of establishing the diagnosis of peptic ulcers, with a reported sensitivity of 92% and specificity of 100%. In addition to identifying the ulcer and its features, location, and size, endoscopy provides an opportunity for biopsies to test for *H. pylori* and exclude malignancy and for therapeutic interventions for bleeding ulcers. Duodenal ulcers are most often benign and do not require routine biopsy. Multiple biopsies are indicated for all gastric ulcers as even lesions with a benign appearance harbor malignancy in 5 to 11% of cases.

Given the advances in the use of endoscopy in recent decades, the role of radiographic UGI series in the diagnosis of PUD has decreased, leading to a parallel decline in technical expertise. However, it can be useful when endoscopy is unavailable or in patients not eligible to undergo endoscopy. Diagnosis of peptic ulcer on UGI series requires demonstration of barium within an ulcer niche, which is generally round or oval and may be surrounded by a smooth mound of edema. Secondary changes include folds radiating to the crater and regional deformities secondary to spasm, edema, and scarring. The accuracy of the UGI radiography varies with the technique being used; single-contrast techniques (using liquid barium only) may miss as many as 50% of duodenal ulcers, whereas double-contrast studies (using air and liquid barium) create a more detailed view of the stomach lining and can detect 80 to 90% of ulcers. When radiographic features suggestive of cancer are present, endoscopy with biopsies is warranted.

Routine laboratory tests are of limited value in evaluating patients with uncomplicated PUD. When ZES is suspected, a fasting serum gastrin level may be useful.

However, laboratory tests do play an important role in the diagnosis of *H. pylori* infection. The serologic determination of anti-*H. pylori* IgG antibodies in peripheral blood has high sensitivity (90 to 100%) but variable specificity (76 to 96%). Serology is preferred for initial diagnosis when endoscopy is not required. Urea breath testing has a sensitivity of 88 to 95% and a specificity of 95 to 100% for establishing the presence of *H. pylori* infection and is the test of choice for confirming successful eradication of the organism 4 to 6 weeks after the cessation of antibiotic treatment.

Although endoscopy is not indicated solely for the purpose of establishing the presence of *H. pylori*, gastric biopsy specimens can be used in the diagnosis of *H. pylori* by rapid urease test, histology, and bacterial culture. Rapid urease testing of the biopsy specimen is the preferred method when histopathology is not required because it is fast (results within hours), is less expensive, and has a sensitivity of 90 to 95% and a specificity of 95 to 100%. Histologic examination of tissue samples remains the gold standard for determining the presence of *H. pylori* and can also provide additional information regarding the presence of gastritis, intestinal metaplasia, or mucosa-associated lymphoid tissue.

Routine culture of the gastric biopsy specimens for *H. pylori* is not recommended as it is relatively expensive, and diagnosis requires more time (3 to 5 days). However, the incidence of antimicrobial resistance is high in patients with refractory peptic ulcers, and they may benefit from culture and antibiotic sensitivity testing.

**TREATMENT**

The medical management of PUD includes treatment with drugs that reduce the production of gastric acid (antacids, histamine receptor antagonists, and most notably PPIs), create a protective coating over the ulcer crater to prevent further peptic damage (sucralfate), increase mucosal blood flow and the secretion of bicarbonate and mucus (prostaglandin analogues such as misoprostol used in NSAID-induced ulcers), and treat *H. pylori* infection. In addition to drug therapy, general measures such as cessation of smoking and alcohol and discontinuation of NSAID use are recommended.

In patients infected with *H. pylori*, the current pharmacologic therapy consists mainly of a combination of PPIs and antibiotics against *H. pylori* [see Table 2]. Eradication of *H. pylori* infection has resulted in very high ulcer healing rates (90%) and decreased recurrence rates of approximately 2%, particularly in patients with a duodenal ulcer. Furthermore, it also has a prophylactic effect on recurrent ulcer bleeding.
Triple therapy with two antibiotics, amoxicillin and clarithromycin, and a PPI for a week is considered the \textit{H. pylori} treatment of choice. However, given the recent increase in resistance to clarithromycin, quadruple therapy with a combination of a PPI, bismuth, metronidazole, and tetracycline for 10 to 14 days is recommended in areas with a clarithromycin resistance over 15 to 20\%.

Maintenance therapy with a PPI after \textit{H. pylori} eradication can significantly reduce ulcer recurrence and complications. Standard-dose maintenance PPI treatment should be prescribed for 4 weeks in patients with duodenal ulcers and for 8 weeks in patients with gastric ulcers.

In patients with PUD without evidence of \textit{H. pylori} infection or a history of NSAID use, further investigations are necessary to determine the etiology of the ulcer disease before establishing specific therapy.

**Management of Complicated Peptic Ulcer Disease**

Although the vast majority of ulcer patients are successfully managed medically, the role of surgical therapy for PUD is now largely reserved for the management of the acute complications of bleeding and perforation as well as for more chronic and refractory issues, such as gastric outlet obstruction and intractable PUD. The recognition that medical management successfully prevents ulcer recurrence in most patients has caused surgical management of complicated ulcer disease to evolve into a more minimalist strategy that favors damage control surgery for complications and only infrequently resorts to acid-reducing operations.

A multidisciplinary approach is crucial in the treatment of patients with complicated PUD. Early collaboration between the gastroenterologist, the intensivist, and the surgeon provides a great expertise for appropriate resuscitation, establishment of goals and limits for initial nonoperative therapy, and timely preoperative preparation.

**Hemorrhage**

UGI bleeding secondary to PUD has an overall incidence of about 60 per 100,000 individuals, increasingly related to NSAID use. The mortality associated with bleeding peptic ulcers remains high at 5 to 10\%. Bleeding typically presents with hematemesis or melena, although hematochezia can

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**Table 2** Commonly Used Regimens to Eradicate \textit{Helicobacter pylori}

<table>
<thead>
<tr>
<th>Regimen</th>
<th>Proton pump inhibitor b.i.d. for 7–14 days</th>
<th>Amoxicillin, 1 g b.i.d. for 7–14 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>\textbf{Triple therapy}</td>
<td>Proton pump inhibitor b.i.d. for 7–14 days</td>
<td>Amoxicillin, 1 g b.i.d. for 7–14 days</td>
</tr>
<tr>
<td></td>
<td>Clarithromycin, 500 mg b.i.d. or t.i.d. for 7–14 days</td>
<td>Clarithromycin, 500 mg b.i.d. for 7–14 days</td>
</tr>
<tr>
<td>\textbf{Quadruple therapy}</td>
<td>Proton pump inhibitor b.i.d. (or ranitidine, 150 mg orally b.i.d.) for 10–14 days</td>
<td>Bismuth subsalicylate, 525 mg q.i.d. for 10–14 days</td>
</tr>
<tr>
<td></td>
<td>Metronidazole, 500 mg orally q.i.d. for 10–14 days</td>
<td>Metronidazole, 500 mg b.i.d. for 7–14 days</td>
</tr>
<tr>
<td>Levofloxacin-based triple therapy</td>
<td>Proton pump inhibitor b.i.d. for 10 days</td>
<td>Levofloxacin, 250–500 mg b.i.d. for 10 days</td>
</tr>
<tr>
<td></td>
<td>Amoxicillin, 1 g b.i.d. for 10 days</td>
<td>Amoxicillin, 1 g b.i.d. for the first 5 days followed by</td>
</tr>
<tr>
<td>Sequential therapy</td>
<td>Clarithromycin, 500 mg b.i.d. for the next 5 days</td>
<td>Amoxicillin, 1 g b.i.d. for the first 5 days followed by</td>
</tr>
<tr>
<td></td>
<td>Timidazole, 500 mg b.i.d. for the next 5 days</td>
<td>Timidazole, 500 mg b.i.d. for the next 5 days</td>
</tr>
<tr>
<td>Levofloxacin-based sequential therapy</td>
<td>Proton pump inhibitor b.i.d. for 10 days</td>
<td>Amoxicillin, 1 g b.i.d. for the first 5 days followed by</td>
</tr>
<tr>
<td></td>
<td>Levofloxacin, 250 mg b.i.d. and Timidazole, 500 mg b.i.d. for the next 5 days</td>
<td>Levofloxacin, 250 mg b.i.d. and Timidazole, 500 mg b.i.d. for the next 5 days</td>
</tr>
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</table>
also occur in cases of massive bleeding. The initial management of a patient with UGI bleeding starts with restoring hemodynamic stability and administration of an intravenous PPI. Early treatment with PPI promotes rapid healing, decreases the need for endoscopic treatment, and reduces rebleeding rates.20

After initial stabilization, patients should undergo endoscopy to identify the source of bleeding. In many cases, endoscopic therapy using contact thermal devices, hemoclips, and/or combination therapy with epinephrine injection will control bleeding, rendering surgical management unnecessary. Definitive surgical management is indicated if bleeding leads to hemodynamic instability, an extensive transfusion requirement (i.e., more than 6 units in 24 hours), or rebleeding after initial endoscopic hemostasis or when endoscopic therapy is unavailable.

Objective criteria for surgery must be determined on a patient-by-patient basis. Precise preoperative localization of the bleeding source is essential; a bleeding posterior duodenal ulcer, for example, cannot be managed in the same way as hemorrhage from diffuse severe gastritis. Endoscopy should therefore be performed whenever possible.

Alternatively, if brisk bleeding prevents clear intraluminal visualization of a bleeding site, angiographic localization may be attempted. Furthermore, depending on local resources and expertise, angiographic transarterial embolization can be considered following failed endoscopic hemostasis, particularly in high-risk surgical patients, as it reduces the recurrent bleeding and the need for surgery without increasing the overall mortality and leads to fewer complications.21

Perforation

Perforated peptic ulcer has an incidence of 1.5 to 7.8 cases per 100,000 population per year and a high mortality of approximately 15%. Perforations are most likely in elderly patients on chronic NSAID therapy. The perforation site usually involves the anterior wall of the duodenum (60%), antrum (20%), or lesser-curvature gastric ulcers (20%).22

Perforation of peptic ulcer remains largely a clinical diagnosis. Initial symptoms include severe abdominal pain, worse in the epigastrium, often accompanied by nausea and vomiting. Patients typically present in distress with an acute abdomen. A chest x-ray that demonstrates free intraperitoneal air [see Figure 3] is all that is needed before the patient is taken to the operating room; the decision to proceed to operation should not be delayed by waiting for further images (e.g., computed tomographic [CT] scans). Surgical delay is a critical determinant of survival in perforated peptic ulcer; every hour of delay from presentation to surgery is associated with an adjusted 2.4% decreased probability of survival compared with the previous hour.23

When there is no free air on the plain film and diagnostic uncertainty persists, an UGI series using water-soluble contrast material may be helpful. Perforation is a contraindication for endoscopy because air insufflation may disrupt a sealed perforation and increase spillage of gastrointestinal contents.

In well-selected cases, when there is evidence of contained perforation without free air in a hemodynamically normal patient without peritonitis, nonoperative management with nasogastric drainage, intravenous PPIs, antibiotics, and serial abdominal examinations can be attempted. However, the decision to manage patients without surgery should undergo frequent reevaluations as failure of nonsurgical management is an indication for surgery.

Gastric Outlet Obstruction

Although, historically, PUD was the most common cause of gastric outlet obstruction, with an incidence of up to 90%,24 treatment for H. pylori and the introduction of PPIs have led to a significant decrease in the incidence of ulcer-induced gastric outlet obstruction. Benign obstruction secondary to PUD requires surgical treatment in about 2,000 patients per year in the United States.25 PUD can lead to gastric outlet obstruction by causing acute inflammatory swelling of the pyloric channel or chronic scarring associated with fibrosis. Patients typically have a long history of peptic ulcer pain and present with early satiety, bloating, nausea, vomiting, and epigastric fullness. Patients with longstanding obstruction commonly present with progressive weight loss and malnutrition.

Gastric outlet obstruction can be confirmed with UGI barium contrast radiography, which shows a markedly dilated stomach and delayed gastric emptying. After decompression of the stomach for 12 to 24 hours, endoscopy is performed to visualize the gastric outlet and perform...
biopsies to rule out cancer. Nonoperative management of peptic gastric outlet obstruction with repeated endoscopic dilations and H. pylori eradication has been shown to have good long-term results and in 70 to 80% of cases can avoid subsequent surgical therapy.\textsuperscript{26,27} Patients with refractory obstruction require a surgical drainage procedure and treatment of the predisposing ulcer with an acid-reducing procedure. Typically, this will require a resection procedure such as antrectomy with reconstruction and vagotomy.

\textit{Intractable Peptic Ulcer Disease}

Occasionally, patients present for surgery with refractory peptic ulcers. If a gastric ulcer has not healed after 12 weeks of optimal medical therapy, resection is indicated to rule out an occult gastric malignancy. In such cases, the preoperative workup should include endoscopic biopsies of the ulcer base and the surrounding gastric mucosa so that a preoperative diagnosis of malignancy can be made if possible. In view of the concern about a possible gastric malignancy, it is reasonable to obtain a preoperative chest x-ray and a CT scan of the abdomen so as to detect possible nodal or distant metastases.

The first step in the management of refractory duodenal ulcerations is a complete workup for the possible causes, such as gastrinoma, Crohn disease, lymphoma, and carcinoma. Once diseases with identifiable etiologies have been ruled out, maximal medical therapy should be instituted.

Highly selective vagotomy is the acid-reducing procedure of choice in patients with intractable PUD.

\textbf{Surgical Procedures for Peptic Ulcer Disease}

\textbf{Bleeding Ulcers}

For patients with bleeding duodenal ulcers, especially those with a first ulcer who have not been on PPIs or other acid suppression therapy, ligation of the bleeding vessel and closure of the duodenum/pylorus with pyloroplasty may be sufficient. Acid-reducing procedures such as truncal vagotomy with pyloroplasty are rarely employed as primary therapy for PUD. However, such acid-reducing procedures do play a role in the emergency management of bleeding duodenal ulcers after oversewing the bleeding vessel, especially in patients already on PPIs or patients at high risk for noncompliance with medical therapy.

\textit{Oversewing of Bleeding Ulcer, Pyloroplasty, and Vagotomy}

\textbf{Operative technique  Step 1: ligation of bleeding vessel}

After a longitudinal incision across the pylorus, the ulcer bed is oversewn with figure-of-eight sutures placed at the cephalad and caudad portions of the ulcer to occlude the gastroduodenal artery. An additional U stitch is placed to control small transverse pancreatic branches that may cause late bleeding. Care should be taken to avoid the underlying common bile duct.

\textbf{Step 2: pyloroplasty}

The longitudinal incision is closed transversely in two layers (Heineke-Mikulicz pyloroplasty) [see Figure 5]. If substantial duodenal scarring is present, a tension-free Heineke-Mikulicz pyloroplasty may not be possible, and a side-to-side anastomosis between the distal stomach and the proximal duodenotomy may be preferable (Finney pyloroplasty) [see Figure 6].

\textbf{Step 3: vagotomy}

Downward traction on the greater curvature of the stomach is essential to apply gentle tension to the esophagogastric junction and the proximal vagi. The left (anterior) vagus is best identified by applying traction to the right and posteriorly so that the nerve can be traced into the posterior mediastinum. The main nerve trunk is clipped proximally and distally, and a 2 cm long segment is excised. The right (posterior) vagus is exposed by applying traction...
Finney pyloroplasty. If a tension-free Heineke-Mikulicz pyloroplasty is not feasible, a Finney pyloroplasty may be performed instead. (a) The distal stomach and the proximal duodenum are aligned with stay sutures; meticulous lysis of surrounding adhesions is essential. An inverted U-shaped incision is made. (b) The pyloroplasty is created in two layers. The posterior portion of the outer layer, consisting of seromuscular Lembert sutures, is placed first, followed by an inner layer constructed with a continuous full-thickness absorbable suture. (c) The anterior portion of the inner layer is closed with a continuous full-thickness suture. Some surgeons prefer a Connell suture at this site. (d) The anterior portion of the outer layer, consisting of silk Lembert sutures, is placed.

Exploration for Bleeding Gastric Ulcers

Gastric ulcer bleeding is often self-limited and typically managed endoscopically with coagulation, injection, or clipping bleeding vessels. The presence of active pulsatile bleeding or a visible vessel is predictive of a higher rate of rebleeding. Emergency operations for bleeding gastric ulcers should be tailored to the type of ulcer identified. For patients with a bleeding gastric ulcer, the preferred approach is partial gastrectomy because of the risk of gastric malignancy. For patients who are at high risk because of advanced age, comorbid disease, or intraoperative instability, ulcer excision combined with truncal vagotomy and pyloroplasty...
is an option. Ulcer excision alone is associated with rebleeding in approximately 20% of patients.

Operative technique  Step 1: anterior gastrotomy The stomach is opened through a longitudinal anterior gastro-tomy in the distal stomach or antrum and explored for a bleeding source.

Step 2: ulcer resection Resection of a type I (lesser curvature) or II/III (prepyloric ± duodenal) bleeding ulcer will require antrectomy or distal gastrectomy. Type IV ulcers (juxtaesophageal) require a more technically difficult resection, including subtotal gastrectomy with upward extension to include the ulcer followed by Roux-en-Y reconstruction (Csendes procedure). Rarely, for severe bleeding, devascularization of the stomach with ligation of the left gastric artery can be performed while leaving the short gastric arcade intact. This can be accompanied by oversewing the ulcer. Vagotomy is usually not added in the absence of a failure of medical management.

Step 3: reconstruction Reconstruction via a gastroduodenal anastomosis (Billroth I) [see Figure 8], a gastrojejunal anastomosis (Billroth II) [see Figure 9], or a Roux-en-Y gastrojejunostomy must be tailored to the resection performed.

EXPLORATION FOR PERFORATED ULCERS

Omental Patch for Duodenal Perforation (Graham Patch)

Perforated duodenal ulcers are generally managed with an omental patch. With adequate acid suppression and adequate treatment of H. pylori, symptoms of duodenal ulcer are unlikely to recur and vagotomy is usually not required. The 1-year recurrence rate after omental patching is substantially lower when both therapies are employed than when only a PPI is given (5% versus 38%).

Operative technique  Step 1: exposure The ulcer is identified and débrided if possible.

Step 2: closure Primary closure is not attempted if the duodenal wall is overly edematous to avoid excess tension. The defect is plugged with a well-vascularized omental pedicle [see Figure 10].

Step 3: acid-reducing procedure A vagotomy is avoided if there is no history of PUD, if the patient is hemodynamically unstable, or if there is gross abdominal contamination to avoid dividing the peritoneum over the esophagus and exposing the mediastinum.

Antrectomy

The primary indication for gastric resection in the setting of PUD is chronic obstruction caused by scarring, typically from a pyloric channel ulcer. In addition, antrectomy may be required and may be the elective operation of choice for intractable type I, II, and III gastric ulcers, as well as a primary emergency surgical option for perforated or bleeding gastric ulcers. A primary Billroth I gastroduodenostomy [see Figure 8] has been the preferred procedure, but surrounding scar tissue may limit the mobility of the duodenum, in which case, a Billroth II gastrojejunostomy [see Figure 9] may be required for a tension-free anastomosis. Antrectomy is typically combined with truncal vagotomy.

Operative technique  Step 1: exposure The proximal border of the antrum on the greater curvature extends to a point between the pylorus and the fundus; on the lesser curvature, it extends to a point just above the incisura. Distally, the dissection is carried past the pylorus.

Step 2: division of stomach A gastrointestinal anastomosis (GIA) stapler is used to divide the stomach at the estimated borders of the antrum. The right gastroepiploic artery and vein are divided just distal to the pylorus. The duodenum is then divided distal to the pylorus with a transverse anastomosis (TA) stapler.

Step 3: reconstruction When feasible, a Billroth I reconstruction [see Figure 8] may be performed to maintain physiologic antegrade flow and avoids the complications associated with a Billroth II reconstruction [see Figure 9], such as afferent and efferent loop syndromes and duodenal stump leaks. A generous Kocher maneuver is necessary to minimize tension on the anastomosis [see Figure 11].

If a primary gastroduodenostomy is not possible, a Billroth II reconstruction [see Figure 9] is indicated. If the duodenum is not scarred or inflamed, simple staple closure will suffice; if closure proves difficult, a lateral duodenostomy tube may help decompress the stump. Alternatively, if the duodenum cannot be closed without significant tension, closure may be accomplished around a red rubber or mushroom catheter. In addition, omental patch reinforcement of the duodenal stump may be desirable. The segment used for the anastomosis should be as short as possible while still being able to reach the stomach without tension;
approximately 20 cm of proximal jejunum should be sufficient to serve as the afferent limb. Passing the jejunum through a retrocolic window places less tension on the mesentery than an antecolic approach does.

The gastrojejunostomy may be handsewn in two layers either to the posterior wall of the stomach or to the inferior portion of the excised staple line [see Figure 9]. Alternatively, the gastrojejunostomy may be created by means of stapling. Some authors recommend the use of a Braun enteroenterostomy between the efferent and afferent limbs to reduce bile reflux and decompress the duodenal stump [see Figure 12a]. Staple closure of the afferent limb above the enteroenterostomy may also be performed to limit bile reflux into the stomach; this measure creates a configuration referred to as an uncut Roux-en-Y [see Figure 12b]. Enteroenterostomy at this site may also be performed on an emergency basis to treat afferent limb syndrome. Staple closure of the afferent limb may discourage bile reflux, with the effect of limiting bile reflux, gastritis, and esophagitis. 30

Complications Following antrectomy, a number of potential complications must be anticipated and recognized.
Leakage from the duodenal stump necessitates prompt reoperation, washout, and drainage. The diagnosis of duodenal stump leakage is confirmed by aspirating bilious fluid from a right upper quadrant fluid collection or by performing a technetium 99m-labeled hepatoiminodiacetic acid scan. Duodenal leaks can rarely be closed primarily. Duodenostomy may be indicated to further decompress the afferent limb and prevent continuous leakage. The goals are to create a controlled fistula to the skin and to prevent the accumulation of biliary fluid in the abdomen.

Delayed gastric emptying after gastrojejunostomy may occur and is generally managed conservatively. On rare occasions, reoperation is required for delayed anastomotic function.

Afferent limb obstruction may occur as a consequence of adhesions, internal herniation, volvulus, or a kink at the angle formed with the gastric remnant. Obstruction to outflow of the afferent limb creates a closed-loop obstruction, with persistent secretion of bile and pancreatic fluids into the loop. Correction of the obstruction may necessitate conversion to a Roux-en-Y reconstruction, shortening of the afferent limb, or a side-to-side enterenterostomy with the efferent limb [see Figure 12].

Alkaline reflux gastritis is one of the most common long-term complications of gastrectomy, developing in 5 and 15% of patients after gastric surgery. This complication is most
frequently associated with Billroth II reconstructions. Although reflux is common, symptoms (e.g., epigastric pain, nausea, and bilious emesis) are relatively rare. Medical management is generally ineffective. If surgery is required, conversion of the Billroth II reconstruction to a Roux-en-Y reconstruction is indicated. In those patients who have a Roux-en-Y rather than a Billroth II reconstruction, the preferred treatment is to divert alkaline contents to a location 45 to 60 cm beyond the gastric remnant.

**Highly Selective Vagotomy**

Acid-reducing operations such as vagotomy with antrectomy or vagotomy with pyloroplasty will provide relief from duodenal ulcers but may also be associated with significant complications. Highly selective vagotomy (HSV), also referred to as parietal cell vagotomy, avoids vagal denervation of viscera other than the parietal cell mass while keeping the pylorus mechanically and functionally intact. HSV was found to reduce postvagotomy diarrhea and dumping dramatically; however, it was also found to be associated with a much higher rate of recurrent ulceration (greater than 10% at 5 years). Today, in an era characterized by greatly improved medical management of PUD, most gastrointestinal surgeons rarely perform HSV, if at all.

In the current context, this procedure could be used to treat intractable ulcer disease in young patients (after ZES and other hypersecretory conditions are ruled out) or in patients noncompliant with PPI therapy. If the surgeon is not experienced with HSV, then in the emergency situation, a truncal vagotomy and pyloroplasty are adequate treatment.

**Operative technique**

**Step 1: dissection of anterior and posterior nerve branches to lesser curvature** The distal branches of the nerve of Latarjet are defined, with care taken to preserve the so-called crow’s foot, which is typically located near the incisura angularis, approximately 6 to 7 cm proximal to the pylorus. Gentle downward traction is applied to the stomach, the left gastric vascular arcade is identified, and all tissue between this arcade and the lesser curvature is divided and ligated. The dissection should proceed upward as far as the esophagogastric junction. The posterior branches are approached either by rotating the stomach or by proceeding directly through the lesser omentum [see Figure 13a].

**Step 2: dissection of esophagogastric junction** The distal esophagus is cleared of all nerve fibers for a distance of approximately 5 cm above the esophagogastric junction. The posterior esophagogastric junction is exposed by means of gentle traction and slight rotation of the distal esophagus. Exposure is facilitated by downward traction provided by a Penrose drain placed around the esophagogastric junction. The dissection must stay close to the lesser curvature and the esophagus, avoiding the tissues to the right of the esophagus, where the main vagal trunks lie.

The left side of the distal esophagus must be manually stripped so that the so-called criminal nerve of Grassi [see Figure 13b] can be identified and ligated.

**Gastrinoma (Zollinger-Ellison Syndrome)**

ZES has been estimated to cause 0.1 to 1% of PUD. It consists of hypergastrinemia, gastric acid hypersecretion, severe PUD, and non–β-islet cell tumors of the pancreas. Classically, ZES is associated with multiple duodenal ulcers, ulcers in unusual locations (beyond the first portion of the duodenum; jejunum), or ulcers that fail to respond to standard therapy.

Patients with ulcers refractory to treatment with PPIs, those with ulcers in the distal duodenum or jejunum, and those with recurrent ulcers despite treatment should be evaluated for ZES. In addition, patients with PUD who are not infected with *H. pylori* and who do not use NSAIDs should also be evaluated for ZES. The biochemical diagnosis
Figure 13  Highly selective vagotomy. (a) Anterior and posterior branches of the nerve of Latarjet to the lesser curvature are ligated and divided. The distal branches, comprising the so-called crow’s foot, are left intact. The posterior branches may be approached via the lesser omentum. (b) Note the criminal nerve of Grassi.
is established by findings of elevated fasting serum gastrin levels, reduced gastric pH, or increased basal acid output.

Patients should have PPI held for 1 week before measuring fasting gastrin as acid suppression with PPI will artificially elevate the gastrin. H₂ blockers should be held for 2 days. All patients with ZES will have a fasting gastrin greater than 100 pg/mL.

Demonstration of acid hypersecretion (in the presence of elevated gastrin) is also critical to make the diagnosis of ZES as achlorhydria could also lead to hypergastrinemia. Basal acid output greater than 15 mEq/hr is diagnostic, as is pH less than 2.3.

Elevations of serum gastrin levels of greater than 200 pg/mL above baseline after administration of secretin can confirm ZES when the diagnosis is equivocal. Preoperatively, ZES patients are treated with higher-than-normal doses of PPI, and imaging studies are undertaken in an attempt to localize the tumor. Somatostatin receptor scintigraphy is the study of choice, with a sensitivity of 85%. Treatment of localized gastrinoma is surgical resection.

**Laparoscopic Treatment of Peptic Ulcer Disease**

With the advent of minimal access techniques in recent decades, laparoscopy has become an accepted approach in the treatment of peptic ulcers. Numerous reports of laparoscopic truncal vagotomy and HSV have been published over the past two decades. The laparoscopic versions of these procedures proceed in much the same way as the traditional open versions. In a laparoscopic HSV, the ultrasonic shears are frequently used to divide the anterior and posterior vagal branches to the lesser curvature from the crow’s foot to the esophagogastric junction. Mixed procedures that combine posterior truncal vagotomy with more selective anterior gastric denervation are common. In the Taylor procedure [see Figure 14], an anterior seromyotomy is performed from the angle of His to the crow’s foot, and the seromuscular layers are subsequently closed primarily. A variant of the laparoscopic Taylor procedure includes an anterior linear gastrectomy, in which a linear strip of the stomach wall is removed parallel to the lesser curvature with an

![Figure 14](https://www.scientificamerican.com/article/0614-gastro-diagnosis-and-management-of-benign-gastric-and-duodenal-disease/)
endoscopic GIA stapler. This procedure seems to be functionally equivalent to HSV. The majority of the studies of laparoscopic vagotomy, however, have been individual case series that do not compare the laparoscopic approach with a traditional open approach. Undoubtedly, the scarce comparative data reflect the declining indications for vagotomy for intractable PUD. Thus, although laparoscopic HSV has been well described, it is by no means clear that it should be recommended over open HSV for those rare patients in whom this procedure is indicated.

With increasing laparoscopic expertise and advancement in equipment, laparoscopic repair of perforated duodenal ulcers has become more common. In experienced hands, the laparoscopic approach is considered as safe and effective as open Graham patch repair. The longer operating time associated with laparoscopic repair has no effects on the outcomes.

Selecting patients who are good candidates for laparoscopy is very important. The laparoscopic repair is contraindicated in patients with prolonged perforation for more than 24 hours, shock on admission, and significant systemic illness (defined as American Society of Anesthesiologists grades III–IV).

Laparoscopic procedures for perforation include either suture closure of the perforation followed by omental patch or omental patch alone and should be attempted only by surgeons with advanced laparoscopic skills. Intracorporeal knotting is preferred because extracorporeal suturing may cut through the friable edge of the duodenal perforation.

The threshold for conversion to an open procedure should be low if the ulcers are particularly large or prove difficult to localize.

Laparoscopic duodenotomy and oversewing of the gastroduodenal and transverse pancreatic arteries for the bleeding duodenal ulcer have been shown to be technically feasible in select patients with slow-bleeding duodenal ulcers. However, the role of laparoscopic surgery for bleeding peptic ulcers is not yet established.

**Duodenal Diverticula**

Incidental duodenal diverticula are common. They are false diverticula, including only mucosa and submucosa. Most are located in the medial wall of the second portion of the duodenum, within 2 cm of the ampulla of Vater. Complications include ulceration and bleeding, compression of the CBD with cholangitis or pancreatitis, and, in cases of perforation, abscess formation with peritonitis. CT scanning is useful for differentiating this condition from choledocholithiasis or pancreatitis.

Incidentally found duodenal diverticula should be left alone. In cases of vague abdominal complaints, duodenal diverticula are rarely the cause and are usually not resected in the absence of complications. When the duodenal diverticulum produces biliary obstruction, diverticulectomy is the appropriate procedure. In high-risk patients, endoscopic sphincterotomy or stenting can be performed. Diverticular bleeding may require endoscopic hemostatic therapies, angiographic embolization, or lateral duodenotomy followed by direct suturing of the bleeding point. Diverticulitis is first managed with antibiotics and bowel rest if no perforation has occurred. Perforation with abscess will require an operative procedure.

**Duodenal Diverticulectomy**

**Operative Technique**

When indicated, the main surgical options are simple diverticulectomy with drainage and transduodenal diverticulectomy (as described by Iida). If the duodenum is free of inflammation, the transduodenal approach is preferred because it minimizes the need for dissection of the diverticulum from the surrounding pancreas. However, if the diverticulum does not involve the pancreas, simple excision flush with the duodenal wall, followed by closure in two layers, may be sufficient. The ensuing technical description focuses on transduodenal diverticulectomy.

**Step 1: exposure and duodenotomy** After a generous Kocher maneuver, the duodenum is opened by making a 4 cm longitudinal incision along the antimesenteric border, and the ampulla is either visualized or palpated. To identify the ampulla, it may be necessary to place a catheter into the CBD via a separate choledochotomy [see Figure 15a].

**Step 2: diverticulectomy** The orifice of the diverticulum is identified, and the mucosa is inverted into the lumen of the duodenum. The neck of the diverticulum is transected 2 mm from the junction with the duodenal wall. The diverticular opening is then closed with interrupted seromuscular Lembert sutures of 3-0 silk and interrupted mucosal sutures of 4-0 polyglaclin [see Figure 15b]. Inadvertent closure of the CBD may be prevented by inserting a catheter into the duct. The duodenum is closed in two layers, also with an inner layer of 4-0 polyglaclin and an outer layer of seromuscular Lembert sutures of 3-0 silk. Closed-suction drainage adjacent to the duodenum is indicated.

If the duodenum is markedly inflamed, suture line breakdown is likely, eventually leading to a duodenal fistula. The area can be isolated by means of pyloric exclusion or antrectomy with Billroth II reconstruction. In addition, bile flow can be diverted by performing a choledochojejunostomy to a Roux-en-Y intestinal limb to prevent combined leakage of pancreatic fluid and bile.

Diverticula arising from the third or fourth portion of the duodenum may be excised either primarily or via a transduodenal approach. Inverting the diverticulum without excising it is not recommended, because it may lead to duodenal obstruction.

**Special case: perforated duodenal diverticulum** In the setting of acute inflammation, duodenotomy is avoided. Instead, the abscess is evacuated, and the diverticulum is excised along with just enough of the adjacent duodenal wall to ensure that only healthy tissue is left. If the resulting duodenal defect is large, either sleeve resection of the duodenum or drainage of the open duodenal defect into a Roux-en-Y jejunal limb may be required.
Duodenal diverticulectomy. (a) The duodenum is opened by making a 4 cm longitudinal incision along the antimesenteric border. The orifice of the diverticulum is identified, and the mucosa is inverted into the lumen of the duodenum. To identify the ampulla, a catheter is placed into the common bile duct via a separate choledochotomy. (b) The neck of the diverticulum is transected 2 mm from the junction with the duodenal wall. The diverticular opening is then closed with interrupted seromuscular Lembert sutures of 3-0 silk and interrupted mucosal sutures of 4-0 polyglactin.

References


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