Chapter 10
Antrectomy

J. Spencer Liles  John D. Christein

DEFINITION

- By strict definition, antrectomy refers to removal of the gastrin-secreting portion of the stomach and when combined with a vagotomy results in an 85% reduction in gastric acid secretion. In the 1960s and 1970s, antrectomy with or without vagotomy was routinely performed for treatment of benign gastric and duodenal ulcers but, due to pharmacologic developments in reducing acid secretion and elucidation of the role of Helicobacter pylori in ulcer development, is now rarely performed for ulcer disease.

- Today, the term antrectomy is loosely applied to any distal gastric resection and is indicated in recurrent or persistent gastric ulcers to rule out malignancy, complicated peptic ulcer disease (i.e., obstruction, hemorrhage, perforation), and for resection of certain neoplasms of the antrum and pyloric channel (Table 1).

- When an antrectomy is performed for complicated peptic ulcer disease, a vagotomy may be included to reduce the chance of anastomotic ulcer formation in patients who are not candidates for H. pylori treatment and lifelong proton pump inhibitor therapy due to unreliability, noncompliance, or medication side effects.

- Antrectomy is named by the type of gastrointestinal (GI) anastomosis performed.
  - Billroth I procedure—antrectomy and gastroduodenostomy
  - Billroth II procedure—antrectomy and gastrojejunostomy
  - A modification of the Billroth II procedure that involves a gastrojejunostomy via a Roux limb and is known as a Roux-en-Y gastrojejunostomy

DIFFERENTIAL DIAGNOSIS

- Complicated peptic ulcer disease and distal gastric neoplasms, both benign and malignant, account for the vast majority of the antral resections performed today. These diagnoses will be discussed separately.

- Peptic ulcer disease
  - Peptic ulcer disease refers to irritation of GI mucosa from gastric acid due to either increased acid presence or weakness in the mucosal protection and typically presents with epigastric pain. Peptic ulcers can occur anywhere in the GI tract, but duodenal and gastric ulcers are most common. Duodenal ulcers typically arise within 2 cm of the pylorus, are highly associated with H. pylori infection (>90%), and frequently resolve with appropriate H. pylori therapy. Gastric ulcers are less likely to be associated with H. pylori infection and are classified into five types based on their location and association with acid secretion (Table 2).

- The differential diagnosis of epigastric pain similar to that found in complicated peptic ulcer disease is chronic cholecystitis, acute pancreatitis, chronic pancreatitis, functional indigestion or dyspepsia, gastritis, and reflux esophagitis. Complicated peptic ulcer disease can also present with upper GI hemorrhage, and a differential should include esophagitis (reflux and infectious); gastroesophageal varices, arteriovenous malformations; Mallory-Weiss tear; stress gastritis; and neoplasm of the esophagus, stomach, duodenum, pancreas, and biliary tree.

- Lastly, pyloric obstruction due to chronic inflammation and scarring will cause nausea, emesis, and early satiety. The differential for these symptoms includes gastric motility disorders (i.e., gastroparesis), gastroenteritis, small bowel obstruction, electrolyte abnormalities, and extrinsic compression from pancreatic pseudocysts or neoplasms.

- Distal gastric neoplasms—Gastric neoplasms include benign polyps, adenocarcinoma, neuroendocrine tumors, lymphoma, B-cell mucosa-associated lymphoid tissue (MALT) lymphomas, GI stromal tumors, leiomyomas, and leiomyosarcomas. Any gastric neoplastic process can cause upper GI bleeding, epigastric pain, and luminal obstruction, and a differential similar to peptic ulcer disease should be considered.

PATIENT HISTORY AND PHYSICAL FINDINGS

- All patients should undergo a thorough history and physical exam with questions focusing on the nature of the symptoms, specifically determining the relationship between symptoms and eating, deciphering whether symptoms are acute or chronic, and determining the severity of the symptoms. A vast majority of patients will have abdominal pain. Pain related to peptic ulcer disease that results from the corrosive effect of gastric acid on vulnerable GI mucosa and typically occurs in the epigastrum is described as gnawing or burning and follows a daily cycle. This pain typically arises shortly after eating breakfast and persists until lunch at which time the oral intake alleviates the pain. Relief is transient and pain recurs in the early afternoon and again persists until dinner. Meals, specifically ones consisting of milk and dairy products, and antacids provide temporary relief from ulcer pain. Acute, severe epigastric pain can signify ulcer perforation, whereas back pain suggests ulcer penetration into the pancreas.

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Table 1: Indications for Antrectomy

<table>
<thead>
<tr>
<th>Peptic ulcer disease</th>
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<tbody>
<tr>
<td>Intractability—persistent or recurrent gastric ulcers in order to rule out malignancy</td>
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<tr>
<td>Perforation—distal gastric or duodenal ulcer</td>
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<tr>
<td>Bleeding—type I, II, or III gastric ulcer</td>
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<td>Obstruction—any gastric outlet or duodenal obstruction due to chronic ulcer scarring</td>
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<th>Neoplasm</th>
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<tr>
<td>Benign—single giant gastric polypl or multiple gastric polyps not amenable to endoscopic resection, leiomyoma, lipoma</td>
</tr>
<tr>
<td>Malignant—leiomyosarcoma, gastrointestinal stromal tumor, early-stage adenocarcinoma, neuroendocrine tumor</td>
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<tr>
<td>Gastroparesis—role is debated in chronic gastroparesis only after extensive workup</td>
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Acute or chronic upper GI bleeding can signify complicated ulcer disease and may present as melena, weakness, fatigue, general malaise, or a recent diagnosis of anemia.

- Risk factors for developing ulcer disease include a history of *H. pylori* infection; smoking; Zollinger-Ellison syndrome; and use of nonsteroidal antiinflammatory drugs (NSAIDs), steroids, and other immunosuppressive medications. Therefore, an accurate medication list should be obtained and reviewed with the patient. History of previous ulcer disease should be elicited, and the success and timing of previous treatment modalities should be documented. Presence of *H. pylori* infection, completion of antibiotic therapy, and documentation of eradication is crucial (Table 3). Ulcers that persist despite appropriate

<table>
<thead>
<tr>
<th>Location</th>
<th>Common Complications</th>
<th>Acid Secretion</th>
<th>Other</th>
</tr>
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<tbody>
<tr>
<td>Esophageal</td>
<td>Gastroesophageal junction and distal esophagus</td>
<td>Hemorrhage</td>
<td>High</td>
</tr>
<tr>
<td>Gastric</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>Gastric body on lesser curvature near the angularis incisura</td>
<td>Perforation</td>
<td>Normal or low</td>
</tr>
<tr>
<td>II</td>
<td>Two ulcers; gastric body and duodenal ulcer</td>
<td>Hemorrhage, obstruction, or perforation</td>
<td>High</td>
</tr>
<tr>
<td>III</td>
<td>Prepyloric</td>
<td>Hemorrhage, perforation</td>
<td>High</td>
</tr>
<tr>
<td>IV</td>
<td>High on lesser curvature</td>
<td>Hemorrhage</td>
<td>Low</td>
</tr>
<tr>
<td>V</td>
<td>Anywhere</td>
<td>Perforation</td>
<td>Normal</td>
</tr>
<tr>
<td>Duodenal</td>
<td>95% occur within 2 cm of pylorus</td>
<td>Hemorrhage, obstruction, or perforation</td>
<td>Normal or high</td>
</tr>
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</table>

NSAID, nonsteroidal antiinflammatory drug.
Table 3: *Helicobacter pylori* Testing

<table>
<thead>
<tr>
<th>Method</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Other</th>
</tr>
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<tbody>
<tr>
<td>Urease breath test</td>
<td>&gt;95%</td>
<td>&gt;90%</td>
<td>Determines active infection but must stop PPI 2 wk prior; takes 30–60 min and is used to confirm eradication</td>
</tr>
<tr>
<td>Fecal antigen detection Serology</td>
<td>&gt;90%</td>
<td>&gt;95%</td>
<td>Determines active infection but is positive for up to 12 wk after eradication</td>
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<tr>
<td>Invasive (endoscopic) Biopsy urease test</td>
<td>&gt;90%</td>
<td>&gt;95%</td>
<td>Decreased sensitivity with ongoing PPI, H2 antagonist, antibiotic, and bismuth compound use or with recent GI bleeding</td>
</tr>
<tr>
<td>Histology</td>
<td>&gt;90%</td>
<td>&gt;95%</td>
<td>Multiple available stains and widely used; increased sensitivity with increased biopsies</td>
</tr>
<tr>
<td>Culture</td>
<td>80%</td>
<td>100%</td>
<td>Difficult and expensive; reserved for persistent infections and antibiotic sensitivity testing</td>
</tr>
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PPI, proton pump inhibitor; GI, gastrointestinal.

Clinical presentation of gastric ulcer disease varies. Symptoms depend on the location of the ulcer and the type of causative acid-secreting factor (H. pylori, C. ulcer, NSAID). Gastric ulcers are typically midline and centered on the lesser curvature, whereas duodenal ulcers are generally found in the prepyloric area and the pyloric channel. All gastric ulcers frequently occur in the first portion of the duodenum, the prepyloric area, and the pyloric channel. All gastric ulcers are sharply demarcated; often have exposed underlying submucosa; and frequently occur in the first portion of the duodenum, the prepyloric area, and the pyloric channel. All gastric ulcers should be biopsied at least six times at the ulcer edge and brush biopsied to evaluate for underlying malignancy. Even if negative for malignancy, repeat endoscopy after medical treatment is indicated to evaluate for therapeutic response, and in instances of persistent or intractable disease, ulcer resection is indicated.

A subjective assessment of nutrition and functional status is necessary to evaluate the patient’s ability to tolerate a major surgical procedure.

**IMAGING AND OTHER DIAGNOSTIC STUDIES**

- Diagnostic evaluation of suspected peptic ulceration and upper GI lesions can include endoscopy, contrast radiography, and computed tomography (CT) (FIG 1).
- Endoscopy is felt to be superior because it allows for tissue sampling. Sampling of gastric mucosa can be used to confirm the presence of *H. pylori*. On endoscopy, ulcers are sharply demarcated; often have exposed underlying submucosa; and frequently occur in the first portion of the duodenum, the prepyloric area, and the pyloric channel. All gastric ulcers

...treatment of *H. pylori*, cessation of NSAID use, or are found in *H. pylori*-negative patients should raise suspicion for underlying malignancy.

- A gastric lesion can also present with epigastric pain and obstruction. This pain is typically vaguer in nature and lacks a gnawing or burning component. Furthermore, these patients may describe a sensation of persistent fullness and early satiety despite hunger.

- A subjective assessment of nutrition and functional status is necessary to evaluate the patient’s ability to tolerate a major surgical procedure.

**FIG 1** - Radiology. A. CT scan of pyloric obstruction from chronic pyloric inflammation. B. Endoscopic image of near-complete pyloric obstruction from chronic pyloric inflammation (left); endoscopic image of a large gastric ulcer with a central necrotic region (right). C. Contrast radiography demonstrating pyloric obstruction from chronic pyloric inflammation.
Several factors must be considered when deciding between performing a Billroth I and Billroth II procedure. The advantage to a Billroth I procedure is that the anatomic arrangement of the GI tract is preserved, which maintains the innate regulatory pathways of bicarbonate and pancreatic enzymes and significantly decreases the rate of postprandial dumping. Unfortunately, the lack of a pylorus results in bile reflux gastritis in a majority of patients. A Billroth I procedure cannot always be performed due to inflammation and scarring from prepyloric, pyloric, or duodenal ulcers. In these instances, the Billroth II procedure allows for a tension-free anastomosis of noninflamed tissue but introduces the problems of potential afferent loop syndrome and bile reflux gastritis, whereas a Roux-en-Y gastrojejunostomy diminishes the occurrence of bile reflux at the cost of a second anastomosis. Lastly, in cases of invasive neoplasms or concerning gastric masses, a Billroth II procedure with or without reconstruction with a Roux-en-Y gastrojejunostomy is preferred as it allows for dissection of much wider margins and is less likely to obstruct in the unfortunate setting of recurrent disease.\textsuperscript{10,11}

Positioning

- The patient should be positioned supine with arms out. A urinary catheter and a nasogastric tube should be placed to decompress the stomach. Positioning should allow for attachment of a self-retaining retractor system to the operating room table.

**EXPOSURE**

- A midline supraumbilical incision is made and carried to the level of the xiphoid. The falciform ligament is divided and a self-retaining retractor system is placed to widely expose the upper abdomen (FIG 2).

**GASTRIC MOBILIZATION**

- Mobilization of the distal stomach is best achieved by starting on the greater curvature. The gastrocolic ligament is identified and incised to enter the lesser sac (FIG 3). Downward traction on the transverse colon and upward traction on the stomach will help expose this plane (FIG 4). Identification of the right and left gastroepiploic vessels along the greater curvature is essential. In benign disease, the plane of dissection can be very close to the stomach inside the gastroepiploic vessels. A large part of this plane is avascular and can be divided with electrocautery, whereas encountered vessels should be divided between clamps and ligated with 3-0 silk ligatures. Once the lesser sac is identified, electrosurgical devices can be used to further mobilize the greater curvature (FIG 5). Proximally, dissection is carried to the midpoint of the greater curvature preserving the left gastroepiploic artery. Distally, the plane is developed beyond the pylorus to the duodenum and, once identified, the right gastroepiploic artery should be clamped, ligated with 2-0 silk ligatures, and divided. Here, one should be aware of the underlying pancreatic tissue and dissection should be meticulous.

- Dissection along the greater curvature allows entrance to the lesser sac, and the stomach can be lifted superiorly exposing its posterior surface and the congenital...
Traction on the stomach and the colon will aid dissection through the gastrocolic ligament allowing entrance to the lesser sac. As seen here, the lesser sac will be a true space that lies deep to the greater omentum.

Once the lesser sac is identified, the greater curvature can be dissected both proximally and distally taking care to identify the gastroepiploic vessels.

Dissection of the greater curvature. A. Division of the greater omentum along the greater curvature will allow access to the lesser sac. The gastroepiploic vessels should be identified and care should be taken to not damage the transverse colon and its mesentery. B. Cross-sectional view of the upper abdomen demonstrating the plane of dissection that allows entrance to the lesser sac and mobilization of the greater curvature. This dissection can start in the transparent pars flaccida and is carried proximally to the incisura and distally to the right gastric artery, which should be clamped, ligated with 2-0 silk ligatures, and divided. Again, electrocautery or electrosurgical devices can be used along the lesser curvature. One must be aware of an aberrant or replaced left hepatic artery originating from the left gastric artery and traversing the gastrohepatic ligament. If encountered, attempts should be made to preserve this vessel. After clamping but before division of the right gastric artery, blood flow to the liver should be confirmed by palpation of the hepatoduodenal ligament.
Duodenal ulcer

**FIG 8** Division of the stomach.

**DIVISION OF THE STOMACH**

- A stapling device is used to divide the stomach along a plane from just proximal to the incisura angularis on the lesser curvature to a point on the greater curvature two-thirds of the way from the gastroesophageal junction to the pylorus. Some recommend placing Babcock forceps distal to the staple line to prevent sliding or rotation of the gastric mucosa as the stapler is closed. This will help ensure a clean, even cut across the anterior and posterior layers of gastric mucosa (FIG 8).

- Division of the duodenum varies based on the type of reconstruction planned. The duodenum is divided between Potts clamps for a Billroth I procedure in order to facilitate the gastroduodenal anastomosis. If possible, a stapling device should be used to divide the duodenum in a Billroth II procedure.

**FIG 7** Dissection of the lesser curvature.

**FIG 6** Dissection of the greater curvature allows for superior retraction of the stomach exposing its posterior surface and the underlying pancreas. Here, the lesser curvature and the left gastric artery are seen from the posterior aspect of the stomach.

**RECONSTRUCTION:**

**GASTRODUODENOSTOMY (BILLROTH I)**

- If a Billroth I anastomosis is planned, a Kocher maneuver is needed to mobilize the duodenum.

- The duodenum is transected distal to the diseased area but proximal to the ampulla between Potts clamps, and the specimen is handed off the sterile field (FIG 9). It is essential that all gastric antrum be resected to prevent persistent ulcer disease from retained gastric antrum. If there is question, a frozen section can be sent to confirm duodenal tissue at the resection line.

At this point, one can gauge the mobility of the stomach, and if limited, the attachments of the fundus to the base of the diaphragm can be divided along with the gastroepiploic vessels. This further mobilization should allow for a tension-free gastroduodenal anastomosis. If there
is concern about the ability to perform a tension-free anastomosis due to difficulty mobilizing the duodenum or the stomach, then a Billroth II procedure should be performed.

For a Billroth I procedure, the duodenal stump is cleared of adjacent adipose tissue for roughly 1.5-cm distance in preparation for an end-to-end anastomosis. This anastomosis is constructed in standard two-layer fashion. Silk 3-0 seromuscular “stay” sutures are placed at the ends of the anastomosis to approximate the duodenum to the inferior aspect of the gastric staple line and the posterior layer is completed with interrupted 3-0 silk seromuscular sutures. Next, the inferior portion of the gastric staple line is removed using electrocautery for a length that correlates with the width of the duodenal stump. Two running 3-0 polydioxa-none (PDS) sutures are used for the inner layer and an anterior layer of interrupted 3-0 silk seromuscular sutures completes the anastomosis. The remaining gastric staple line can be oversewed with interrupted 3-0 silk sutures (FIG 10).
gastrojejunal anastomosis is formed by creating a posterior wall gastrotomy and an antimesenteric enterotomy. Care is taken to ensure that the anastomosis is away from the prior gastric staple line on the posterior wall of the stomach. The common enterotomy is then closed with a 3-0 PDS full-thickness layer and an overlying layer of interrupted 3-0 silk sutures (FIG 11). Interrupted 3-0 silk sutures are then used to close the colonic mesentery defect to prevent bowel herniation (FIG 12).

FIG 12 • Retrocolic gastrojejunostomy.

PEARLS AND PITFALLS

| Indication | • Today, there is no role for antrectomy in treatment of noncomplicated peptic ulcer disease.  
| • A vast majority of peptic ulcers complicated by hemorrhage or perforation can be controlled with less invasive procedures. |
| Operative planning | • Preoperative endoscopy is essential to evaluate for the scope of disease and to allow biopsy of persistent gastric ulcers to rule out malignancy.  
| • In the setting of malignancy, preoperative staging is warranted and a more extensive, oncologic resection may be indicated. |
| Laparotomy | • Prior to extensive dissection, the extent of disease should be gauged to determine the operative plan and feasibility of a Billroth I anastomosis. |
| Gastric mobilization | • With mobilization of the greater curvature, the middle colic vein is at risk of injury. Caudal traction on the transverse colon during dissection can help prevent an inadvertent injury. |
| Duodenal transection | • Great care should be taken to not fracture or injure the head of the pancreas, which can be closely adherent due to chronic inflammation.  
| • If the common bile duct is difficult to identify, a cholecystectomy can be performed and a catheter can be placed in the cystic duct. Palpation of the catheter can aid in safely identifying the portal structures during dissection of the duodenum. |
Reconstruction
- Billroth I and II procedures are performed with acceptable postoperative morbidity.
- A Billroth II anastomosis should be performed if there is concern about mobility of the duodenum or the stomach.
- A short afferent limb (10 to 15 cm from the ligament of Treitz) can help minimize the likelihood of significant postoperative complications.

POSTOPERATIVE CARE

- Unless concerning comorbidities exist, patients can be monitored on the hospital floor/ward. A nasogastric tube is positioned intraoperatively in the proximal stomach and typically can be removed on postoperative day (POD) 1. There is no role for postoperative antibiotic prophylaxis. Unless contraindicated, all patients receive chemical prophylaxis for deep vein thrombosis and are encouraged to ambulate on POD 1 and begin pulmonary toilet with incentive spirometry. There is no convincing evidence for routinely placing abdominal drains after Billroth I and Billroth II procedures. If there is concern for the GI anastomosis or adequate closure of the duodenal stump, a closed suction abdominal drain can be placed. Oral intake is reintroduced on POD 3 and advanced as tolerated.

OUTCOMES

- Serious morbidity from postgastrectomy syndromes develops in 3% to 5% of patients.
- Thirty-day mortality for uncomplicated gastric ulcer disease is 1% to 2% and increases in emergency settings.\(^\text{11}\)

COMPLICATIONS

- After an antrectomy, short-term complications include delayed gastric emptying, anastomotic leak, bleeding, and pancreatitis. Long-term complications include the postgastrectomy syndromes (described below) and anastomotic stenosis. Additionally, chronic anemia, neuropathy, and osteopenia can result from iron, copper, and calcium malabsorption due to bypassing of the proximal small bowel in a Billroth II procedure.\(^\text{12}\)

- Postgastrectomy syndromes
  - Afferent loop syndrome
    - Postprandial right upper abdominal colicky pain that accumulates in bilious emesis that alleviates the pain
    - Results from chronic dilation, obstruction, or stasis of the duodenum (afferent limb) after a Billroth II procedure
    - Rarely occurs but can be corrected by revision of the Billroth II, conversion to a Roux-en-Y reconstruction, or performing an afferent to efferent bypass (Braun enterointerostomy)
  - Reflux gastritis—Patients report epigastric burning pain resulting from reflux of bile into the stomach. As expected, bile reflux is more common after Billroth II and Billroth II procedures than Roux-en-Y reconstruction and if severe can be treated by conversion to a Roux-en-Y reconstruction.
  - Dumping—Early dumping presents as crampy abdominal pain and diarrhea shortly after eating due to the large hyperosmolar load of simple sugars which quickly enter the small bowel in the absence of a pylorus. Late dumping occurs roughly 2 hours postprandial with the symptoms of hypoglycemia likely due to insulin response to the large sugar bolus. Dumping occurs in 5% of postgastrectomy and is controlled with diet modifications, and rarely, octreotide is given with success in severe and refractory cases.
  - Retained gastric antrum—Incomplete antrectomy with retained G cells within the duodenal stump can result in recurrent ulceration from continued intense gastrin secretion. Exposure of the jejunum to high levels of acid results in an anastomotic or marginal ulcer. A sodium 99m technetium scan identifies antral tissue and reexcision is needed for complete symptom relief.

REFERENCES