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OPERATIVE TREATMENT OF ULCER DISEASE

Part of "CHAPTER 22 - DUODENAL ULCER"

Surgical Goals

Operative intervention is reserved for the treatment of complicated ulcer disease. Three complications are most common and constitute the indications for peptic ulcer surgery—hemorrhage, perforation, and obstruction. The first goal in the surgical treatment of the complications of ulcer disease should be alteration of the ulcer diathesis so that ulcer healing is achieved and recurrence is minimized. The second goal is treatment of coexisting anatomic complications, such as pyloric stenosis or perforation. The third major goal should be patient safety and freedom from undesirable chronic side effects. To achieve these goals, the gastric surgeon can direct therapy through endoscopic, radiologic, or operative means, the appropriate choice depending on the clinical circumstances.

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Operative Procedures

A number of operative procedures have been used to treat peptic ulcer, but three procedures—truncal vagotomy and drainage, truncal vagotomy and antrectomy, and proximal gastric vagotomy—have been most widely used. In the operative treatment of peptic ulcer disease, vagotomy has had a central role. With increasing frequency, surgical therapy of peptic ulcer is directed exclusively at correction of the immediate problem (e.g., closure of duodenal perforation) without gastric denervation. The underlying ulcer diathesis is then addressed after surgery by antibiotic therapy directed at *H. pylori.* This approach is applicable to most patients with peptic ulcer undergoing emergent operation and predicts a diminishing role for vagotomy in the future. Nonetheless, vagotomy is currently central to the surgical management of complicated ulcer disease, and an understanding of the physiologic alterations attending vagotomy is crucial to gastric surgeons.

Division of both vagal trunks at the esophageal hiatus—truncal vagotomy—denervates the acid-producing fundic mucosa as well as the remainder of the vagally supplied viscera (Fig. 22-4). Because denervation impedes normal pyloric coordination and can result in impairment of gastric emptying, truncal vagotomy must be combined with a procedure to eliminate pyloric sphincteric function. Usually, gastric drainage is ensured by performance of a pyloroplasty. Several methods of pyloroplasty have been described; often they are referred to eponymously (Fig. 22-5). The Heineke-Mikulicz pyloroplasty is performed by making a longitudinal incision of the pyloric sphincter extending into the antrum and the duodenum for approximately 2 cm on either side. The incision is closed transversely, thereby increasing the lumen of the pyloric channel. A Finney pyloroplasty is formed as a

gastroduodenostomy with transection of the pyloric sphincter. The inner curve of the duodenum is approximated to the dependent aspect of the antrum and pyloric channel. A U-shaped incision is then made, crossing the pylorus. The pyloroplasty is completed by suturing the anterior duodenal wall to the antrum. For some cases in which severe pyloric scarring makes division of the pyloric channel difficult or hazardous, the Jaboulay procedure, a side-to-side gastroduodenostomy, can be used; this procedure differs from the Finney pyloroplasty only in that the incision is not completed across the pyloric sphincter.

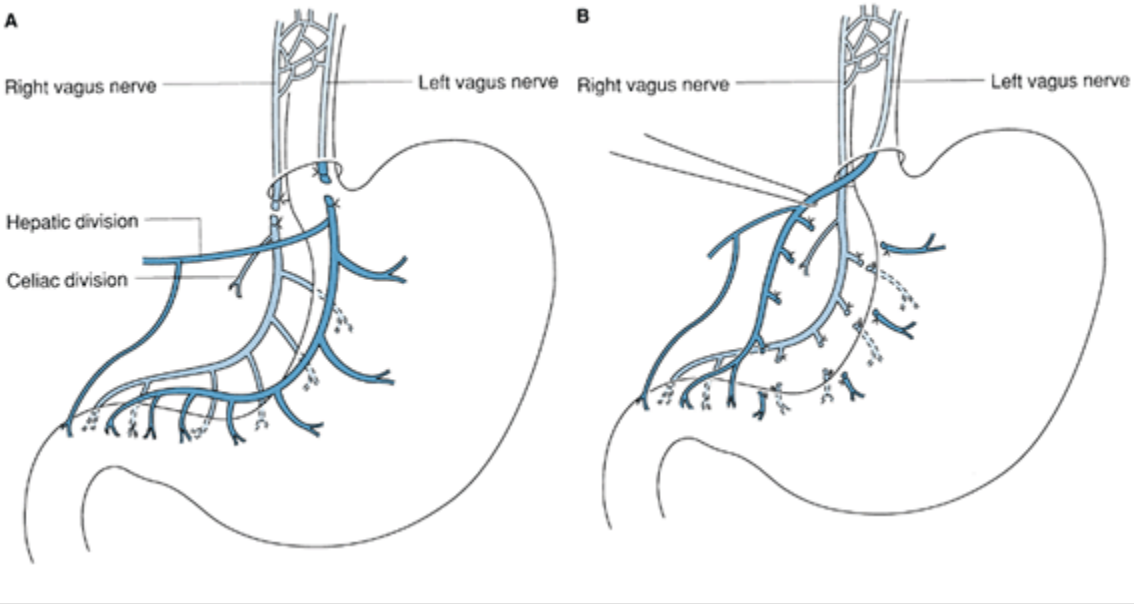

Figure 22.4. Truncal vagotomy and proximal gastric vagotomy. *(A)* With truncal vagotomy, both nerve trunks are divided at the level of the diaphragmatic hiatus. *(B)* Proximal gastric vagotomy involves division of the vagal fibers that supply the gastric fundus. Branches to the antropyloric region of the stomach are not transected, and the hepatic and celiac divisions of the vagus nerves remain intact.

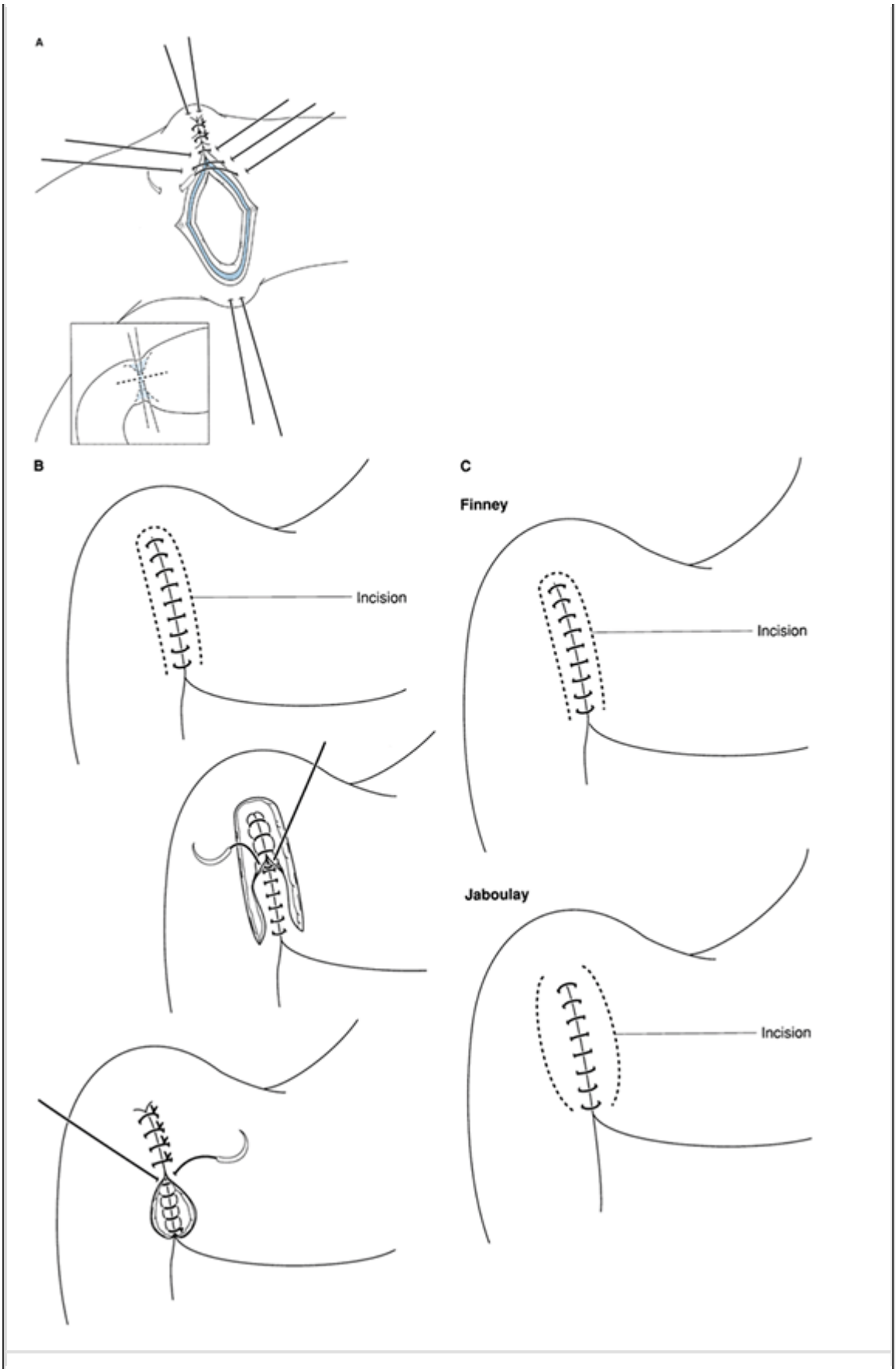
A
B
Incision
C
Finney
Incision
Jaboulay
Incision

Figure 22.5. Pyloroplasty formation. A Heineke-Mikulicz pyloroplasty *(A)* involves a longitudinal incision of the pyloric sphincter followed by a transverse closure. The Finney pyloroplasty *(B)* is performed as a gastroduodenostomy with division of the pylorus. The Jaboulay pyloroplasty *(C)* differs from the Finney procedure in that the pylorus is not transected.

Truncal vagotomy can also be combined with resection of the gastric antrum to effect a further reduction in acid secretion, presumably by removing antral sources of gastrin. The limits of antral resection are usually defined by external landmarks, rather than the histologic transition from fundic to antral mucosae. The stomach is divided proximally along a line from a point above the incisura angularis to a point along the greater curvature midway from the pylorus to the gastroesophageal junction. Restoration of gastrointestinal continuity by a gastroduodenostomy is termed a Billroth I reconstruction. A Billroth II procedure uses a gastrojejunostomy (Fig. 22-6).

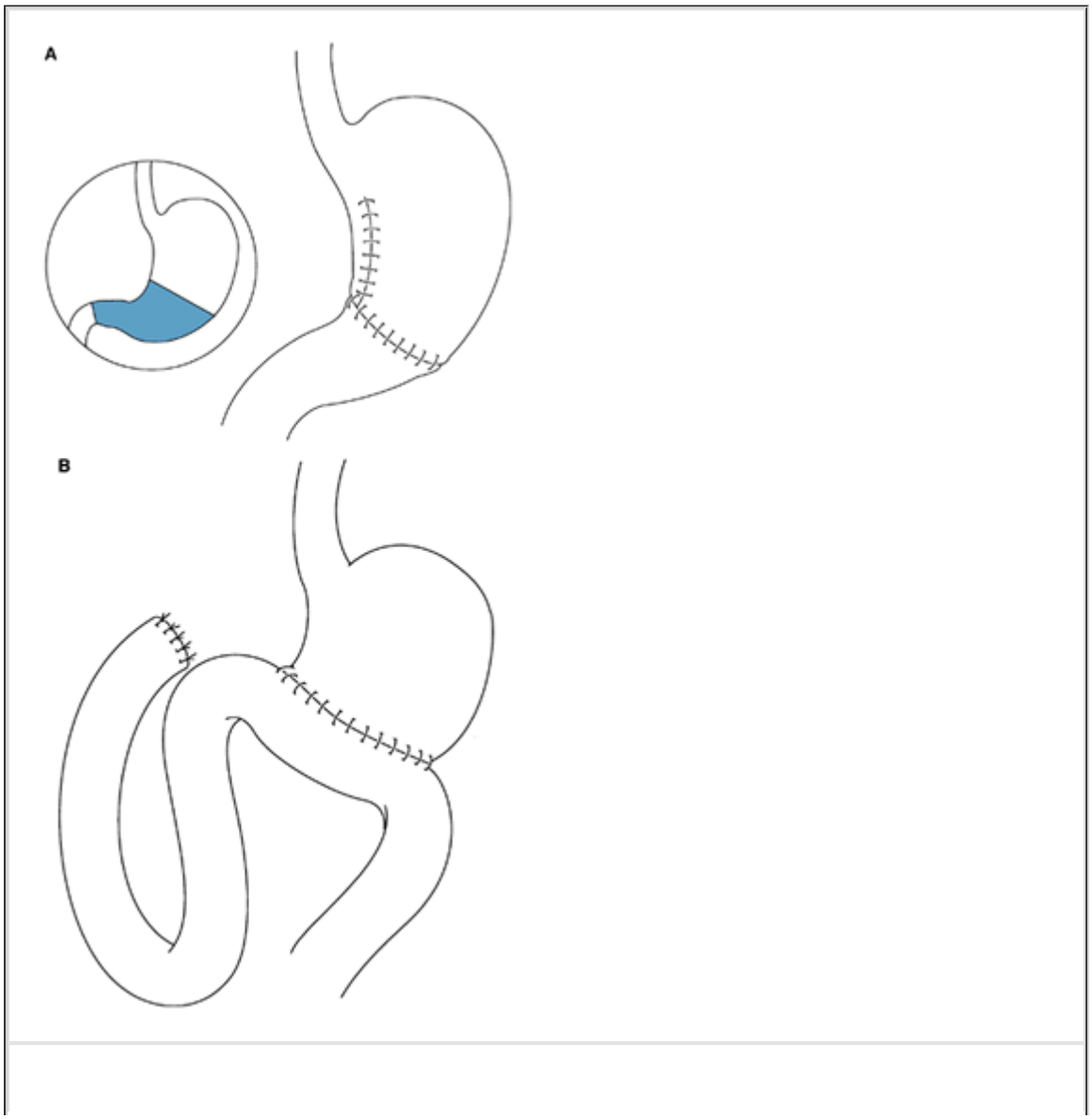

Figure 22.6. Antrectomy involves resection of the distal stomach *(blue area in inset)*. Restoration of gastrointestinal continuity may be accomplished as a Billroth I gastroduodenostomy *(A)* or Billroth II gastrojejunostomy *(B)* reconstruction.

Proximal gastric vagotomy differs from truncal vagotomy in that only the nerve fibers to the acid-secreting fundic mucosa are divided (Fig. 22-4). Vagal nerve fibers to the antrum and pylorus are left intact, and the hepatic and celiac divisions are not transected. The denervation begins approximately 5 cm from the pylorus and extends proximally along the lesser curvature. In proximal gastric vagotomy, the distal esophagus is also skeletonized for a distance of 5 to 7 cm to divide any vagal fibers traveling to the fundus intramurally within the esophagus. The operation has also been called parietal cell vagotomy to emphasize its most important functional consequence.

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Physiologic Consequences of Operation

Division of efferent vagal fibers directly affects acid secretion by reducing cholinergic stimulation of parietal cells. In addition, vagotomy also diminishes parietal cell responsiveness to gastrin and histamine. Basal acid secretion is reduced by approximately 80% in the immediate postoperative period. Basal acid secretion increases slightly within months of surgery but remains unchanged thereafter. The maximal acid output in response to exogenously administered stimulants such as pentagastrin is reduced by approximately 70% in the early period after surgery. After 1 year, pentagastrin-stimulated maximal acid output rebounds to 50% of prevagotomy values but remains at this level on subsequent testing. Acid secretion due to endogenous stimulation by a liquid meal is reduced by 60% to 70% relative to normal subjects. The acid-reducing properties of proximal gastric vagotomy and truncal vagotomy are roughly equivalent in most series (Table 22.3).

GASTRIC EFFECTS
Decreased basal acid output
Reduced cholinergic input to parietal cells
Decreased stimulated maximal acid output
Diminished sensitivity to histamine and gastrin
Decreased meal-induced acid secretion
Increased fasting postprandial gastrin
Gastrin cell hyperplasia
Accelerated liquid emptying
Altered emptying of solids
NONGASTRIC EFFECTS
Decreased pancreatic exocrine secretion
Decreased pancreatic enzymes and bicarbonate
Decreased postprandial bile flow
Increased gallbladder volumes
Diminished release of vagally mediated peptide hormones

Table 22.3. PHYSIOLOGIC ALTERATIONS CAUSED BY TRUNCAL VAGOTOMY

The inclusion of antrectomy with truncal vagotomy causes further reductions in acid secretion. Pentagastrin-stimulated maximal acid output is reduced by 85% relative to values recorded before surgery. Little rebound in acid secretion occurs with the passage of time.

Truncal vagotomy and proximal gastric vagotomy both cause postoperative hypergastrinemia. Fasting gastrin values are elevated to approximately twice preoperative levels, and the postprandial response is exaggerated. Immediately after vagotomy, hypergastrinemia appears to be due to decreased luminal acid, with loss of feedback inhibition of gastrin release. Loss of vagal inhibitory pathways can also be important. Chronic hypergastrinemia, sustained long term in most cases, is caused by gastrin cell hyperplasia in addition to loss of inhibitory feedback. When antrectomy is added to vagotomy, circulating gastrin levels are decreased. Basal gastrin values are reduced by approximately half and postprandial gastrin levels by two thirds. The major form of circulating hormone after antrectomy is gastrin 34, released from the duodenum.

Operations that involve vagotomy alter gastric emptying. Proximal gastric denervation abolishes vagally mediated receptive relaxation. Thus, for any given volume ingested, the intragastric pressure rise is greater and the gastroduodenal pressure gradient higher than in normal subjects. As a result, emptying of liquids, which depends critically on the gastroduodenal pressure gradient, is accelerated after proximal gastric vagotomy. Because nerve fibers to the antrum and pylorus are preserved, the function of the distal stomach to mix and triturate solid food is preserved, and emptying of solids is nearly normal in patients who have undergone proximal gastric vagotomy. Truncal vagotomy affects the motor activities of both proximal and distal stomach. Solid and liquid emptying rates are usually increased when truncal vagotomy is accompanied by pyloroplasty.

Truncal vagotomy affects a number of other gastrointestinal functions because of the removal of efferent vagal innervation. Pancreatic exocrine secretion in response to a meal is diminished, with decreased bicarbonate and enzyme outputs. Postcibal biliary secretion is decreased, and gallbladder distention is observed. Fecal fat excretion doubles after truncal vagotomy, although clinical steatorrhea is unusual. Stimulated release of a number of gastrointestinal hormones—including pancreatic polypeptide, cholecystokinin, and secretin—is decreased. In most instances, these extragastric alterations in digestive function are subclinical. Proximal gastric vagotomy, in which the vagal innervation to nongastric viscera is preserved, produces fewer physiologic alterations than does truncal vagotomy.

A number of prospective, randomized trials have compared the various surgical options in terms of postoperative symptoms, including dumping, diarrhea, weight loss, and disturbance of lifestyle (Table 22.4). In most comparisons, proximal gastric vagotomy has proved superior to other operations in these measures. Dumping, a postprandial symptom complex of abdominal discomfort, weakness, and vasomotor symptoms of sweating and dizziness, occurs in 10% to 15% of patients with truncal vagotomy and antrectomy in the

early postoperative period and is chronically disabling in 1% to 2%. After truncal vagotomy and pyloroplasty, dumping is present initially in 10%, and remains severe in approximately 1%. Permanent symptoms of dumping are rare after proximal gastric vagotomy. The incidence of diarrhea, which is presumably caused by denervation of the pylorus and small bowel and by elimination of pyloric function, parallels the incidence of dumping after truncal vagotomy and antrectomy or pyloroplasty. Persistent or disabling diarrhea is present in less than 1% of patients after proximal gastric vagotomy. After truncal vagotomy, weight loss averages 2 kg in the first postoperative year, whereas with proximal gastric vagotomy, a weight gain is recorded. Reoperation after proximal gastric vagotomy is rarely needed for symptoms resulting from the operation.

	PGV (%)	TV + P (%)	TV + A (%)
Mortality rate	0	0.5–1	1–2
Acid reduction			
Basal	80	70	85
Stimulated	50	50	85
Ulcer recurrence	10	12	1–2
Gastric emptying			
Liquids	Accelerated	Accelerated	Accelerated
Solids	No change	Accelerated	Slowed
Dumping			
Mild	<5	10	10–15
Disabling	0	1	1–2
Diarrhea			
Mild	<5	25	20
Disabling	0	2	1–2

PGV, proximal gastric vagotomy; TV + P, truncal vagotomy and pyloroplasty; TV + A, truncal vagotomy and antrectomy.

Table 22.4. CLINICAL RESULTS OF DUODENAL ULCER SURGERY

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Proximal gastric vagotomy has the lowest operative mortality rate, the lowest incidence of postoperative symptoms, and an acceptable risk of recurrent ulcer. Collected series of proximal gastric vagotomies have reported an operative mortality rate of less than 0.05% (20), lower than the reported mortality rate for any other gastric procedure for peptic ulcer. Truncal vagotomy and pyloroplasty has a reported mortality rate of 0.5% to 0.8%, whereas the mortality rate after truncal vagotomy and antrectomy approximates 1.5%.

The lower incidence of postoperative symptoms is obtained at the cost of a higher postoperative ulcer recurrence rate (22). The reported recurrence rates for proximal gastric vagotomy are variable, probably reflecting differences in experience and individual surgical skill. In addition, all prospective surgical series examining ulcer recurrence rates were reported in the era before the pathogenic role of *H. pylori* was appreciated. With appropriate use of postoperative antimicrobials directed against *H. pylori,* these ulcer recurrence rates would currently be expected to be much lower. Although recurrence rates

(without *H. pylori* treatment) as low as 5% have been reported, a more generally accepted figure is 10%. This rate is similar to that after truncal vagotomy and drainage (approximately 12%) but considerably greater than that reported after truncal vagotomy and antrectomy (1% to 3%). The reported ulcer recurrence rates after proximal gastric vagotomy can be adversely affected by the inclusion of prepyloric and pyloric channel ulcers. For reasons that are not clear, proximal gastric vagotomy is significantly less effective when used to treat ulcers in this position than when used for duodenal ulceration.