Postgastrectomy Syndromes

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KEYWORDS

• Postgastrectomy syndromes • Diagnosis • Management

The first postgastrectomy syndrome was noted not long after the first gastrectomy was performed: Billroth[1] reported a case of epigastric pain associated with bilious vomiting as a sequel of gastric surgery in 1885. Several classic treatises exist on the subject; we cannot improve on them and merely provide a few references for the interested reader.[1–3]

However, the indications for gastric resection have changed dramatically over the past 4 decades, and the overall incidence of gastric resection has decreased. The most marked reduction in the frequency of gastric resection has occurred among patients with peptic ulcer disease. For example, in Olmstead County, Minnesota, the incidence of elective operations on previously unoperated patients declined 8-fold during the 30-year study period between 1956 and 1985[4] and undoubtedly has declined even further since. One population-based study concluded that elective surgery for ulcer disease had "virtually disappeared by 1992–1996."[5] Whereas emergency operations for bleeding and perforation are still encountered, acid-reducing procedures are being performed less frequently in these situations in favor of a damage control approach.[6] Even for gastric cancer, resection rates decreased approximately 20% from 1988 to 2000 in the United States.[7] An estimated 21,000 new cases of stomach cancer occurred in the United States in 2010,[8] so that the number of cases of gastric resection for cancer is probably less than 15,000 per year in the United States. The virtual disappearance of elective surgery for peptic ulcer has also changed the demographic profile of the postgastrectomy patient: patients who have gastric cancer tend to be older and there is only a slight male preponderance. These significant changes in the gastric surgery population make it worthwhile to revisit postgastrectomy syndromes.

The frequency with which postgastrectomy symptoms/syndromes are found can depend on how hard they are looked for. Loffeld,[9] in a survey of 124 postgastrectomy

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patients, most of whom had undergone surgery more than 15 years earlier, found that 75% suffered from upper abdominal symptoms, and 1 or more symptoms that indicate dumping were found in 70% of patients who had undergone Billroth-II (B-II) reconstruction. However, the lack of age-matched and sex-matched controls in this study may have overstated the frequency of symptoms caused by the surgical procedure. Mine and colleagues[10] conducted a large survey of 1153 patients after gastrectomy for cancer and found that 67% reported early dumping and 38% late dumping. By contrast, Pedrazzani and colleagues[11] surveyed 195 patients who underwent subtotal gastrectomy and B-II reconstruction for gastric adenocarcinoma for up to 5 years postoperatively, and concluded that "the incidence of late complications was low and the majority of them recovered within one year after surgery." Our personal experience tends to corroborate that most patients have a good functional result after gastric resection for cancer, provided that the cancer is cured. This article focuses on the small proportion of patients with severe, debilitating symptoms; these symptoms can challenge the acumen of the surgeon who is providing the patient's long-term follow-up and care.

This article does not attempt to deal with the sequelae of bariatric surgery, which has become the most common indication for elective gastric surgery with an estimated 71,190 patients having laparoscopic gastric bypass in the United States in 2006.[12]

NUTRITIONAL AND METABOLIC SEQUELAE

Virtually all patients who submit to subtotal or total gastrectomy experience significant weight loss within the first several months postoperatively. On average, the amount of weight lost is approximately 10% of preoperative weight. Body weight usually stabilizes by 3 months postoperatively, in the absence of superimposed clinical problems. Body composition studies performed at 6 and 12 months postoperatively reveal that the weight loss is comprised entirely of body fat and that lean body cell mass is unchanged.[13] If this loss of body fat were the only consequence of gastric resection, one would probably ascribe it as a benefit.

The incidence of significant bone disorders after gastrectomy is considerable. Zittel and colleagues[14] found that 55% of 60 gastrectomized patients had vertebral fractures or osteopenia when studied 5 to 20 years after gastrectomy. The risk of having a vertebral deformity was increased 6-fold after gastrectomy compared with age-matched and sex-matched controls. Approximately half of the patients in this study had undergone gastrectomy for peptic ulcer disease and the other half for carcinoma, and the magnitude of bone disorder was similar for both groups. Forty of the study patients had undergone distal subtotal gastrectomy, with 20 Billroth-I (B-I) and 20 B-II reconstructions, and the prevalence of bone abnormality was similar between them. Twenty patients in this study had undergone total gastrectomy, and the prevalence of postgastrectomy bone disease was greatest in this group.[14] In a population-based study of patients who underwent surgery for peptic ulcer in Rochester, Minnesota between 1956 and 1985, the risk of distal radial, proximal femur, and vertebral fracture was increased between 2.2-fold and 4.7-fold at a median follow-up of 14.8 years.[15] The most likely explanation for postgastrectomy bone disorder seems to be decreased calcium absorption caused by bypass of upper small bowel absorptive area in patients reconstructed by B-II and Roux-Y techniques and caused by decreased dissolution and ionization of calcium salts in an acid-free environment after gastrectomy.[16] Milk intolerance, maldigestion caused by pancreatic insufficiency or pancreaticocibal asynchrony, malabsorption caused by rapid food transit, or steatorrhea leading to the

formation of insoluble calcium soaps are other possible mechanisms.[14] Several studies report that the accelerated bone density loss after gastrectomy occurs early (within the first 2–3 years postoperatively), suggesting that therapeutic intervention needs to be started soon after surgery.[17,18] In animal models, feeding soluble fiber or nondigestible disaccharides after total gastrectomy improved calcium absorption and protected against osteopenia.[19,20] Postgastrectomy effects on vitamin D metabolism and parathyroid hormone secretion have not been well characterized.

Severe copper deficiency presenting as a syndrome of ataxia, myelopathy, and peripheral neuropathy, clinically mimicking vitamin B_{12} deficiency, has been reported in patients undergoing prior gastrectomy.[21,22] Copper is absorbed primarily in the duodenum.

Anemia is common among patients undergoing prior gastrectomy. Ingested iron is absorbed primarily in the duodenum, which is bypassed with either B-II or Roux-Y reconstruction after gastric resection. Also, ingested iron must be reduced by the acid environment of the stomach to be efficiently absorbed. Ingested vitamin B_{12} requires intrinsic factor, produced by the proximal stomach, to be absorbed. In 1 study, among 72 patients evaluated for anemia after prior gastrectomy 94% had iron deficiency anemia and 79% had vitamin B_{12} deficiency; combinations of the 2 were present in most patients. By contrast, folate deficiency was uncommon, occurring in only 4% of the patients with anemia.[23] In an experimental model, ingestion of a nondigestible disaccharide prevented gastrectomy-induced iron malabsorption and anemia, possibly by cecal fermentation of the disaccharide,[24] but this therapy has not been applied in human studies. Iron deficiency anemia in a postgastrectomy patient should not be presumed to be caused by the postgastrectomy state without first ruling out other clinically significant disease in the upper and lower gastrointestinal tract by endoscopy.[25]

DUMPING SYNDROME

The dumping syndrome is caused by rapid gastric emptying as a result of loss of pyloric regulation of gastric emptying and, possibly, impaired accommodation of the proximal gastric remnant.[26] The rapid emptying of liquid-phase simple sugars presents the small bowel with a large, nonphysiologic, hyperosmolar solute load. Symptoms of early dumping include crampy abdominal pain and diarrhea within 30 minutes after oral intake, associated with weakness, light-headedness, and rapid heart rate. These symptoms are mediated by local peristaltic responses in the gastrointestinal tract, plasma volume changes, gut hormones (including insulin and glucagonlike peptide), and humoral factors including norepinephrine.[27,28] Objective criteria for diagnosis and correlation with a provocative test using an oral challenge with 50 g of glucose have been developed.[29] Late dumping occurs approximately 2 hours after meals, and the symptoms are those of hypoglycemia. The mechanism probably involves a reactive hypoglycemia brought on by the rapid and high initial glucose load presented to and absorbed by the small intestine, resulting in an inappropriately high insulin response, leading to hypoglycemia. Both early and late dumping may also be seen after surgical procedures that only incidentally remove the distal stomach or ablate the pylorus, such as the classic Whipple procedure or esophagogastrectomy for esophageal cancer in which pyloromyotomy or pyloroplasty is added.

In a retrospective study of 310 patients undergoing distal subtotal gastrectomy with B-II reconstruction for gastric cancer, Pedrazzani and colleagues[11] noted that the dumping syndrome was uncommon and tended to resolve with time, being present

in only 5% of patients 2 years after surgery. However, for the small number of patients affected, symptoms can be severe, life-altering, and debilitating.[27]

Mine and colleagues,[10] in a survey of 1153 patients after gastrectomy for gastric cancer, found on multivariate analysis that early dumping syndrome was significantly less likely to occur in older patients, patients undergoing pylorus-preserving gastrectomy, and patients having Roux-Y reconstruction after distal gastrectomy. Late dumping syndrome was significantly more frequent among patients who had early dumping syndrome or were female. Nunobe and colleagues[30] compared the results of B-I and Roux-Y reconstruction in 385 patients undergoing subtotal distal gastrectomy for early gastric cancer. In a gastrointestinal quality-of-life survey carried out 5 years after surgery, no significant differences were found between the Roux-Y and B-I groups with respect to symptoms of early or late postprandial dumping. This finding is not surprising because dumping is a result of resection of the pylorus and is unaffected by the type of reconstruction.

Initial therapy consists of dietary evaluation and counseling. Daily intake should be divided into at least 6 meals. Liquids should be avoided with meals. Diets should be high in protein and fat, and simple sugars should be avoided. Vasomotor symptoms can often be ameliorated if the patient lies down for 30 minutes after meals.

For patients with severe postgastrectomy dumping symptoms refractory to diet therapy, the somatostatin analogue octreotide is the pharmacologic therapy of choice. It acts through its inhibitory effects on insulin and gut hormone release, a delay of intestinal transit time, and inhibition of food-induced circulatory changes. A review of 7 small randomized controlled trials (RCTs) of short-acting octreotide for the treatment of severe dumping syndrome found evidence of significant clinical benefit in all studies and recommended the use of octreotide for severe or refractory dumping syndrome.[31] In a comparative study of octreotide taken 3 times a day versus monthly long-acting repeatable octreotide in 30 patients with dumping syndrome unresponsive to dietary intervention, the formulations were equally effective in blunting objective measures of the dumping syndrome, but the long-acting preparation scored significantly better on quality-of-life measures.[32] These findings were confirmed by a crossover study among 12 patients reported by Penning and colleagues.[33] However, octreotide therapy for dumping was found to lose efficacy over time: in a long-term study by Didden and colleagues,[34] 50% of patients with initial excellent relief of symptoms discontinued therapy because of side effects or loss of efficacy, and Vecht and colleagues[35] found that long-term use is frequently limited by side effects, chiefly diarrhea and steatorrhea. Acarbose, 50 mg 3 times per day, can be used for the prevention of late dumping.[36,37] Acarbose is a competitive inhibitor of α-glycoside hydrolase and delays carbohydrate digestion and absorption.

A surgical approach to the primary prevention of the dumping syndrome (the preservation of an intact pylorus in patients with early gastric cancer of the midbody of the stomach [segmental gastrectomy]) has been extensively evaluated in Japan and has been shown to significantly decrease the incidence of postoperative dumping.[38–41] The preservation of an intact anteropyloric grinding mechanism with a reduction of the capacitance function of the proximal stomach after resection can serve to promote esophageal reflux. This is the argument against extensive proximal gastrectomy with pyloric preservation. Another surgical approach to treat dumping that has been described and is included for completeness is the use of a reversed 10-cm jejunal segment to slow the transit of intestinal contents.[42,43] The twisting of a segment of bowel on its mesentery violates the tenets of gastrointestinal surgery and we do not favor it.

However, caution should be exercised when preserving the pylorus after proximal gastrectomy with jejunal interposition: Nakane and colleagues[44] found a significantly

higher incidence of delayed gastric emptying, reflux gastritis, and bile regurgitation, and suboptimal weight maintenance 1 year postoperatively among patients in whom pyloroplasty was omitted during proximal gastrectomy with jejunal interposition. These problems might be overcome by preserving vagal innervations to the pylorus.[45]

AFFERENT LOOP SYNDROME

This entity is mentioned mostly out of historical interest; we have not seen a case for many years. The virtual disappearance of this postgastrectomy syndrome is because of the dramatic decrease in the frequency with which partial gastrectomy and B-II reconstructions are performed for peptic ulcer disease. Diagnosis is made almost entirely based on the patient's history of repetitive episodes of postprandial right upper abdominal colicky pain building to a crescendo, culminating in almost projectile bilious vomiting with simultaneous relief of pain. In a patient with this history, the diagnosis may be corroborated by a computed tomography scan showing chronic dilation of the duodenum, or by right upper quadrant ultrasound after a provocative meal to show the acute distension of the afferent limb. When afferent loop syndrome is diagnosed, reoperation is required to either revise the B-II anastomosis or to convert to a Roux-Y reconstruction. Another possible solution to afferent loop syndrome is an afferent to efferent loop bypass. This bypass procedure, originally described by Braun,[46] is particularly useful when dissection of the original gastrojejunal anastomosis is not required or technically difficult.

DELAYED GASTRIC EMPTYING

Severe, prolonged delayed gastric emptying that prevents oral alimentation is rare after gastric resection for cancer. However, food retention in the gastric remnant after distal subtotal gastrectomy for cancer is commonly seen at postoperative endoscopy. Jung and colleagues[47] found food retention at endoscopy in 21% of patients at 24 months postoperatively after an overnight fast. The incidence of food retention was higher after B-I than after B-II reconstruction. Kubo and colleagues[48] reported a similar incidence of food retention after distal subtotal gastrectomy for gastric cancer and found that food retention was more frequent after B-I than after Roux-Y reconstruction. However, none of the patients in either of these studies had clinically significant delayed gastric emptying, and no association was found between food retention and symptoms or body weight change. The medical management and strategies to restore gastric electrical stimulation to treat delayed gastric emptying are thoroughly covered elsewhere in this issue and are not included in this section. When delayed gastric emptying symptoms affect a patient's quality of life and medical management or pacing fails, reducing or eliminating the remnant stomach is the only option. Speicher and colleagues[49] reported a series of 44 patients over a 20-year period who required completion gastrectomy for chronic gastric atony after prior gastric surgery. The indication for the initial gastric operation was peptic ulcer disease in 75%, morbid obesity in 11%, gastroesophageal reflux disease in 9%, and bile reflux in 5%.

ROUX STASIS

Roux stasis, or Roux limb syndrome, is characterized by abdominal pain, nausea, vomiting, and postprandial bloating. Although some investigators report up to a 30% incidence, others have not found this syndrome to be nearly as clinically relevant.[50]

Although debating this entity may be academic, the physiologic changes possibly causing it are worthy of discussion.

Pacesetter potentials are cyclical electrical changes in the small bowel.[51] These potentials are fastest in the duodenal pacemaker and spread distally to the terminal ileum, generating action potentials that induce muscle contraction along the way. When the jejunum is transected, the frequency of pacesetter potentials in the distal bowel decreases and ectopic pacemakers appear that drive the potentials retrograde, toward the stomach.[52] Canine studies have revealed reduced transit of liquids through the segment of bowel with orally moving pacesetter potentials, and provide a physiologic explanation for the Roux stasis syndrome.[52] However, correlation of symptoms in human subjects has not been so definitive. Miedema and colleagues[53] found that although transit was uniformly slowed in the Roux limb, there was not a transit difference between asymptomatic patients and those with stasis symptoms.

Bile Reflux Gastritis

This entity seems to be less common now than it was in the past. The most likely explanation for this situation is that Roux-Y reconstruction after distal subtotal gastrectomy for cancer has been preferred by most surgeons in the United States for many years, and cancer resections have become the most common indication for gastrectomy with the precipitous decline in surgery for peptic ulcer disease. A previously cited study comparing B-I and Roux-Y reconstructions 5 years after distal subtotal gastrectomy for cancer showed a significantly lower incidence of gastritis on endoscopy and fewer symptoms of epigastric discomfort after Roux-Y compared with B-I.[30] A similar type of comparative retrospective study of Roux-Y, B-I, and B-II anatomy using a quality-of-life survey and quantitative measurement of bile reflux using the Bilitec (Synectics Medical AB, Stockholm, Sweden) probe showed significantly fewer symptoms and less bile reflux in the Roux-Y group.[54] Better outcomes with Roux-Y than with B-II were also seen in a study by Kronert and colleagues[55] using the Bilitec probe and a symptom survey in a group of patients after partial gastrectomy for benign peptic ulcer disease. Another recent study compared Roux versus B-I versus B-II patients 14 days after surgery for gastric cancer with the Bilitec probe and found bile reflux to be significantly less with Roux-Y reconstruction (B-II reconstruction had the highest incidence of bile reflux, noted in 70% of patients).[56] Endoscopy 3 months postoperatively showed significantly less reflux gastritis in Roux-Y patients, although symptom surveys were not significantly different between the 3 groups. Remnant gastritis was not affected by *Helicobacter pylori* status in this study.

Csendes and colleagues[57] reported the status after an average of 15.5 years of 75 patients randomized to Roux-Y versus B-II reconstruction after vagotomy and distal gastrectomy for duodenal ulcer disease. Roux-Y patients had fewer symptoms, better Visick scores, and an absence of chronic gastritis on endoscopy (vs an 80% incidence of chronic gastritis in the B-II group), despite the fact that *H pylori* was present in a similar proportion of patients. In a study (nonrandomized) by Fukuhara and colleagues[58] comparing Roux-Y versus B-I versus B-II patients 3 months after distal gastrectomy for cancer, interleukin 8 levels in gastric mucosa (on endoscopic biopsy specimens) were significantly lower in Roux patients both in the absence and the presence of active *H pylori* infection. They concluded that Roux Y reconstruction is better able to prevent remnant gastritis.

Although bile reflux and gastritis are more common after B-I and B-II reconstruction than after Roux-Y, debilitating symptoms are infrequent. For the few patients with severe bile reflux gastritis after partial or subtotal distal gastrectomy with B-I or B-II reconstruction, the best solution is to reoperate and convert to Roux-Y anatomy.

Reflux Esophagitis

Available evidence suggests that Roux-Y reconstruction results in a lower incidence of reflux esophagitis compared with B-I or B-II reconstruction. One study relates this to the preservation of a narrow angle of His with Roux-Y.[59] Nunobe and colleagues[30] showed a significantly lower incidence of heartburn symptoms and grade B or worse esophagitis in Roux-Y patients than in B-I patients after distal subtotal gastrectomy for early gastric cancer. Csendes and colleagues[57] found that Roux-Y patients had significantly less esophagitis on endoscopy and less Barrett metaplasia, compared with B-II patients, in a study of 75 patients randomized to Roux-Y versus B-I reconstruction after vagotomy and distal gastrectomy for duodenal ulcer disease studied 15.5 years after operation.

Cholelithiasis, Biliary Colic, and Cholecystitis

In a series of 463 patients with a normal gallbladder who underwent gastrectomy for gastric cancer and survived the operation, 85 of the 281 patients who underwent radical gastrectomy and 9 of the 182 patients who underwent simple gastrectomy developed gallstones ($P<.001$).[60] The mean interval between gastrectomy and gallstone formation was shorter in the radical gastrectomy patients (31.4 ± 20.9 months) than in the simple gastrectomy patients (48.0 ± 12.8 months) ($P<.05$). The investigators recommend prophylactic cholecystectomy on patients with a normal gallbladder having radical gastrectomy. Nunobe and colleagues[30] found a significantly higher incidence of postoperative gallstone formation in the Roux-Y group (28%) than in the B-I group (15%) 5 years after distal subtotal gastrectomy for early gastric cancer. The increased risk of gallstone formation is related to loss of vagal innervation of the gallbladder with decreased gallbladder motility after radical gastrectomy, and a result of loss of cholecystokinin release from the duodenum with bypass of the duodenum when Roux-Y reconstruction is performed.

Recurrent/Anastomotic Ulceration

Recurrent ulceration after peptic ulcer surgery most commonly results from incomplete vagotomy and nonsteroidal antiinflammatory drug use.[61] The most common sites for recurrence include peripyloric, duodenal, and peristomal areas. The role of *H pylori* is less clear than in the cases of initial peptic ulcer.[62] Treatment consists of proton pump inhibitors and discontinuing ulcerogenic medications. When this treatment fails, and incomplete vagotomy is diagnosed, transthoracic truncal vagotomy is recommended.[63]

Less common causes include hypersecretory states (Zollinger-Ellison syndrome) and retained antrum. In cases of retained antrum, there is residual antral tissue within the duodenal stump after gastric resection with B-II anastomosis. The G cells are continuously bathed in alkaline duodenal fluid, resulting in continuous secretion of gastrin, causing sustained intense stimulation of acid production by parietal cells in the proximal stomach remnant. The exposure of the unprotected jejunum to this high acid level results in what is termed a marginal ulcer (**Fig. 1**). Diagnosis can be made with a sodium 99m technetium scan, and reexcision is curative.

Gastric Remnant Carcinoma

The risk of cancer in the stomach remnant after partial gastrectomy has been debated for many years.[64–66] However, early studies suffered from a heterogeneous patient mix, including patients with peptic ulcer disease and gastric cancer, and from not ensuring patients with gastric ulcers did not have a missed gastric cancer. More

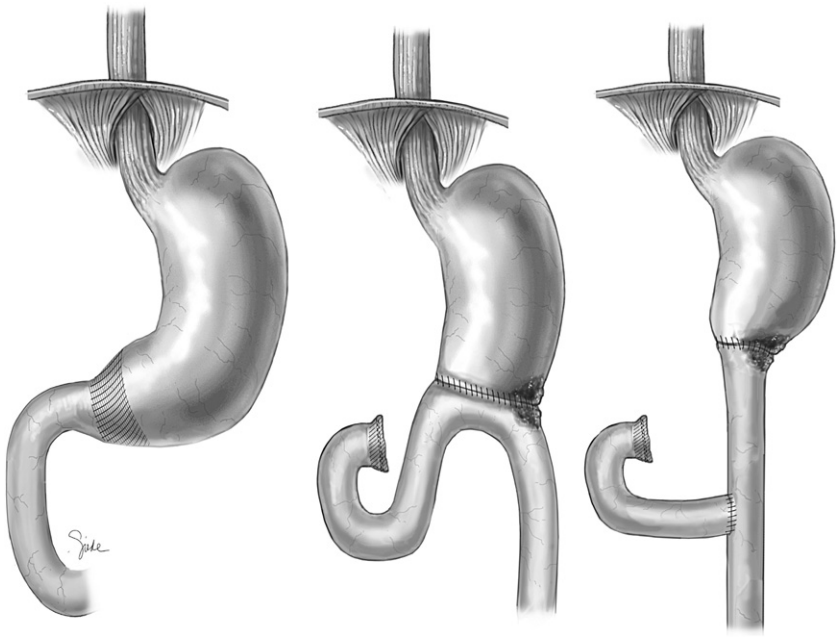

Fig. 1. Pathophysiology of retained antrum syndrome: incompletely resected antrum during distal gastrectomy with B-II or Roux-Y reconstruction results in intense gastrin secretion by the remaining antrum, which is constantly bathed in the alkaline environment of the adjoined duodenum.

recently, larger studies, limited to patients with clearly benign disease, incorporating selection criteria to minimize pathologic error, have been reported, although without absolute consensus. In a review of 1000 patients who had gastric resection with B-II reconstruction for duodenal ulcer, Fischer and colleagues[67] noted only 13 cases of remnant carcinoma, not different from the general population using the life table method. Viste and colleagues[68] noted remnant carcinoma in 87 of 3470 (2.5%) postgastrectomy patients with initially benign disease, with an observed/expected ratio of 2.1 ($P<.001$). Risk was not increased in the first 5 to 10 years after surgery, but after 40 to 45 years it was 7.3-fold higher than the expected value for the population. In 1988, 2 published cancer registry-based studies only added to the controversy, with differing conclusions.[69,70] As had been noted by previous investigators, the Swedish study[70] revealed an increased remnant cancer risk in patients with a B-II reconstruction compared with those with a B-I and in patients undergoing gastrectomy for gastric ulcer compared with those with other benign diseases. In 1993, a Veterans Affairs study[71] compared more than 7000 patients undergoing gastric resection for a benign disorder with a matched control group and noted an increased risk of remnant cancer (standardized rate ratio 1.9, confidence interval 1.3–2.4), especially in those with gastric ulcer.

Although absolute consensus may be lacking, physiologic changes related to gastrectomy could produce a procarcinogenic state. Miwa and colleagues[72] found duodenal reflux into the rat stomach induced adenocarcinoma. Ruddell and colleagues[73] reported an increase in gastric juice nitrite concentrations in hypochlorhydric subjects, and it is known that hypochlorhydria allows increased luminal

bacteria that are able to convert nitrates to nitrites.[74] Nitrites are precursors of the known carcinogenic *N*-nitroso compounds, more commonly noted in the remnant after B-II reconstruction.[75] Furthermore, secondary bile acids, known to promote gastritis and metaplasia, constantly bathe the gastric stump anastomosis after B-II and may explain a recent report that remnant carcinomas after B-II are more likely at the intestinal anastomosis, whereas after B-I they are noted equally throughout the stomach.[76]

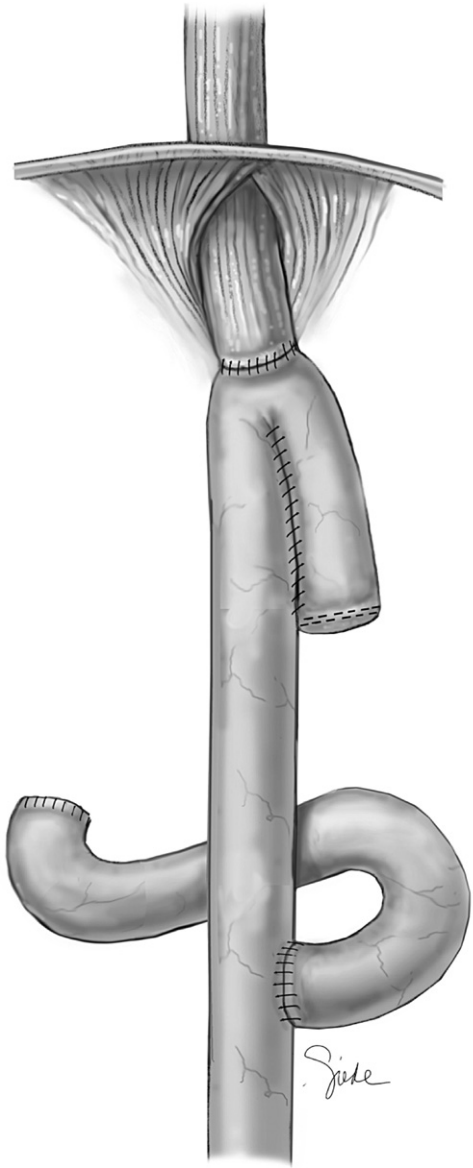

Fig. 2. Proximal pouch Roux-Y reconstruction.

Although many papers have been published on the risk of remnant carcinoma, perspective is needed. Caygill and colleagues[77] reviewed outcomes on more than 5000 patients undergoing gastric resection. Whereas 37 deaths resulted from remnant gastric cancer in patients living longer than 20 years, there were 991 deaths from lung cancer and nonneoplastic causes. With 80% of patients with peptic ulcers being smokers,[78] efforts at lifestyle modification rather than endoscopic screening of the gastric remnant may be more fruitful in improving the long-term outcome of the post-gastrectomy patient.

Does the Quality of Life After Gastrectomy Depend on the Type of Reconstruction?

A nonrandomized retrospective comparison of B-I, B-II, and Roux-Y 3 years after partial gastrectomy, including multiple end points (Visick grading, dumping score, afferent and efferent loop syndromes, bile reflux, regurgitation, overall quality of life) found significantly better outcomes with Roux-Y reconstruction.[79] A second retrospective comparison of B-I and Roux-Y reconstruction 5 years after distal

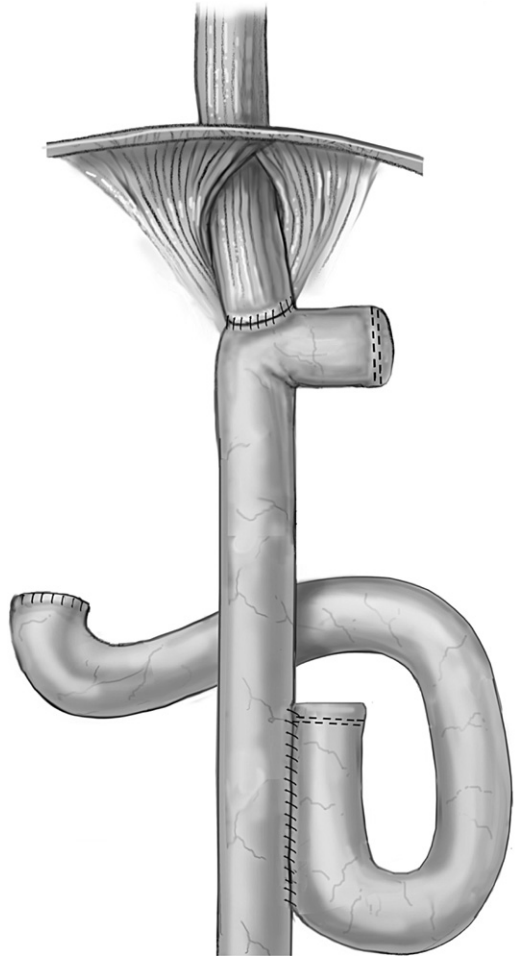

Fig. 3. Distal pouch Roux-Y reconstruction.

gastrectomy for early gastric cancer found that Roux-Y reconstruction was superior; however, the incidence of gallstone development was higher after Roux-Y.[30] In a randomized comparison of B-II and Roux-Y reconstruction after vagotomy and distal gastrectomy for duodenal ulcer, Roux-Y patients had significantly fewer postoperative symptoms and better Visick grading than did B-II patients ($P<.001$).[57] However, another small randomized comparison of Roux-Y versus B-I reconstruction after distal gastrectomy for gastric cancer found a significantly longer hospital stay in the Roux-Y group ($P<.05$) as a result of gastrojejunal stasis and no significant difference in postoperative nutritional status.[80]

After Total Gastrectomy, Does a Pouch Matter?

Construction of a jejunal pouch as a substitute for the stomach after total gastrectomy was first proposed by Hunt in 1952.[81] The technique has not been widely accepted, but multiple RCTs over the past 15 years have, for the most part, supported the use of a pouch.[13,82–91] These RCTs have been small in size and have used different techniques for pouch construction. The differences between the pouch and nonpouch groups have been modest. Examples of several techniques of pouch reconstruction are provided in **Figs. 2–4**. A recent meta-analysis encountered several methodologic difficulties but concluded that "a pouch reconstruction after total gastrectomy is clearly beneficial for patients with expected long-term survival."[92,93] The benefit was

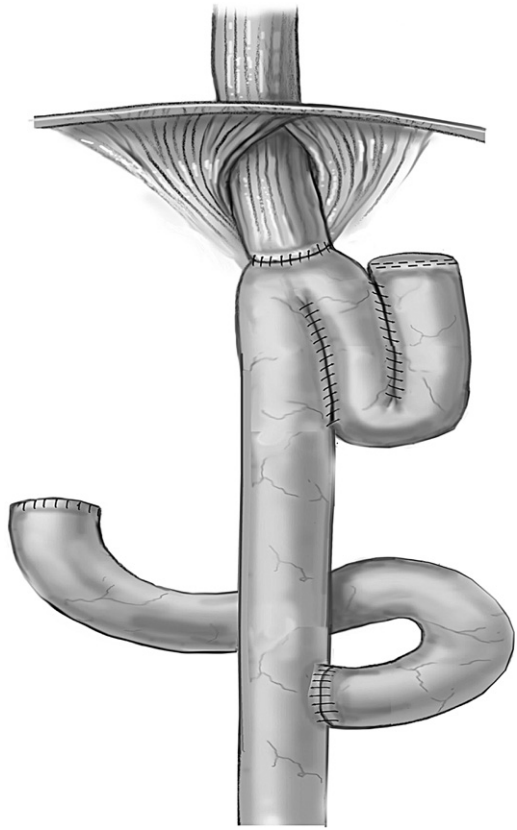

Fig. 4. S pouch Roux-Y reconstruction creating a larger reservoir.

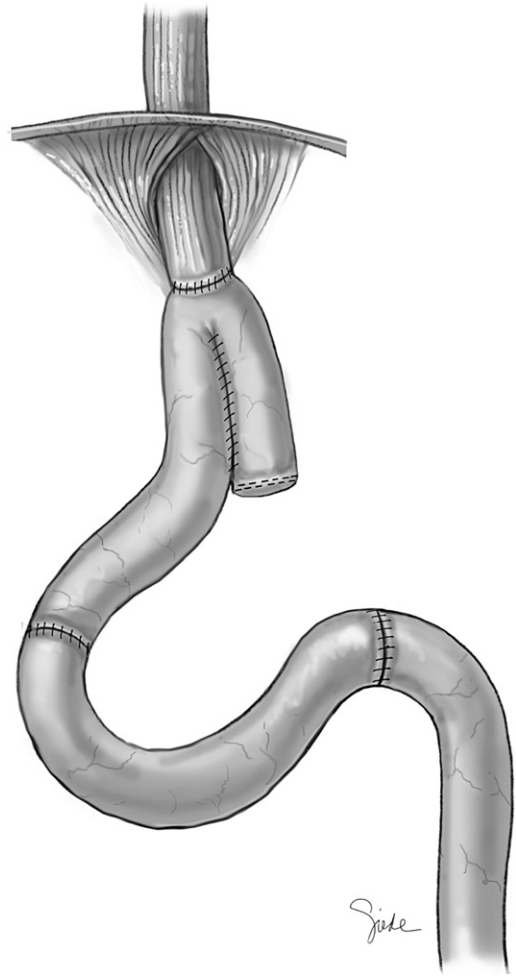

Fig. 5. Pouch interposition: note that duodenal continuity with the food stream is maintained.

most clearly evident in the lower incidence of dumping and heartburn reported by patients and higher reported food intake postoperatively. In particular, the Gastrointestinal Quality of Life Index scores for pouch patients were significantly better than for nonpouch patients.[90] Objective evidence of improved body weight was not reported in this meta-analysis. The findings apply only to Roux-Y pouch formation after total gastrectomy. A few small RCTs comparing pouch interposition reconstruction (maintaining duodenal passage of the food stream, **Fig. 5**) with Roux-Y pouch reconstruction have reported variable results; overall, the studies are not robust enough to permit meta-analysis.[85,88,94,95]

SUMMARY

The frequency with which gastric resection is performed in the United States has decreased significantly over the past 4 decades. As a direct result of this situation,

the number of patients with severe postgastrectomy syndromes is probably decreasing. Gastric cancer resection is the most frequent indication for gastrectomy in the United States and worldwide. As long-term survival rates for patients who have resected gastric cancer improve, long-term quality of life is increasingly important; this is already true in East Asia, where 60% of gastric cancers are early and innovative new techniques are being used to avoid or minimize postgastrectomy symptoms. Most patients who have gastric cancer maintain healthy body weight and lean body mass postoperatively and have satisfactory gastrointestinal quality of life after gastric resection, but a small number (<5%) have persistent debilitating symptoms of a postgastrectomy syndrome. In this article, the major postgastrectomy syndromes are presented and their prevention and treatment discussed. After total gastrectomy for cancer, Roux-Y reconstruction is preferred. There is no evidence that the added operative time required to recreate the reservoir function of the stomach by pouch reconstruction is warranted. The incidence of gallstones after gastrectomy is significant.

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