# Experimental and Clinical Results with Proximal End-to-End Duodenojejunostomy for Pathologic Duodenogastric Reflux

TOM R. DEMEESTER, M.D., KARL H. FUCHS, M.D., CHRIS S. BALL, M.D., MARIO ALBERTUCCI, M.D., TOM C. SMYRK, M.D.,* and JOSEPH N. MARCUS, M.D.*

*From the Departments of Surgery and Pathology,* Creighton University, School of Medicine, Omaha, Nebraska*

**Existing Roux-en-Y bile diversion procedures for duodenogastric reflux coupled with distal gastric resection or antrectomy and vagotomy have varied success due to interruption of the physiologic relationships between stomach and duodenum, the reduction of the gastric reservoir, the side effects of vagotomy, and the effect of the Roux limb on gastric emptying. A new bile diversion procedure, suprapapillary Roux-en-Y duodenojejunostomy, was studied, which eliminates the need for gastric resection to prevent jejunal ulcers by preserving duodenal inhibition of gastric acid secretion and the protective effects of duodenal secretion on the surrounding mucosa. Experimentally, the incidence of jejunal ulceration was significantly decreased by the preservation of the proximal duodenum. Clinically, bile diversion by suprapapillary Roux-en-Y duodenojejunostomy alleviates symptoms of duodenogastric reflux disease without being ulcerogenic (in the presence of normal gastric secretion) or prolonging gastric emptying.**

ALKALINE REFLUX GASTRITIS is a recognized complication after operations on the stomach that distort or remove the pylorus.[1-3] It may also occur as a consequence of altered pyloric or duodenal function in patients who have not had gastric surgery.[4-6] There is evidence that the regurgitation of duodenal juice into the stomach is associated with gastric mucosal damage[7-9] and can cause symptoms of epigastric pain, nausea, and bilious vomiting.[6,10-12] Conservative therapy of the condition has been largely ineffective,[13] and in its severe form a Roux-en-Y biliary diversion procedure or a jejunal interposition is usually necessary for relief of symptoms.[2] When the former is used in patients without previous gastric resection, an antrectomy and vagotomy are required to protect against the development of jejunal ulcers. This interrupts the physiologic relationship between the stomach and duodenum, reduces the gastric reservoir, and gives rise to side effects associated with a vagotomy.[14] In addition, the Roux-en-Y limb can severely delay gastric emptying.[15,16] For these reasons, the success of a Roux-en-Y biliary diversion in improving symptoms has been only 50–80% and the incidence of jejunal ulceration has been 10–15%.[12,17-19] Consequently, a new surgical approach is needed for patients with pathologic duodenogastric reflux, especially those who have not had previous gastric surgery or whose previous surgery did not include a resection. The current study reports on the experimental development and the clinical evaluation of a new biliary diversion procedure, namely a suprapapillary Roux-en-Y duodenojejunostomy succinctly called a "duodenal switch."

## Experimental Study

The purpose of this study was to determine if duodenal inhibition of gastric acid secretion and the protective effect of duodenal secretion on the surrounding mucosa could be maintained in a bile diversion procedure that preserved only the proximal duodenum in continuity with the stomach. To do so, an attempt was made to reduce the 95% incidence of jejunal ulcer formation after the Mann-Williamson preparation[20] by performing a suprapapillary duodenojejunostomy instead of a pylorojejunostomy.

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Reprint requests and correspondence: Tom R. DeMeester, M.D., Department of Surgery, Creighton University, School of Medicine, 601 North 30th Street, Omaha, NE 68131.

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## Method

Under general anesthesia, 29 adult mongrel dogs were subjected to one of four different arrangements of Roux-en-Y biliary diversion (Fig. 1). Seven dogs had an end-to-end pylorojejunostomy with an end-to-side jejunoileostomy 10 cm proximal to the ileocecal valve (classic Mann-Williamson preparation: group A). Five dogs had an end-to-end pylorojejunostomy with an end-to-side jejunojejunostomy 40 cm down the jejunum (group B). Seven dogs had an end-to-end suprapapillary duodenojejunostomy with an end-to-side jejunoileostomy 10 cm proximal to the ileocecal valve (group C). Ten dogs had an end-to-end suprapapillary duodenojejunostomy with an end-to-side jejunojejunostomy 40 cm down the jejunum (duodenal switch procedure: group D). All dogs were given the same diet and were weighed and had endoscopy at monthly intervals for up to 1 year or until death. All dogs were autopsied with inspection and removal of both surgical anastomoses. Statistical differences in the incidence of ulcer formation and degree of weight loss were determined using the Student's t-test.

## Results

The incidence of ulcer formation, ulcer perforation, and degree of weight loss is shown in Figure 1. Dogs with jejunoileostomy showed profound malnutrition. When the proximal duodenum was preserved, the incidence of ulceration was dramatically reduced. The results showed that the end-to-side jejunojejunostomy was necessary to maintain nutrition, and preservation of the proximal duodenum in the acid stream was necessary to protect against ulceration. Dogs without both of these components died of either malnutrition or ulceration within 3 months.

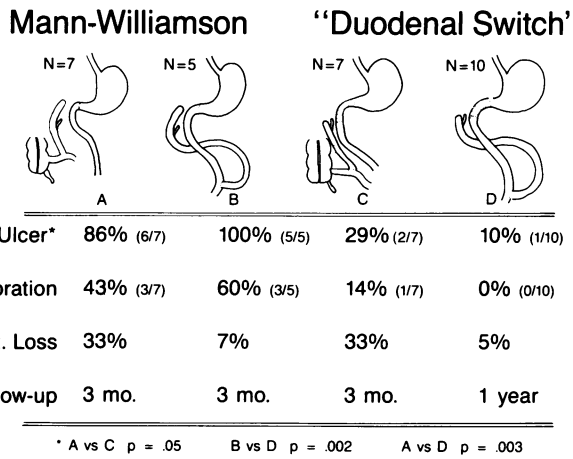


FIG. 1. Results of four bile diversion procedures with their effect on ulcer incidence, perforation, and weight loss.

## Clinical Study

The purpose of this study was to determine if biliary diversion by a suprapapillary Roux-en-Y duodenojejunostomy for pathologic duodenogastric reflux can: (1) provide symptomatic improvement; (2) heal endoscopic gastritis; (3) preserve normal gastric emptying; and (4) prevent jejunal ulceration wihtout vagotomy in patients with normal acid secretion.

## Method

### *Patient Selection*

Between January 1984 and January 1987, 202 patients were referred to the Creighton University Surgical Gastrointestinal Diagnostic and Research Unit for evaluation of foregut symptoms. Of these patients, 10 with epigastric pain, nausea, bilious vomiting, and endoscopic gastritis unresponsive to medical therapy for 2–20 years were selected for bile diversion. There were four men and six women with an age range of 28–57 years. Five patients had primary duodenogastric reflux and duodenogastric reflux developed in five patients after previous gastrointestinal surgery (Table 1). Of the three presenting complaints, epigastric pain was dominant in six of ten patients. In four patients it became worse with eating. The pain was described as burning by five patients, dull by three patients, and sharp by two patients. In six patients the pain radiated: to the chest in three, to the back in two, and to the lower abdomen in one. Nausea was the dominant symptom in two of ten patients. Nausea was associated with epigastric pain in four patients and the intake of food in two patients. Vomiting was the dominant symptom in two of ten patients, and was bilious in color in nine patients; one patient, who had a previous Nissen fundoplication, only retched. Vomiting produced relief of the epigastric pain in three patients.

TABLE 1. *Post-surgical Duodenogastric Reflux*

| Sex/Age (years) | Previous Surgery |
|---|---|
| M/57 | Nissen fundoplication<br>Redo Nissen fundoplication |
| M/34 | Esophageal myotomy<br>Redo esophageal myotomy and Belsey<br>Esophagectomy, colon interposition, pyloroplasty |
| M/39 | Truncal vagotomy and pyloroplasty<br>Esophageal myotomy and cardioplasty |
| M/28 | Esophageal myotomy and Belsey<br>Pyloroplasty |
| F/53 | Nissen fundoplication<br>Pyloroplasty |

TABLE 2. *Objective Evidence of Duodenogastric Reflux*

| | Patient No. | DISIDA Scan | Alkaline pH Shift | Discriminant pH Score |
|---|---|---|---|---|
| Primary group | 1 | − | + | + |
| | 2 | +* | + | + |
| | 3 | +* | − | − |
| | 4 | − | − | + |
| | 5 | − | + | + |
| Postsurgical group | 1 | − | + | − |
| | 2 | +* | + | + |
| | 3 | + | + | + |
| | 4 | +* | + | + |
| | 5 | + | + | + |

* After administration of cholecystokinin.

In each patient the symptomatic and endoscopic evidence of duodenogastric reflux was supported by one or more of three objective tests: DISIDA cholescintigraphy, alkaline pH shift on 24-hour gastric monitoring, and discriminant analysis of 24-hour gastric pH data (Table 2). Twenty healthy volunteers who were asymptomatic for gastrointestinal disease and had normal upper gastrointestinal barium studies provided a control group for the normal parameters of these tests.

*DISIDA cholescintigraphy.* Five μCi of Tc-99m labeled disofenin was given by intravenous injection in the fasting state. Gamma camera imaging commenced immediately and at 5-minute intervals for 60 minutes. The images acquired were stored by computer. If no duodenogastric reflux was observed during the first 60 minutes, imaging was continued for an additional 60 minutes, after which cholecystokinin was given intravenously in a dose of 0.02 μg/kg body weight over 1 minute in an attempt to provoke reflux. The timing and degree of spontaneous or provoked reflux of isotope into the stomach was noted along with abnormalities of the biliary tract and the symptoms experienced by the patient after injection of cholecystokinin. When performed on 20 control subjects, four (20%) showed evidence of duodenogastric reflux. In three controls, reflux was spontaneous and in one control it was provoked by cholecystokinin.

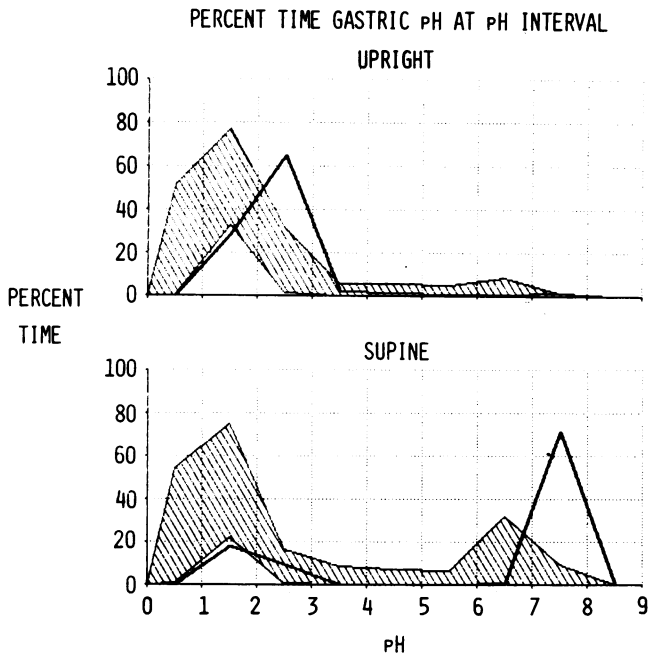


FIG. 2. Graphic display of percentage of time the gastric pH was at a whole pH interval. Control 95th and 5th percentile ranges are the shaded area. Patient data are represented by the solid line.

*Twenty-four-hour pH monitoring.* Outpatient monitoring of esophageal and gastric pH was performed using a computerized portable recording unit connected to combined glass probes, positioned transnasally 5 cm above and below the manometrically defined distal esophageal sphincter. The probes were calibrated in standard buffer solution at pH 7 and pH 1 before and after monitoring. The patient or volunteers were sent home and instructed to remain in the upright or sitting position until they retired for the evening, avoid strenuous exertion, and follow a diet restricted to three meals composed of food with a pH between 5 and 7. Only water was permitted between meals. A diary was kept of food and fluid intake, symptoms experienced during the monitored period, the time a supine position was assumed in preparation for sleep, and the time of rising in the morning. The patient's gastric pH data were displayed graphically as the percentage of time the pH was at a whole pH interval against a background of control data to detect an alkaline pH shift (Fig. 2). The pH data were further evaluated by a computerized discriminant analysis program developed in our unit to identify pH changes with a high probability of resulting from pathologic duodenogastric reflux. Comparison of patients with control subjects for this analysis is shown in Fig. 3.

## *Operative Procedure*

Through an upper midline incision, the duodenum is kocherized sufficiently to allow it and the head of the pancreas to be swung freely for exposure of their posterior surfaces, and the position of the ampulla is palpated through the duodenal wall. With the thumb and index finger encircling the duodenum, it is possible to feel their close approximation at a point along the medial duodenal border just proximal to the site of the ampulla and about 2–3 cm distal to the pylorus. At this location the duodenum is dissected free of the pancreatic head by dividing the small vessels coursing between the two. This dissection is done alternately on the anterior and posterior surface taking small bites of tissue with delicate

clamps. Care must be taken to avoid damage to the pancreatic portion of the common bile duct. It lies embedded just beneath the posterior surface of the pancreatic head and is identified by its linear appearance compared with the more globular adjacent pancreatic tissue. The dissection is continued until the index finger can be freely passed between the duodenum and pancreas.

At the distal end of the dissection the duodenum is divided using a GIA stapler. A pursestring suture is placed around the lateral corner of the stapled distal duodenal closure and the corner is opened. The flow of bile from this opening on compression of the liver confirms the suprapapillary transection of the duodenum. The pursestring is tied and the distal duodenal closure is oversewn (Fig. 4).

The jejunum, 25 cm distal to the ligament of Treitz, is brought through an opening in the transverse mesocolon to the right of the midcolic vessels. Its mesenteric border is freed for a distance of 2 cm and the bowel is divided between clamps (Fig. 5). Further division of the mesentery is avoided to preserve innervation.

The distal jejunal limb is anastomosed end-to-end to the proximal duodenum using an interrupted single-layer suturing technique (Fig. 6), and the proximal jejunal limb is withdrawn back through the opening in the mesocolon. This maneuver can be facilitated by initially applying the proximal jejunal clamp through the mesenteric opening before dividing the bowel. The proximal jejunal limb is anastomosed end-to-side to the distal jejunal limb 55 cm caudal to the duodenojejunal anastomosis (Fig. 7). The opening in the mesocolon is closed by suturing the margins to the circumference of the duodenojejunal anastomosis. The complete "duodenal switch" is shown in Figure 8.

This procedure was performed on the 10 patients in the study. In three patients, one of whom had a previously failed antireflux procedure, a Nissen fundoplication was added for coexisting gastroesophageal reflux disease. One patient who had gastric hypersecretion had a concomitant limited proximal gastric vegotomy to reduce acid secretion. One patient had a cholecystectomy added for cholelithiasis. Two patients had a temporary rise in their serum amylase level during the immediate postoperative recovery period. The average length of hospital stay was 13 days with a range of 9–31 days.

*Evaluation of the Operation*

All patients had a preoperative and postoperative scoring of their symptoms and endoscopic evaluation of their stomach and duodenum. With few exceptions all had gastric biopsy, gastric emptying scan, gastric secretion analysis, and a serum gastrin assay before and after bile diversion. The postoperative evaluation was performed 10–33 months (median: 24 months) after operation. Student's t-test was used to determine statistical significance between preoperative and postoperative data when applicable.

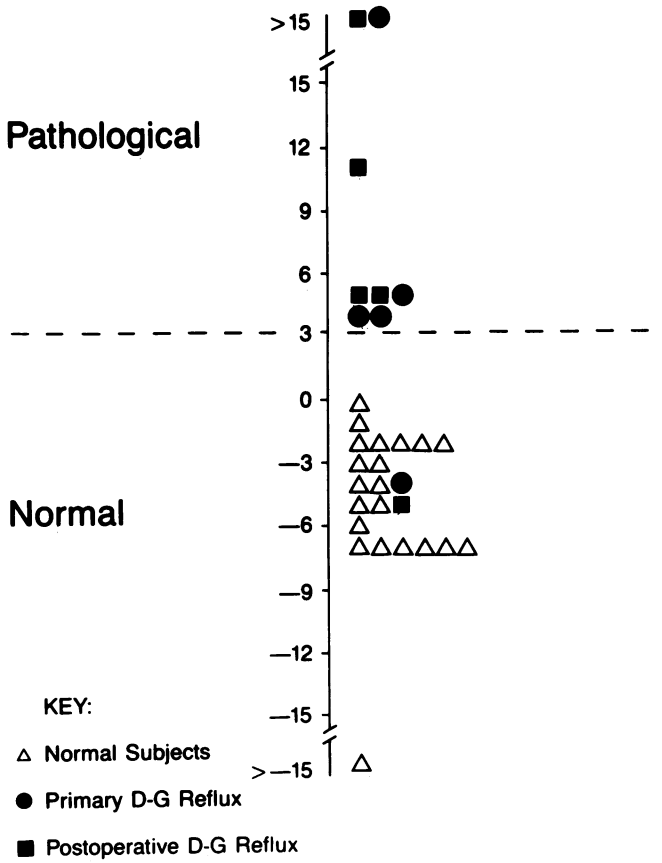


FIG. 3. Discriminant analysis of gastric pH data in duodenogastric reflux patients and controls.

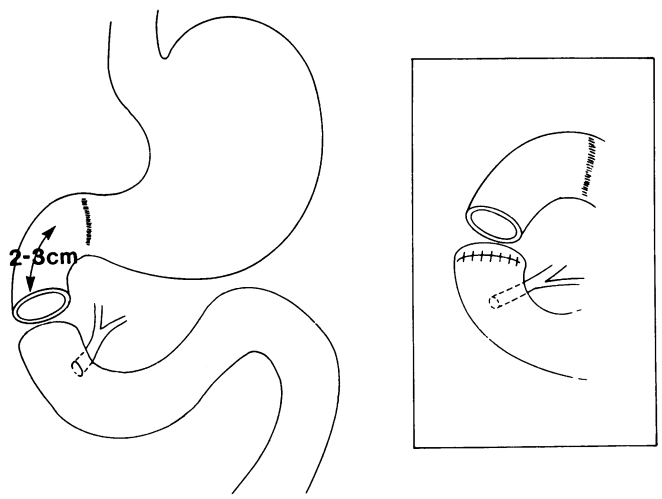


FIG. 4. Schematic diagram showing the suprapapillary transection of the duodenum 2–3 cm distal to the pylorus. Insert shows closure of the distal duodenum.

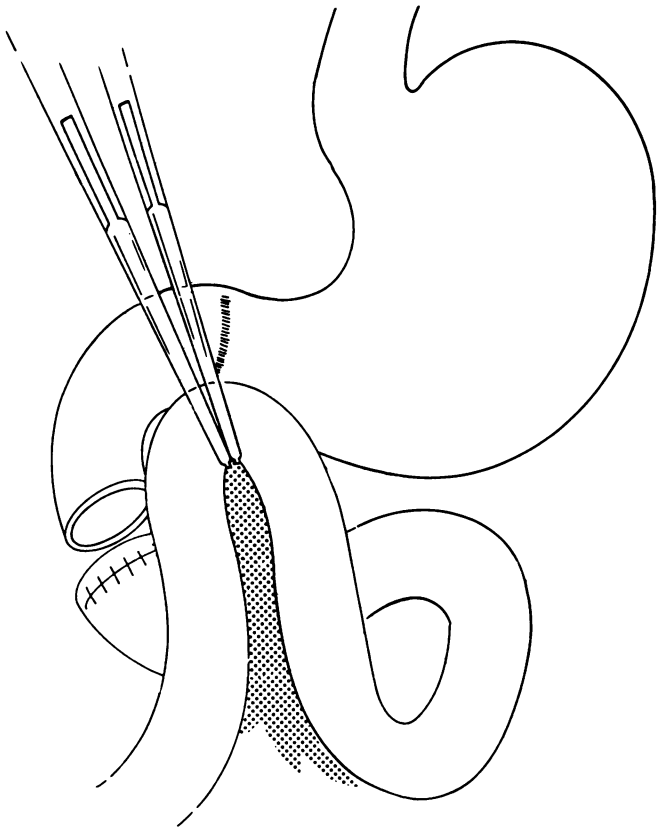

FIG. 5. Schematic diagram showing the initial construction of the Roux-en-Y in juxtaposition to the duodenum. This position is obtained by passing the loop through an opening in the mesocolon and allows construction of the Roux-en-Y without division of the mesentery.

*Symptom scoring.* Symptoms were scored using a detailed questionnaire completed by one of the authors. A scoring system shown in Table 3 was used to quantitate the symptoms of epigastric pain, nausea, and bilious vomiting. An overall symptom score was obtained by summing the scores for each symptom. The maximum score obtainable was 18.

*Gastric biopsy.* Multiple endoscopic biopsies were taken from the gastric antrum and body, fixed in formalin, and stained with hematoxylin and eosin. A scoring system was used to evaluate severity of gastritis.[21] A score of 0 to 4 was given for inflammation, 0 to 4 for corkscrew appearance of gastric tubules, and 0 to 4 for surface hyperplasia. The maximum score obtainable was 12.

*Gastric emptying scan.* A meal was given in the fasting state consisting of 56 g of instant oatmeal, 6 g of sugar, and 175 mL of water, mixed with 500 μCi Tc-99m sulfur colloid and followed by 240 mL of 2% milk. Caloric values of the test meal are shown in Table 4. Gamma camera counts were taken in the upright anterior and posterior projections for 40 seconds at the completion of the meal and every 15 minutes for 120 minutes. Counts per minute were adjusted to account for the decay of Tc-99m. The percentage of the initial count was plotted against time for the anterior projection, posterior projection, one-half times the sum of both projections and for a calculated geometric mean. The curve was displayed against the 95 and 5 percentile limit obtained from the 20 control subjects. In the early part of the study only the anterior projection was obtained.

*Gastric analysis and serum gastrin levels.* Gastric juice was aspirated using a Salem sump nasogastric tube (Sherwood Medical, St. Louis, MO) after an overnight fast. The first 15-minute sample was saved as residual basal secretion, and the following 60-minute aspirate was considered a representative basal sample. Pentagastrin was given subcutaneously in a dose of 6 μg/kg body weight and stimulated samples were obtained continuously for 15- and 60-minute periods. Hydrogen ion concentration was measured by a colorimetric titration, and the basal acid output (BAO) and maximum acid

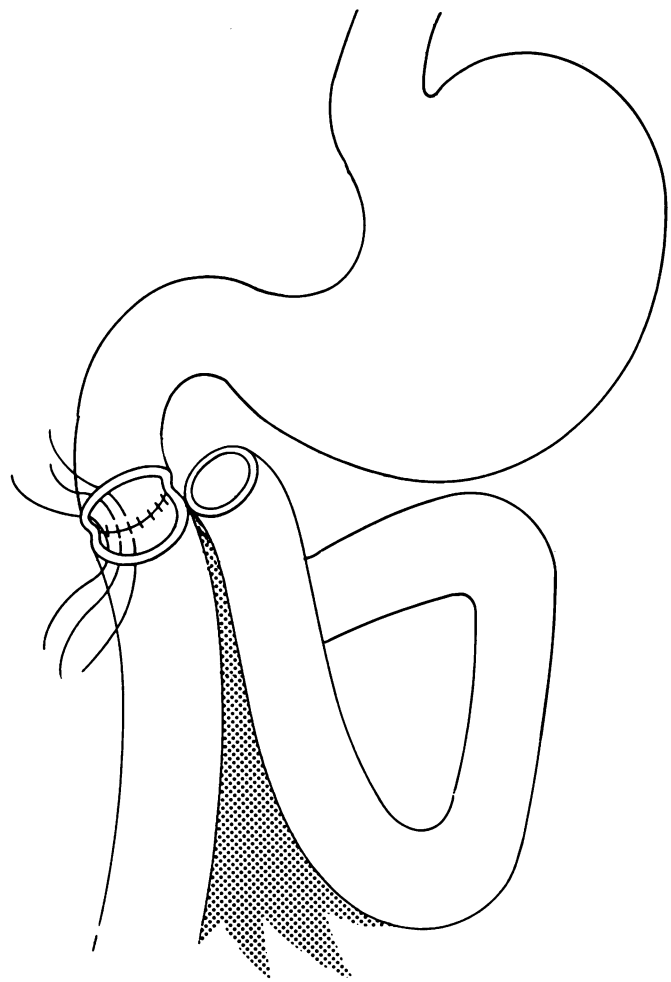

FIG. 6. Schematic diagram showing the end-to-end anastomosis of the distal jejunal limb to the duodenum.

output (MAO) in mEq/h were calculated. Hypersecretion was diagnosed when BAO was above 5 mEq/h and MAO was greater than 35 mEq/h. Fasting serum gastrin levels were measured by a radioimmune assay and recorded in picograms per milliliter.

## Results

### *Symptoms*

The preoperative and postoperative scoring of each symptom is shown in Figures 9 and 10. Bile diversion by a suprapapillary Roux-en-Y duodenojejunostomy statistically improved the overall symptom score for the five patients with primary duodenogastric reflux ($p < 0.05$), but no statistical improvement occurred in the five patients with postsurgical duodenogastric reflux. In the one patient who had persistent symptoms in the primary group, a postoperative jejunal ulcer developed that was healed completely by H2 antagonists in 6 weeks. This patient had preoperatively borderline gastric hypersecretion. In the postsurgical group, two patients had persistent symptoms. one patient had a previous colon interposition for severe reflux esophagitis after a Heller myotomy and Belsey antireflux procedure. The other patient had an esophageal myotomy and pyloroplasty for an esophageal motility disorder. Consequently, it was difficult to determine the cause of their continuing symptoms. When each patient was asked

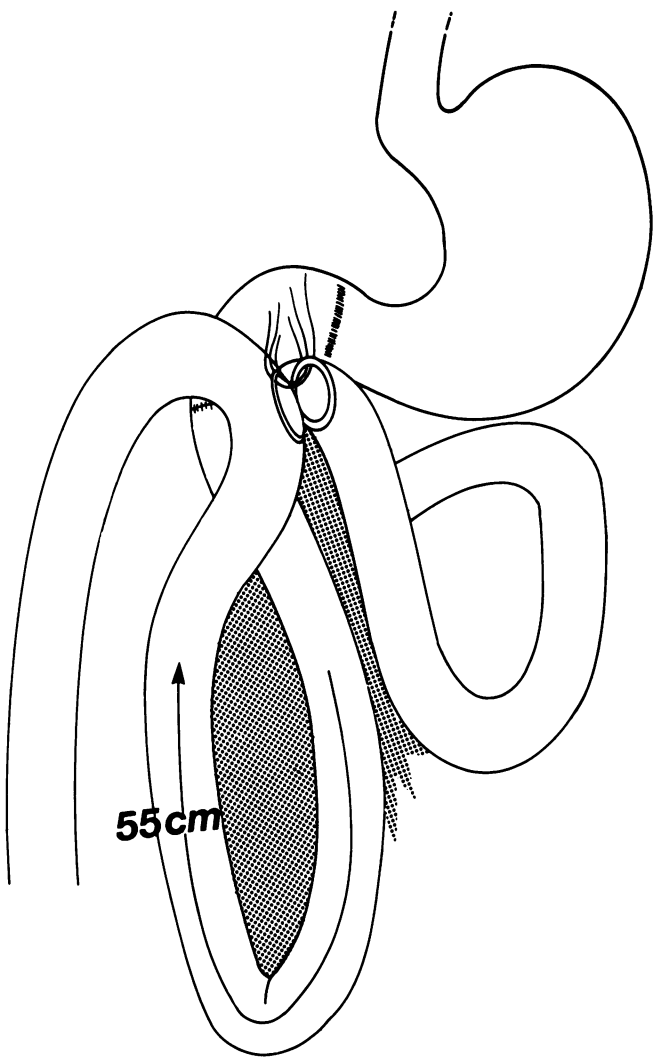


FIG. 7. Schematic diagram showing an end-to-side anastomosis of the proximal jejunal limb to the distal limb 55 cm caudal to the duodenojejunal anastomosis.

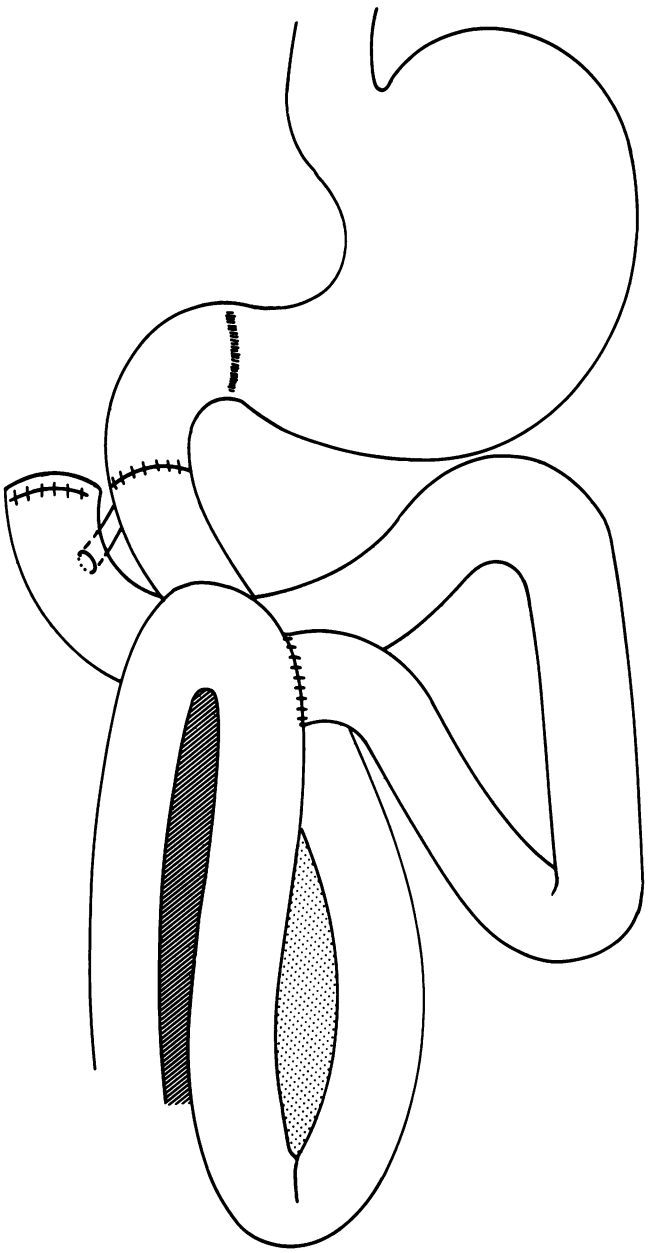

FIG. 8. Completed duodenal switch procedure used for diversion of the bile.

TABLE 3. *Scoring of Epigastric Pain, Nausea, and Biliary Vomiting*

| | Score |
|---|---|
| Frequency of symptom | |
| Daily | 4 |
| >1 episode per week | 3 |
| >1 episode per month | 2 |
| <1 episode per month | 1 |
| Absence of symptom | 0 |
| Relationship of symptom to alimentation | |
| Reduced appetite or discouraged eating | 1 |
| No effect on appetite or eating | 0 |
| Severity of symptom* | |
| Interferes with daily activity | 3 |
| Nonrestrictive but always apparent | 2 |
| Nonrestrictive but intermittently apparent | 1 |

* For epigastric pain only.

whether he or she would have the operation if faced with the choice again, nine of ten said yes.

### *Endoscopy*

Table 5 shows the preoperative and postoperative endoscopic findings. Before operation, all patients had endoscopic gastritis and no evidence of esophageal or duodenal mucosal abnormalities. After operation, the endoscopic gastritis healed in all but one patient who had atrophic gastritis associated with pernicious anemia. The duodenojejunal anastomosis was normal in all patients and no bile was seen in the stomach. One of the 10 patients had a jejunal ulcer that, as mentioned earlier, healed completely with H2 antagonists in 6 weeks.

### *Histology*

Gastric biopsy was done in six preoperative and ten postoperative patients. Histologic scores of the biopsies are shown in Table 6. Despite the improvement in endoscopic gastritis, the histologic changes persisted in five patients (score: >1), although they were only minor in three patients. The two patients with the score of seven had intestinal metaplasia that persisted over follow-up periods of 21 and 33 months. Overall, there was no statistical improvement in the postoperative histologic scores.

TABLE 4. *Caloric Values of Gastric Emptying Test Meal*

| | Oatmeal | Milk | Sugar | Total |
|---|---|---|---|---|
| Calories (kcal) | 200 | 12 | 60 | 380 |
| Protein (g) | 10 | 8 | | 18 |
| Carbohydrate (g) | 17 | 11 | | 28 |
| Fat (g) | 4 | 5 | | 9 |

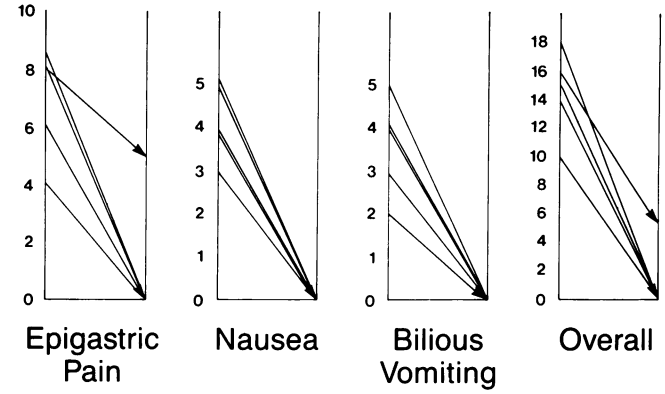


FIG. 9. Comparison of preoperative and postoperative symptoms in patients with primary duodenogastric reflux.

### *Gastric Emptying*

Preoperative and postoperative gastric emptying curves are shown in Figure 11. All patients with primary duodenogastric reflux had normal preoperative gastric emptying. Two patients with postsurgical duodenogastric reflux had preoperatively rapid gastric emptying and in one patient the half-time was less than 15 minutes. Both patients had a previous pyloroplasty and the patient with particularly rapid emptying had a previous truncal vagotomy. With one exception, postoperative curves for both groups fell within the normal range as defined from scans on healthy volunteers and demonstrates that suprapapillary Roux-en-Y duodenojejunostomy does not retard gastric emptying. The one exception is a patient whose postoperative emptying became slightly rapid. When the postoperative geometric mean curves from seven patients were analyzed, the finding was similar.

### *Gastric Analysis and Serum Gastrin Levels*

Table 7 shows preoperative and postoperative values for basal acid output, maximal acid output, and serum

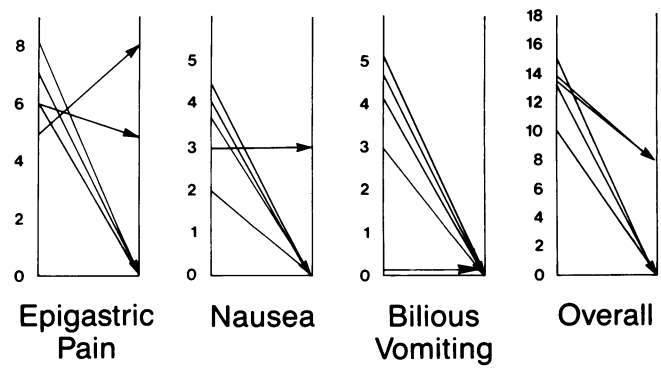


FIG. 10. Comparison of pre- and postoperative symptoms in patients with postsurgical duodenogastric reflux.

gastrin level. One patient (number 3 in the primary group) had hypersecretion before operation with elevated values for both basal and stimulated secretion. A limited proximal gastric vagotomy at the time of duodenal switch restored her acid output to within the normal range. A second patient (number 4 in the primary group) had acid secretion at the upper limit of normal range. After operation, her values crossed the upper limit and a jejunal ulcer developed that responded to H2 blockers.

Two patients with primary duodenogastric reflux had high preoperative serum gastrin levels, both of which remained elevated after operation. One patient had pernicious anemia and the other patient had normal gastric acid production and a normal secretin test.

TABLE 5. *Endoscopic Findings*

| Patient No. | Primary Group | |
|---|---|---|
| | Before Operation | After Operation |
| 1 | No esophagitis, diffuse atrophic gastritis | No esophagitis, diffuse atrophic gastritis, anastomosis healthy, no jejunal ulcer |
| 2 | No esophagitis, antral gastritis | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |
| 3 | No esophagitis, antral gastritis | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |
| 4 | No esophagitis, antral gastritis with superficial ulceration | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer* |
| 5 | No esophagitis, antral and body gastritis | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |
| | Postsurgical Group | |
| | Preoperative | Postoperative |
| 1 | No esophagitis, antral gastritis | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |
| 2 | Colonic interposition, antral gastritis, pyloroplasty | Colonic interposition, no gastritis, anastomosis healthy, no jejunal ulcer |
| 3 | No eosphagitis, antral gastritis, pyloroplasty | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |
| 4 | No esophagitis, antral, body and fundic gastritis, pyloroplasty | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |
| 5 | No esophagitis, antral, body and fundic gastritis, slipped Nissen, pyloroplasty | No esophagitis, no gastritis, anastomosis healthy, no jejunal ulcer |

* A postoperative jejunal ulcer developed 1 year after surgery. The ulcer healed on H2 blockers without complication.

TABLE 6. *Histologic Score for Alkaline Reflux Gastritis**

| Patient No. | Primary Group | |
|---|---|---|
| | Before Operation | After Operation |
| 1 | — | 7 |
| 2 | 5 | 7 |
| 3 | 5 | 1 |
| 4 | — | 0 |
| 5 | 1 | 3 |
| | Postsurgical Group | |
| 1 | 1 | 1 |
| 2 | — | 1 |
| 3 | — | 3 |
| 4 | 8 | 0 |
| 5 | 4 | 3 |

* Scale of 0 to 12.

*Body Weight*

There was no statistically significant change in body weight after surgery.

## Discussion

Suprapapillary Roux-en-Y duodenojejunostomy was designed to provide diversion of bile and pancreatic juice while leaving the proximal duodenum, pylorus, and stomach undisturbed. Conceptually, this has benefits over the classical Roux-en-Y gastrojejunostomy diversion procedure: it allows preservation of a normal gastric reservoir, antral and pyloric function, duodenal inhibition of acid secretion, and stimulation of the duodenal mucosa by chyme. An important part of the latter is the release of gastrointestinal hormones and digestive gland secretion. These functions are lost after the classical operation and result in early satiety, delayed gastric emptying, dumping, and the occurrence of jejunal ulcers. The latter has been reported to be as high as 10–15%. The incidence of ulcers can be reduced by the addition of vagotomy,[18] but this is not without its own side effects. Even with vagotomy, the possibility of jejunal ulceration still exists.[17]

The fear of performing a proximal duodenojejunostomy is the possibility of a high incidence of jejunal ulcers since the jejunum is exposed to the full barrage of gastric acidity without neutralization by biliary and pancreatic secretions. This fear may be unwarranted because of two physiologic factors. First is the observation that instillation of acid into the proximal duodenum suppresses basal and stimulated gastric acid secretions. This effect was first shown in dogs by Sokalov[22] and widely confirmed by others.[23–29] The same mechanism

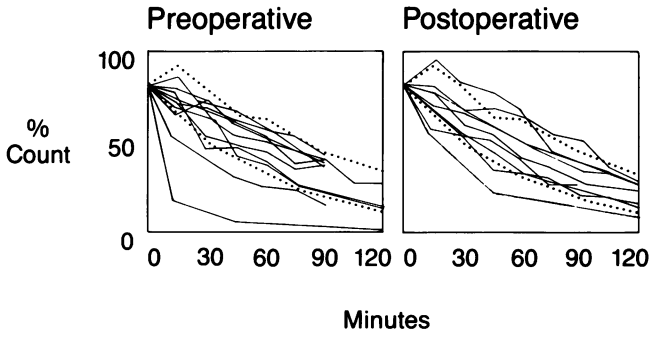


FIG. 11. Comparison of preoperative and postoperative gastric emptying scans taken in the anterior projection in patients who have undergone a duodenal switch procedure. The dotted line represents the 95th and 5th percentile limits measured in control subjects.

was shown in humans by Griffiths[30] and Shay et al.[31] Second is the observation in animals[32] and humans[33] that the proximal duodenum secretes bicarbonate, which, along with the overlying layer of mucus, provides a barrier to protect the surrounding mucosa from acid and peptic damage.

The current study indicates that these factors remain operational when the proximal duodenum is preserved in continuity with the stomach after diversion of biliary and pancreatic secretions. In the animal studies, there was a significant reduction in the incidence of jejunal ulceration compared with that observed with a direct pylorojejunostomy. In the human studies, jejunal ulceration did not occur unless gastric hypersecretion was present.

The advantage of a suprapapillary Roux-en-Y duodenojejunostomy is that a vagotomy and alteration of gastric or pyloric anatomy are not necessary to prevent jejunal ulceration. This allows preservation of normal gastric function and avoids the complications associated with vagotomy and resection.[12,34,35] Critchlow et al. reported an experience with a similar procedure in three patients for the treatment of symptomatic duodenal diverticula.[36] No jejunal ulceration occurred and the patients remained symptom free on subsequent follow-up. An important observation is that patients do not have delayed gastric emptying after a "duodenal switch" as reported after a Roux-en-Y gastrojejunostomy and vagotomy. Consequently, this delay must be due to either the vagotomy or denervation of the Roux limb. For this reason, we avoid dividing the mesentery to preserve the nerve supply to the jejunum when constructing the Roux-en-Y.

The endoscopic gastritis observed in patients with duodenogastric reflux is believed to be due to regurgitated bile and pancreatic enzymes. Both have been shown experimentally to be capable of damaging the gastric mucosa in animals,[37,38] and in humans, the presence of both in the stomach has been associated with gastric ulcer.[39,40] The "duodenal switch" procedure effectively diverted bile and pancreatic secretion and resulted in endoscopic healing of gastritis.

The histologic gastritis observed before operation rarely resolved. Persistence of intestinal metaplasia after bile diversion appears to account for elevated postoperative histologic scores. This phenomenon may be analogous to the development of Barrett's epithelium with gastroesophageal reflux and, like Barrett's, a return of the mucosa to normal histology, after removal of the stimulus to its production, may never occur or may take many years.

In humans, duodenogastric reflux occurs sporadically.[41] Increased duodenogastric reflux has been measured in patients with esophagitis, gastric ulcer, cholelithiasis, and previous cholecystectomy.[6,9,42–44] We reported five patients with the primary abnormality discovered during an evaluation of foregut symptoms. All complained of epigastric pain, nausea, and bilious vomiting. Two patients had documented gastroesophageal reflux disease, none had cholelithiasis, but all had endoscopic gastritis. Four of these patients had an alkaline gastric pH shift when measured over 24 hours.

TABLE 7. *Basal and Stimulated Gastric Secretion Analysis (mEq/h)*

| | Patient No. | Preoperative | | Postoperative | | | |
|---|---|---|---|---|---|---|---|
| | | BAO | MAO | BAO | MAO | Serum Gastrin (pg/mL) | |
| Primary group | 1 | 0 | 0 | 0 | 0 | >1000 | >1000 |
| | 2 | 2.1 | 17.4 | 1.0 | 9.8 | 55 | 43 |
| | 3 | 12.5 | 42.3 | 2.0 | 16.6 | 35 | 44 |
| | 4 | 4.7 | 29.0 | 5.0 | 39.0 | — | 15 |
| | 5 | 1.0 | 33.7 | 0.7 | 35.0 | 443 | 378 |
| Postsurgical group | 1 | 3.0 | 17.8 | — | — | — | — |
| | 2 | — | — | 3.8 | 9.3 | — | 60 |
| | 3 | — | — | 1.6 | 2.0 | — | 20 |
| | 4 | 1.5 | 25.1 | 0.6 | 4.4 | 46 | 39 |
| | 5 | 1.5 | 4.5 | 0.4 | 6.8 | — | 50 |

When subjected to a discriminant analysis, the alkaline change had a high probability of being caused by excessive duodenogastric reflux. One patient, with gastric hypersecretion, did not show an alkaline shift or a positive discriminant analysis, but duodenogastric reflux was confirmed by radionuclide scanning. It may be that patients who have duodenogastric reflux after an acid-reducing procedure, initially had the primary disease, which was masked by hypersecretion. A clue that duodenogastric reflux might be present in a patient with hypersecretion is the persistance of bile reflux symptoms while the patient is receiving H2 blockers. Such patients are likely to have persistence of symptoms after vagotomy, and the addition of a duodenal switch to the initial procedure may be, as in our patient, beneficial to the successful control of symptoms.

The mechanism by which reflux occurs in the patient who is not operated on is poorly understood but probably related to a functional abnormality of the duodenum or pylorus.[5,41] The abnormality does not appear to affect gastric emptying in that all our patients who did not have previous surgery showed normal emptying curves.

Lawson showed in animals that severe duodenogastric reflux can cause a reduction in the number of parietal and chief cells in the gastric fundus.[38] In humans, a decrease in acid production has been observed with duodenogastric reflux and was ascribed to the loss of parietal cells or back diffusion of hydrogen ions. In severe forms, this can cause alkalinization of the antrum and elevated serum gastrin levels. Two of our patients with primary disease had a high serum gastrin level, one of whom had pernicious anemia. It may be that pernicious anemia is the end stage of duodenogastric reflux.

Of the five patients with postsurgical duodenogastric reflux, two had persistent epigastric pain and nausea after the biliary diversion procedure. Both patients had a history of esophageal motility disorder requiring a myotomy for relief of dysphagia, and one patient had an esophagectomy and colon interposition for intractable symptoms. This history may account for some of their current complaints.

In two patients with primary disease, a concomitant Nissen fundoplication was done to correct coexisting documented gastroesophageal reflux. All patients with postsurgical disease had a prior esophageal procedure: three had esophageal myotomy, and two had an antireflux operation. Thus the overall incidence of esophageal disease in our patients with duodenogastric reflux was 70% (7 of 10), suggesting that an underlying diffuse foregut disorder exists. Initially, the symptoms of duodenogastric reflux, gastroesophageal reflux, or an esophageal motor disorder may predominate. Correction of the predominant component may allow the symptoms of the other components, if present, to emerge. This emphasizes the importance of a careful preoperative and postoperative evaluation of patients with benign foregut disease. What might be considered a failure of a previous procedure to improve symptoms is, in reality, the emergence of symptoms due to another abnormality not appreciated before the operation.

Prolonged medical therapy consisting of H2 antagonists, antacids, and sucralfate did not relieve symptoms of epigastric pain, nausea, and bilious vomiting, and was the driving force for surgical therapy. Our experience indicates that if the patient has preoperatively marginal or definite hypersecretion, a limited proximal gastric vagotomy should be performed with the initial operation. Patients with normal and low gastric acid secretion do not need a vagotomy to remain free from jejunal ulceration.

In summary, bile diversion by "duodenal switch" preserves the suprapapillary duodenum. Experimentally, this causes a significant decrease in the incidence of jejunal ulceration. Clinically, the operation corrects pathologic duodenogastric reflux, alleviates the symptoms of the primary disease, improves the symptoms of the postsurgical disease, heals endoscopic gastritis, does not delay gastric emptying, and is, in the presence of normal acid secretion, not ulcerogenic.

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## DISCUSSION

DR. JOHN L. SAWYERS (Nashville, Tennessee): In 1965, Toye and Williams in the United Kingdom described a new postgastrectomy syndrome, which was reflux gastritis, and differentiated this from chronic afferent loop syndrome, which had been confused with alkaline gastritis. They treated this "new syndrome" by inserting an isoperistaltic jejunal segment between the gastric pouch and the duodenum. In 1974, Dr. Herrington and I presented at the American Surgical Association results of an isoperistaltic jejunal segment *versus* Roux-en-Y gastrojejunostomy for alkaline reflux gastritis and concluded that the latter was superior.

We currently have 110 patients followed from a minimum of 1 to more than 10 years with a Visick I or II clinical grading, *i.e.*, a satisfactory clinical result, in 79% of the patients. The 21% of patients with less than a satisfactory result have no evidence of bile reflux gastritis into their stomach, but they have motility problems of slow gastric emptying or Roux limb syndrome. In two of the 110 patients, a marginal ulcer developed despite what we believed was a complete truncal vagotomy.

Is Dr. DeMeester's new operation of a duodenal switch an improvement over Roux-en-Y gastrojejunostomy? There are a few things that bother me about the technique, and I wish he would elaborate to us how he locates the exact point in the second part of the duodenum where he puts his GI stapler and avoids injury to the distal end of the common bile duct and accurately locates the ampulla. It is my impression from reading the manuscript that the duodenum is not open to locate the ampulla.

Gastric emptying was almost normal in all of his 10 patients. This is contrary to results that we have seen, and others have reported, after pyloric sparing pancreaticoduodenectomy operations in which about one third of the patients have delayed gastric emptying, at least for a while.

Dr. DeMeester, is this difference due to sparing the duodenal pacemaker because you divide the duodenum more distally in your duodenal switch operation?

Five of his patients who had remedial operation had no previous gastric operation. I have never done a remedial operation on anyone for primary duodenogastric reflux. I am concerned that there is an increasing number of patients diagnosed as having reflux gastritis with insufficient evidence to substantiate the diagnosis. Concern exists that some surgeons may be too eager to do remedial operations on patients with reflux gastritis.

Dr. DeMeester, however, has carefully studied his patients to document reflux, and I am sure that they all were symptomatic.

Although I am enthusiastic about operations to spare the pylorus, I am concerned about postoperative jejunal ulcer developing after this procedure. One of his ten patients has already had a jejunal ulcer, and I would like to ask him why he does not add proximal gastric vagotomy to all these patients since it is a safe type of vagotomy with minimal side effects.

DR. WILLIAM SILEN (Boston, Massachusetts): My colleague, Dr. Jonathan Critchlow, and I about 1 year ago described an identical procedure in the *Annals of Surgery*, but using that procedure for a