WHAT'S NEW IN GENERAL SURGERY

A New Physiologic Approach for the Surgical Treatment of Patients With Barrett's Esophagus

Technical Considerations and Results in 65 Patients

Attila Csendes, M.D., Italo Braghetto, M.D., Patricio Burdiles, M.D., Juan Carlos Díaz, M.D., Fernando Maluenda, M.D., and Owen Korn, M.D.

From the Department of Surgery, University Hospital, Santiago, Chile

Objective

To determine the results of a new surgical procedure for patients with Barrett's esophagus.

Summary Background Data

In addition to pathologic acid reflux into the esophagus in patients with severe gastroesophageal reflux and Barrett's esophagus, increased duodenoesophegeal reflux has been implicated. The purpose of this study was to establish the effect of a new bile diversion procedure in these patients.

Methods

Sixty-five patients with Barrett's esophagus were included in this study. A complete clinical, radiologic, endoscopic, and bioptic evaluation was performed before and after surgery. Besides esophageal manometry, 24-hour pH studies and a Bilitec test were performed. After surgery, gastric emptying of solids, gastric acid secretion, and serum gastrin were determined. All patients underwent highly selective vagotomy, antireflux procedure (posterior gastropexy with cardial calibration or fundoplication), and duodenal switch procedure, with a Roux-en-Y anastomosis 60 cm in length.

Results

No deaths occurred. Morbidity occurred in 14% of the patients. A significant improvement in symptoms, endoscopic findings, and radiologic evaluation was achieved. Lower esophageal sphincter pressure increased significantly (p < 0.0001), as did abdominal length and total length of the sphincter (p < 0.0001). The presence of an incompetent sphincter decreased from 87.3% to 20.9% (p < 0.0001). Three of seven patients with dysplasia showed disappearance of this dysplasia. Serum gastrin and gastric emptying of solids after surgery remained normal. Basal and peak acid output values were low. Twenty-four hour pH studies showed a mean value of 24.8% before surgery, which decreased to 4.8% after surgery (p < 0.0001). The determination of the percentage time with bilirubin in the esophagus was 23% before surgery; this decreased to 0.7% after surgery (p < 0.0001). Late results showed Visick I and II gradation in 90% of the patients and grade III and IV in 10% of the patients.

Conclusions

This physiologic approach to the surgical treatment of patients with Barrett's esophagus produces a permanent decrease of acid secretion (and avoids anastomotic ulcer),

decreases significantly acid reflux into the esophagus, and abolishes duodenoesophageal reflux permanently. Significant clinical improvement occurs, and dysplastic changes at Barrett's epithelium disappear in almost 50% of the patients.

Reflux of duodenal content into the stomach or esophagus can be a normal physiologic event. However, excessive duodenogastric reflux in humans can occur after surgery and has been implicated in the development of gastritis, gastric ulcer, cancer of the gastric stump, and postcholecystectomy dyspepsia.¹⁻⁷ In the past decade, investigators have shown that in patients with Barrett's esophagus, there is not only pathologic acid reflux, there is severe and pathologic reflux of duodenal content into the esophagus.⁸⁻²² In addition, bile reflux has been implicated as playing an important pathogenic role in the development of adenocarcinoma in Barrett's esophagus.²³⁻²⁶

Until now, the main surgical treatment of patients with Barrett's esophagus has been a Nissen fundoplication. With this procedure, acid reflux and bile reflux into the esophagus may be decreased but never abolished completely. ^{27,28} The introduction by DeMeester et al. in 1987 of a new procedure for permanent control of pathologic duodenogastric reflux by the duodenal switch procedure seems ideal to abolish completely alkaline reflux without disturbing gastric motility and emptying. By adding highly selective vagotomy, anastomotic ulcer (which occurs in 10% of patients) also can be avoided (Fig. 1).

The purposes of the current study were to determine the immediate and late clinical results in patients with Barrett's esophagus who undergo duodenal switch procedure; to measure objectively eventual changes in gastric acid secretion, gastric emptying of solids, and serum gastrin after this procedure; to determine changes in acid reflux and bile reflux into the esophagus after this procedure; to determine the effect of permanent abolition of bile reflux on the evolution of dysplasia in Barrett's metaplastic mucosa; and to determine by a randomized study the differences between fundoplication and cardial calibration in the prevention of gastroesophageal reflux.

MATERIALS AND METHODS

Patients Studied

Sixty-five patients (36 women, 29 men) with Barrett's esophagus were studied prospectively from January 1992 to January 1996. The patients ranged in age from 21 to 77 years (mean age, 52.5 years). Patients with severe dysplasia or adenocarcinoma were excluded from the study.

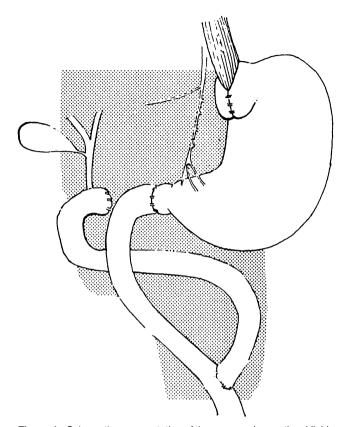


Figure 1. Schematic representation of the proposed operation. Highly selective vagotomy. Antireflux surgery and duodenojejunostomy with Roux-en-Y limb.

A special protocol including subjective and several objective evaluations was designed at the beginning of the study to provide a complete evaluation of each patient. These patients had been treated for at least 12 months with omeprazole, and symptoms of gastroesophageal reflux or endoscopic esophagitis or both had recurred days after suppression of this therapy.

Symptomatic Evaluation

A clinical questionnaire was completed by each patient about the presence of heartburn, regurgitation, dysphagia, respiratory symptoms, epigastric pain, and retrosternal pain. The severity of heartburn, regurgitation, and dysphagia was classified in three categories (mild, moderate, and severe) according to the criteria described by Iascone et al.²⁹

Radiologic Studies

In all 65 patients before surgery and in 44 after surgery, a complete radiologic evaluation of the distal esophagus,

Address reprint requests to Attila Csendes, M.D., Department of Surgery, Hospital José Joaquín Aguirre, Santos Dumont Noº 999, Santiago, Chile.

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stomach, and duodenum; gastric emptying; and anatomic characteristics of the duodenal switch surgery were studied. The presence of esophageal stricture, ulcer, and hiatal hernia also was determined. The maximal internal diameter of the distal esophagus and esophagogastric junction was measured before and after antireflux surgery, because it has been shown that with an internal diameter more than 25 mm, which corresponds to a dilated cardia or esophagogastric junction, free reflux can occur.³⁰

Upper Endoscopy

This procedure was performed with the use of the Olympus (Tokyo, Japan) GIFXQ20 instrument by two of the authors. The presence of Barrett's esophagus was determined when the squamous-columnar junction was seen 3 cm proximal to the endoscopic location of the lower esophageal sphincter (41 patients) or based on the presence of tongues proximal to the squamous-columnar junction of at least 15-mm length (24 patients). In all these patients, biopsy results showed the presence of intestinal metaplasia in these tongues (short Barrett's esophagus). However, the final precise length of Barrett's esophagus was diagnosed by comparing the location of the manometric proximal limit of the lower esophageal sphincter and the endoscopic location of the squamous-columnar junction from the incisors. That is why the precise location of this junction was defined carefully at the beginning and at the end of the endoscopy.³¹ This procedure also was used in the early postoperative period to dilate anastomotic strictures in some patients. At least five biopsy specimens were taken in each patient to confirm the presence of the metaplastic epithelium.

Manometric Evaluation

This test was conducted after patients fasted for 12 hours and while they were in the supine position.^{31,32} The device consisted of four polyvinyl catheters bound together so that the 0.5-mm side hole was 3 cm apart from each of the other holes (Synectics Medical, Stockholm, Sweden).

Each catheter was perfused from a pneumohydraulic pump (Arndorfer Medical Specialties, Milwaukee, WI) at a rate of 0.5 mL/minute. Before each test, the whole system was calibrated, and the sudden occlusion of the side hole produced an increase of 400 mmHg at 1 second. The recording catheter was introduced through the mouth after patients were administered slight pharyngeal anesthesia in the stomach. In this way, the distances from the incisors were comparable to those on endoscopic findings. The end-expiratory fundic pressure was taken as zero reference, and all values were expressed in millimeters of mercury. Three manometric characteristics of the lower

esophageal sphincter were determined: resting pressure, total length, and abdominal length.

This latter measurement was taken from the distal end of the sphincter up to the respiratory inversion point, which is the level at which the end-expiratory pressure changes from a positive to a negative deflection. 31,32 In each patient, three slow pull-throughs were obtained. The mean values were taken of all measurements of the three catheters, that is, 12 determinations in each patient. The location of the distal and proximal ends of the lower esophageal sphincter was measured in centimeters from the incisors. The amplitude of the distal esophageal contractile waves was determined. This test was performed before and 6 months after surgery.

Gastric Acid Secretion

Basal and peak acid output was determined 6 months after surgery using histamine acid phosphate (0.04 mg/kg), as described previously.³³ The values were expressed in millimole per hour.

Gastric Emptying of Solids

This test was performed 6 months after surgery using ovalbumin marked with technetium Tc-99m, determining the time at which 50% of the ingested isotope has been emptied from the stomach (T ½) and the percent of retention at 60 to 90 and 120 minutes. Complete details of this procedure have been described elsewhere. For this study, 30 control subjects without gastrointestinal disease were evaluated. All these subjects showed normal upper gastrointestinal endoscopy.

Serum Gastrin

This test was performed after patients fasted for 12 hours. Peripheral venous samples were taken, and serum gastrin concentrations were determined by radioimmune analysis.³⁵ The normal value is below 200 pg/mL.

24-Hour Intraesophageal pH Study

This test was performed after patients fasted for 12 hours, ^{36,37} at which time the catheter was introduced through the nose to the stomach (Digitrapper Synectics, Stockholm, Sweden). The catheter was then placed 5 cm above the lower esophageal sphincter (manometry is always performed before this procedure), and the microcomputer analyzed more than 20,000 determinations during 24 hours. From six different parameters that can be evaluated, the most useful and practical is the total percentage of time in which the intraesophageal pH remains below 4, with a normal value less than 5% during 24

Table 1. ENDOSCOPIC FINDINGS IN PATIENTS WITH BARRETT'S ESOPHAGUS BEFORE AND AFTER DUODENAL SWITCH

	Before (n = 65)	After (n = 65)
Location of squamo	us columnar junction (cm	from incisors)
Mean	33.6 ± 2.7	$35.0 \pm 2.7 (p < 0.1)$
Median	34	35
Range	29-39	30-40
•	$(34\% \le 32 \text{ cm})$	$(20\% \le 32 \text{ cm})$
	$(66\% \ge 33 \text{ cm})$	$(80\% \ge 33 \text{ cm})$
Presence of erosions	s proximal to squamous of	columnar junction
Absent	27.7%	90% (p < 0.001)
Grade I	43.1%	10%
Grade II	29.2%	0
Mild dysplasia	7 patients (10.8%)	4 patients (6.1%)

hours (72 minutes). This test was performed on the patients before and 6 months after surgery.

24-Hour Monitoring of Esophageal Exposure to Duodenal Juice

This new procedure has been developed to measure spectrophotometrically the intraluminal bilirubin concentration. 20,38,39 It consists of a portable opticoelectronic data logger (Bilitec 2000; Synectics Medical) connected to a fiberoptic probe that is passed transnasally and positioned 5 cm proximal to the lower esophageal sphincter. This sensor measures 3 mm in diameter and 140 cm in length and contains 30 plastic optical fibers bound together. The tip of the probe contains a 2-mm space for sampling. The light source is provided by two light-emitting diodes that emit a 460-nm signal light (blue spectrum) and a 565nm reference light (green spectrum). They are stimulated alternately for a duration of 0.5 second. The specific wavelength of absorption for bilirubin is 453 nm and is highly reproducible, despite pH changes caused by environment or food intake. It allows determination of more than 5400 measurements during 24 hours. The final calculation is based on the percentage of time that bilirubin is measured in the esophagus; below 2% of the time (28 minutes) is normal. Because of its recent introduction, this procedure was used in the last 32 patients and was repeated in 23 patients 6 months after surgery.

Surgical Procedure

The three main principles of the surgical procedures are to decrease acid secretion and to avoid anastomotic ulcer by performing highly selective vagotomy; to avoid acid reflux to the esophagus by performing an antireflux procedure (either fundoplication or cardial calibration);

and to abolish permanently duodenogastroesophageal reflux by performing end-to-end duodenojejunostomy with a 50-cm long limb.

Patients are placed in a tilted position with the chest elevated above the feet. Highly selective vagotomy is performed as described elsewhere. 32,40-43 When posterior gastropexy and calibration of the cardia are performed, 40,43 four nonabsorbable stitches are used, and the last two are left for posterior gastropexy, using a bougie N° 30 for a precise calibration. When a 360° fundoplication is performed, four stitches are used, and the proximal and distal sutures are stitched to the esophageal wall. Two stitches for an anterior fundophrenopexy are always placed to avoid an anterior iatrogenic paraesophageal hernia. Then, the duodenal switch is performed (Fig. 1). The pylorus and the common bile duct are located by performing an extensive Kocher's maneuver. The peritoneum above the

Table 2. MANOMETRIC FEATURES OF LOWER ESOPHAGEAL SPHINCTER IN PATIENTS WITH BARRETT'S ESOPHAGUS BEFORE AND AFTER SURGERY

	Before (n = 63)	After (n = 43)	
Resting pressure (mmHg)			
Mean		12.5 ± 3.6	(p < 0.0001)
Median	7	12	
Range	3-25	5–25	
% increase pressure		50	
% of patients who			
increase LESP		86	
Total length (mm)			
Mean	28.7 ± 9.1	39.1 ± 8.2	
Median	30	40	(p < 0.0001)
Range	8–50	25-60	
% increase length		41	
% patients who			
increase sphincter		70	
length		76	
Abdominal length (mm)	50.50	440 . 04	
Mean Median	5.8 ± 5.6 5	14.3 ± 6.4 15	(= < 0.0004)
	0-23	0-27	(p < 0.0001)
Range % increase length	0-23	160	
% patients who		100	
increase abdominal			
length		76	
Location of LES (distance		70	
from incisors in cm)			
Mean	395 + 33	41.3 ± 2.7	
Median	40	42	(p < 0.007)
Range	32-48	37-47	(p < 0.001)
% patients who			
increase esophageal			
length		74	
ESD = ES prossuro: ES =	lower coopbass	al aphinatar	

LESP = LES pressure; LES = lower esophageal sphincter.

Table 3.	PREVALENCE OF INCOMPETENT ESOPHAGEAL SPHINCTER IN PATIENTS WITH
	BARRETT'S ESOPHAGUS BEFORE AND AFTER DUODENAL SWITCH

Factors	Before $(n = 63)$	After $(n = 43)$	р
LESP < 6 mmHg	26 (41.2%)	2 (4.8%)	< 0.0007
Total length < 20 mm	18 (28.6%)	0	< 0.0003
Abdominal length < 10 mm	53 (84.1%)	6 (14%)	< 0.00001
No. of patients with incompetent LES	55 (87.3%)	9 (20.9%)	< 0.00001
Patients with 1 factor (+)	24	9	
Patients with 2 factors (+)	22	0	
Patients with 3 factors (+)	0	0	

duodenum is opened and the posterior wall of the duodenum is dissected, where the duodenal wall adheres to the pancreas.

At the right surface of the duodenum, it is always necessary to cut one or two short vessels that bleed if they are not tied. A duodenal section is performed with the TA-55 stapler, leaving the duodenal distal stump closed and the proximal duodenal stump open, allowing determination of whether the papilla is located at this portion (which has never occurred). Then the jejunal limb 30 to 35 cm distal to the Treitz's angle is sectioned, to avoid too much dissection of the mesenterium. This 50-cm long limb is placed through the mesocolon exactly at the hepatic angle, leaving it retrocolic.

The end-to-end duodenojejunostomy is made with poliglactine 910 or poliglecaprone in two layers (in the last 20 patients, only one layer was used to avoid strictures that occurred in some of the early patients). Then the jejunojejunal anastomosis is performed in a one-layer procedure with the same suture material. A soft drain is placed around the duodenal stump. The nasogastric tube is left for 2 days if the 24-hour output is less than 300 mL. Patients begin to be fed on the fourth day after surgery, at which time a plain x-ray of the abdomen is performed for surgeons to determine whether gastric dilatation has occurred. The average hospital stay is 7 days after surgery.

Follow-Up

A careful protocol for follow-up was established at the beginning of this study. Clinical examination is performed at 15 days, 1 month, 3 months, 6 months, and 1 year after surgery, then every 6 to 12 months. A barium upper gastrointestinal series is performed 3 months after surgery. Endoscopy, manometry, gastric emptying, serum gastrin, gastric acid secretion studies, 24-hour pH, and Bilitec 2000 tests are performed 6 months after surgery when a full recovery has been achieved. After that, endoscopy and biopsies are performed annually.

Statistical Analysis

For statistical evaluation, the Fisher's exact test, chi square test, and Mann-Whitney test were used, with p < 0.05 considered significant.

Biopsies

For each patient, at least five endoscopic biopsies were taken before surgery, distal to the squamous columnar junction. The specimens were fixed immediately in 10% formaldehyde solution and stained with hematoxylin-eosin. The presence of intestinal metaplasia and mild or moderate dysplasia was evaluated carefully.

RESULTS

Before surgery, heartburn and regurgitation were seen in 100% and 91% of patients, respectively. Sixty-nine percent had retrosternal pain. Forty-three percent of patients had respiratory symptoms. The duration of symptoms corresponded to 92 months, with less than 60 months of duration in 42% of patients and more than 60 months of duration in 58% of patients. Previously, two patients underwent failed antireflux surgery, whereas 45% of patients had cholecystectomy.

Clinical gradation of symptoms showed that almost 50% of the patients had severe heartburn or regurgitation, whereas in the majority of patients dysphagia was mild. The main endoscopic findings before and after surgery are listed in Table 1. Although there was a descent in the squamous—columnar junction, this was not significantly different. The presence of erosive esophagitis proximal to the squamous—columnar junction showed a significant decrease after surgery, with disappearance of erosions in 90% of patients (p > 0.001). The endoscopic length of the Barrett's esophagus showed no change after surgery. Seven patients (10.8%) had mild dysplasia in the metaplastic epithelium after surgery. In three of these patients after surgery, this dysplasia disappeared and instead there

	Before (n = 65)	After (n = 14)	р
Hiatal hernia	24 (37%)	2 (4.5%)	< 0.0001
Stricture	6 (9.2%)	2 (4.5%)	>0.47
Ulcer	15 (23%)	2 (4.5%)	< 0.01
Maximal internal diameter esophagogastric junction (mm)	Mean 35 ± 8.6	10.8 ± 3.6	< 0.0001
	Median 35	10	
	(20-50)	(7-30)	

<24 mm, 6.2%

>25 mm, 93.8%

Table 4. RADIOLOGIC FINDINGS IN PATIENTS WITH BARRETT'S ESOPHAGUS BEFORE AND AFTER DUODENAL SWITCH

was normal intestinal metaplastic mucosa. The manometric features of the lower esophageal sphincter before and after duodenal switch are listed in Table 2. Before surgery, it was performed in 63 patients (97%), and after surgery it was performed in 43 (90%) of the 48 patients, with complete follow-up of more than 12 months. There was a significant increase of lower esophageal sphincter pressure in 50% after surgery (p < 0.0001). In 86% of the patients, there was an increase in sphincter pressure, and in 7% there was a decrease in sphincter pressure. There was a 41% increase in total length (p < 0.0001). Only three patients (7%) showed a decrease in total length.

The abdominal portion of the lower esophageal sphincter increased by 160% in length (p < 0.0001). In six patients (14.3%), there was a decrease in abdominal length. The location of the lower esophageal sphincter moved distally 2.3 cm (p < 0.007), which meant the

Table 5. GASTRIC ACID SECRETION AND SERUM GASTRIN VALUES IN PATIENTS WITH BARRETT'S ESOPHAGUS AFTER DUODENAL SWITCH

Serum gastrin (pg/mL) (n = 30) 27 patients (90%), mean 71 (20-200) 2 patients (6.7%), mean 281 (219-343) 1 patient (3.3%), (4200-1039) Basal acid output (mM/hr) (n = 22)Mean value 1.2 (0-11.4) 60% reduction compared with normal values <1 15 (68.1%) 1.1-2 5 (22.7%) >2-12(9.1)Normal value $3.0 \pm 1 (1-5)$ Peak acid output (mM/hr) (n = 22)Mean value 11.7 (0-26.5) 42% reduction compared with normal values 8 (36.4%) < 10 11(50%) = 11-203 (13.6%) >21 Normal value 20 + 5 (10-30)

whole esophagus was elongated by 10%, because the upper esophageal sphincter, which was located at a mean distance of 16 cm from the incisors, showed no change (esophageal length before surgery was 23 cm, and after surgery it was 25.3 cm).

<15 mm, 97.7%

>16 mm. 2.3%

The amplitude of the distal waves of the esophagus showed no significant change. As listed in Table 3, the prevalence of an incompetent lower esophageal sphincter can be defined when one of the following characteristics is present: lower esophageal sphincter pressure equal to or less than 6 mmHg, total length less than 20 mm, and abdominal length of the sphincter less than 10 mm. The single factor most altered was the abdominal length of the sphincter. Before surgery, 55 (87%) of patients had an incompetent sphincter, which remained so in nine (21%) of the patients after surgery (p < 0.00001). The main radiologic findings before and after surgery are listed in Table 4. Hiatal hernia was present in 37% of the patients and recurred in two patients after surgery (p < 0.0001). A stricture was seen in six patients and disappeared in four patients; marginal ulcer (located at the squamous-columnar junction) was present in 15 patients (23%) and healed in 13 of them.

The maximal internal diameter of the esophagogastric junction was dilated significantly before surgery and decreased to a mean value of 10.8 mm after surgery (p < 0.0001).

No one died as a result of surgery. Early complications were seen in 13.8% of the patients, corresponding mainly to a partial stricture of the duodenojejunostomy, which was managed by endoscopic dilatation (two or three dilatations) in the early postoperative period. This was seen primarily when the two-layer anastomosis was performed.

One patient had a hemoperitoneum because of rupture of the proximal pole of the spleen, which was assessed by several abdominal scanners; the patient did not require reoperation. At the beginning of our study, the jejunal proximal limb was twisted erroneously in one patient, and this was the only patient who underwent reoperation (1.5%). In another patient who had a tight Nissen wrap,

Table 6. GASTRIC EMPTYING OF SOLIDS IN PATIENTS WITH BARRETT'S ESOPHAGUS SUBMITTED TO DUODENAL SWITCH COMPARED WITH CONTROLS (MEAN \pm SD)

	Duodenal Switch (n = 33)	Controls (n = 30)	р
T _{1/2} % retention 60 min % retention 90 min % retention 120 min	73.5 ± 22 (18-220) 50 ± 21 38 ± 18 22 + 19	84 ± 26 62 ± 12 41 ± 17 30 ± 15	NS NS NS
NS = not significant.			

endoscopic dilatation was performed. Six months after surgery, she had a perforation that immediately was reoperated on, and the Nissen procedure was redone. Her course after that was excellent.

A randomized study was performed to compare procedures among the first 40 patients who underwent surgery. When choosing between the Nissen procedure or posterior gastropexy with cardial calibration (cardiogastropexy), 18 patients chose a Nissen procedure and 22 patients chose cardiogastropexy.

The results of serum gastrin and gastric acid secretion studies are listed in Table 5. Serum gastrin was performed in 30 patients 6 months after surgery. In only one patient (3.3%) did it have a significantly high value, which remained high in a second control. She had anacidity during the histamine test, but she remained completely asymptomatic. Gastric acid secretion studies were performed in 22 patients 6 months after surgery. Basal acid output showed a low value compared with that of normal subjects (approximately 60% less than control subjects), whereas peak acid output values were below normal range in 86% of the patients. Gastric emptying results of solids in 33 patients after the duodenal switch procedure are listed in Table 6 and are compared with those of control

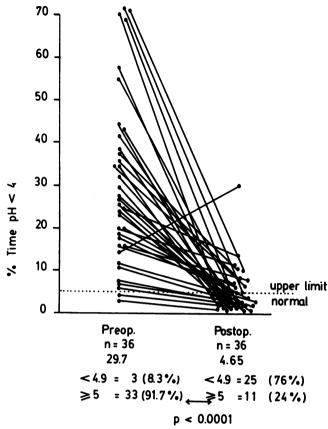


Figure 2. Twenty-four-hour intraesophageal pH monitoring in 36 patients before and after surgery.

values. There were no significant differences with control subjects. Only two patients (6%) showed more delayed emptying than control subjects, whereas in five patients (15%), emptying was faster than in control subjects.

The results of intraesophageal 24-hour pH monitoring before and 6 months after surgery are listed in Table 7 and shown in Figure 2. This test was performed in 59 patients (91%) before surgery and in 37 (72%) of the patients with late follow-up. Before

Table 7. 24-HOUR INTRAESOPHAGEAL pH STUDIES IN PATIENTS WITH BARRETT'S ESOPHAGUS BEFORE AND AFTER DUODENAL SWITCH

	Before (n = 59)	After (n = 37)
% time < 4 in 24 hr		
Mean	24.8 ± 19.6	4.8 ± 5.7
Median	20	3.8 (p < 0.0001)
Range	0.4-75	(0-30)
Ç	8 (13.5%) <4.9%	25 (67.6%) < 4.9%
	33(55.9%) = 5-30%	7 (18.9%) = 5-10%
	18 (30.5%) >30.1%,	5 (13.5%) >10.1%

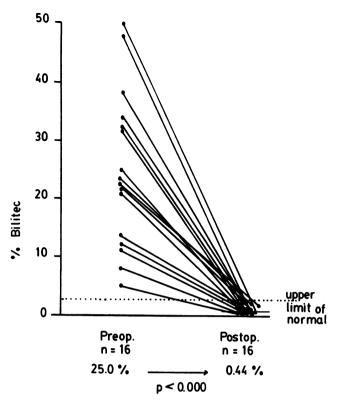


Figure 3. Twenty-four-hour intraesophageal bilirubin monitoring in 16 patients before and after surgery.

surgery, there was significant pathologic acid reflux into the esophagus in 86.5% of patients, which remained positive in 32.4% of patients (p < 0.001). However, when the same patients were evaluated before and after surgery (Fig. 2), 24% of patients still had pathologic acid reflux into the esophagus.

There were no significant differences between fundoplication or cardial calibration with respect to control of acid reflux into the esophagus. The results of the 24-hour intraesophageal bilirubin reflux monitoring into the esophagus are listed in Table 8 and shown in Figure 3. It was performed in 32 patients before

surgery, and, until now, it has been performed in 23 patients late after surgery. In all patients, bile reflux to the esophagus disappeared completely (p < 0.0001). The late results according to Visick grading in the 51 patients with more than 12 months of followup (Table 9) showed that almost 90% of the patients were classified in Visick I or II, whereas five patients (10.3%) were classified in Visick III or IV with a mean follow-up of 28 months. As did a Visick IV patient, these patients had recurrence of hiatal hernia and acid reflux and a decrease of lower esophageal sphincter pressure after surgery. The Visick IV had a Nissen procedure. The results in the four patients with Visick III corresponded to those in two patients with stricture and ulcer who were taking aspirin for cardiovascular disease and showed recurrence of pain, heartburn, and ulcer. They have been treated medically. One patient has heartburn and diarrhea with loss of weight. He showed an increase in 24-hour pH studies from 13% to 30% and has been treated medically because of chronic psychiatric problems. Another patient had weight loss and diarrhea without reflux symptoms. None of the patients have undergone reoperation. There was a similar failure rate after the Nissen procedure (11.1%) compared with after cardiogastropexy (10.6%).

DISCUSSION

Recently, it has been shown that patients with Barrett's esophagus have several pathophysiologic alterations, 18,21,24,27,44-48 such as pathologic acid reflux to the esophagus, presence of an incompetent sphincter, increased acid secretion, delayed gastric emptying, altered motility of distal esophageal wave, and increased duodenogastroesophageal reflux.

The main pathogenic difference between patients with Barrett's esophagus and patients with erosive esophagitis is the presence of an increased reflux of duodenal content into the esophagus.⁸⁻²⁶ However, in recent years, it has been difficult to show this. Methods

Table 8.	24-HOUR BILITEC STUDIES IN PATIENTS WITH BARRETT'S ESOPHAGUS	
	BEFORE AND AFTER DUODENAL SWITCH	

	Before (n = 32)	After (n = 23)	р
% time of bilirubin in 24 hr			
Mean	23.5 ± 15.2	0.7 ± 0.7	< 0.0001
Median	24	0.7	
Range	0–51	0–2	
-	<2.0 = 2 (6.2%)	0 = 7 (30.4%)	
	2.1-20 = 14 (43.8%)	0.1-2 = 16 (69.6%)	
	>20.1 = 16 (50%)	>2.1 = 0	

such as gastric pH monitoring, aspiration techniques, and cholescintigraphy have been criticized because of their low sensitivity and specificity. Fiberoptic monitoring of bilirubin was introduced recently to measure intraluminal exposure of bilirubin for an entire 24-hour cycle on an outpatient ambulatory basis. It has been shown that this is an accurate method to assess bile reflux into the esophagus. With this procedure, we also have shown in our patients with Barrett's esophagus that in 94% of the patients, Bilitec 2000 monitoring showed a markedly increased intraesophageal exposure time to bile.

Forty-five percent of our patients previously underwent cholecystectomy. It has been shown that cholecystectomy increases duodenogastric reflux episodes. 49,50

It is surprising that although the pathogenic changes are different in patients with erosive esophagitis and those with Barrett's esophagus, the same surgical technique, Nissen fundoplication, has been used. Therefore, in many articles, both groups are joined together and results are expressed as a unique group. With the introduction of the duodenal switch procedure by DeMeester et al.⁵ in 1987 to abolish duodenogastric pathologic reflux in patients with alkaline gastritis, it seemed to us adequate for the control of excessive duodenogastroesophageal reflux in patients with Barrett's esophagus. That is why we developed the current physiologic approach for patients with Barrett's esophagus based on the following considerations:

- 1. To perform a highly selective vagotomy to decrease acid secretion, to avoid anastomotic ulcer, and to improve gastric emptying.
- 2. Antireflux surgery either 360° Nissen fundoplication or posterior cardiogastropexy to compare results of both antireflux techniques by randomization.
- Duodenal switch procedure to abolish completely and permanently duodenoesophageal reflux and therefore avoid the development of dysplasia or adenocarcinoma.

In the current article, we have shown that our surgical approach fulfills the following criteria:

- A decrease in gastric acid secretion is accomplished, as measured by basal and peak acid outputs in our patients.
- 2. Gastric emptying is normal or slightly accelerated.
- 3. Serum gastrin is not changed, although there is a decrease in acid secretion.
- 4. Significant improvement of the incompetent sphincter occurs in the majority of patients.
- 5. Significant control of acid reflux into the esophagus by 24-hour pH monitoring is obtained.
- 6. The most important and new finding is the demon-

stration of a complete abolition of duodenoesophageal reflux in these patients.

Our study is the first in patients with Barrett's esophagus. The only other clinical study concerning the duodenal switch procedure was reported by DeMeester et al., who studied 10 patients after this surgery. Among their patients who did not undergo highly selective vagotomy, anastomotic ulcers developed in 10%. Other studies in dogs and baboons have shown that duodenal switch procedure produces no change on gastric emptying, whereas antrectomy and the Rouxen-Y procedure do.

We do not agree with Fuchs et al.,46 who state that there is no place for duodenal diversion in patients with primary reflux disease caused by an incompetent sphincter. This is true in patients with erosive esophagitis in whom surgical augmentation of the lower esophageal sphincter can prevent acid reflux with a success rate of approximately 90%. In patients with Barrett's esophagus, alkaline reflux cannot be prevented completely after an antireflux procedure because some reflux always occurs. 27,28 In fact, it has been shown that antireflux surgery does not prevent the development of an adenocarcinoma in patients with Barrett's esophagus. 51-55 However, in the current study, we have proved for the first time that the duodenal diversion procedure can revert the presence of dysplasia in Barrett's epithelium, as occurred in three of our seven patients.

We have not seen this effect with chronic antireflux surgery (unpublished observation, 1997). This is an important observation and conclusion of our study. Ninety percent of the time, this surgery has produced good results. Ten percent of the time, it has failed because of the persistence of acid reflux, a condition that can be treated easily.

References

- 1. Ritchie NP. Alkaline reflux gastritis: late results on a controlled trial on diagnosis and treatment. Ann Surg 1986; 203:537-544.
- Malagelada JR, Phillips SF, Shoter RG, et al. Postoperative reflux gastritis: pathophysiology and long term outcome after Roux-en-Y diversion. Ann Intern Med 1985; 103:373-383.
- Du Plessis DJ. Pathogenesis of gastric ulceration. Lancet 1965; 1:974-978.
- 4. Warshaw AL. Bile gastritis without prior gastric surgery. Contributing role of cholecystectomy. Am J Surg 1979; 137:527-531.
- DeMeester TR, Fuchs KH, Ball CS, et al. Experimental and clinical results with proximal end-to-end duodenojejunostomy for pathologic duodeno gastric reflux. Ann Surg 1987; 206:414-426.
- Welch NT, Yasin A, Kin CHB, et al. Effect of duodenal switch procedure on gastric acid production, intragastric pH, gastric emptying and gastrointestinal hormones. Am J Surg 1992; 163:37-45.
- Mason RJ, Bremmer CC. Gastric emptying and acid output patterns following a suprapapillary duodeno jejunostomy reconstruction with different length of the afferent limb in the baboons. Am J Surg 1994; 167:322-326.

- Moffart RC, Berkas EM. Bile esophagitis. Arch Surg 1965; 91:963–966.
- Gillen P, Keeling P, Byrne PJ, Hennessy TPJ. Barrett's esophagus: pH profile. Br J Surg 1987; 74:774-776.
- Lillemoe KD, Johnson LJ, Harmon JW. Alkaline esophagitis: a comparison of the ability of components of gastroduodenal contents to injure the rabbit esophagus. Gastroenterology 1983; 85:621-628.
- 11. Mittal RK, Reuben A, Whitney JO, McCallum RW. Do bile acids reflux into the esophagus? Gastroenterology 1987; 92:371-375.
- Gillen P, Keeling P, Byrne PJ, et al. Implications of duodenogastric reflux in the pathogenesis of Barrett's esophagus. Br J Surg 1988; 75:540-543.
- Gotley DC, Morgan AP, Cooper MJ. Bile acid concentrations in the refluxate of patients with reflux esophagitis. Br J Surg 1988; 75:587-590.
- Waring JP, Legrand J, Chinicchien A, Sanouski RA. Duodenogastric reflux in patients with Barrett's esophagus. Dig Dis Sci 1990; 35:759-762.
- Gotley DC, Morgan AP, Ball D, et al. Composition of gastroesophageal refluxate. Gut 1991; 32:1093-1099.
- Attwood SEA, Ball CS, Barlow AP, Jenkison L. Role of intragastric and intraesophageal alkalinization in the genesis of complications in Barrett's columnar lined lower esophagus. Gut 1993; 34:11-15.
- Seto Y, Koboir O. Role of reflux esophagitis and acid in the development of columnar epithelium in the rat esophagus. Br J Surg 1993; 80:467-470.
- Bremmer CG, Mason RJ. Bile in the esophagus. Br J Surg 1993; 80:1374-1376.
- Champion G, Richters E, Valzi MF. Duodenogastroesophageal reflux: relationship to pH and importance in Barrett's esophagus. Gastroenterology 1994; 107:747-754.
- Kauer NKH, Burdiles P, Ireland AP, Clark GNB. Does duodenal juice reflux into the esophagus of patients with complicated GERD? Evaluation of a fiberoptic sensor for bilirubine. Am J Surg 1995; 169:98-104.
- Stein MJ, Feussen H. Alkaline gastroesophageal reflux. Dis Esoph 1994; 7:80–86.
- Braghetto I, González P, Csendes A, et al. The role of duodenogastric reflux in patients with different degrees of reflux esophagitis.
 Dis Esoph 1994; 7:99-102.
- 23. Gillen P, Keeling P, Byrne PJ, et al. Experimental columnar metaplasia in the canine esophagus. Br J Surg 1988; 75:113-115.
- Attwood SEA, DeMeester TR, Bremmer CG, et al. Alkaline gastroesophageal reflux: implications in the development of complications in Barrett's columnar-lined lower esophagus. Surgery 1989; 106:764-770.
- Attwood SEA, Smyrk TC, DeMeester TR. Duodenoesophageal reflux and the development of esophageal adenocarcinoma in rats. Surgery 1992; 111:503-510.
- Attwood SEA. Alkaline gastroesophageal reflux and esophageal carcinoma: experimental evidence and clinical implications. Dis Esoph 1994; 7:87-91.
- 27. De Meester TR, Bonarinal B, Albertucci M. Nissen fundoplication for gatroesophageal reflux disease. Ann Surg 1986; 204:9-20.
- Johansson J, Johnsson F, Joelsson B, et al. Outcome 5 years after 360° fundoplication for gastroesophageal reflux disease. Br J Surg 1993; 80:46-49.
- Iascone C, DeMeester TR, Little AG, Skinner DO. Barrett 's esophagus. Functional assessment proposed pathogenesis and surgical therapy. Arch Surg 1983; 118:543-549.
- Graziani L, De Nigio E, Pesaresi A, Baldell G. Reflux esophagitis: radiologic endoscopic correlation in 39 symptomatic cases. Gastroint Radiol 1983; 8:1-6.
- 31. Csendes A, Maluenda F, Braghetto I, Csendes P. Location of the lower esophageal sphincter and the squamous-columnar mucosal

- junction in 109 healthy controls and 778 patients with different degrees of endoscopic esophagitis. Gut 1993; 34:21-27.
- Csendes A, Braghetto I, Korn O, Cortes C. Late subjective and objective evaluation of antireflux surgery in patients with reflux esophagitis. Analysis of 25 patients. Surgery 1989; 105:374– 382.
- 33. Csendes A, Larrain E, Uribe P. Gastric acid secretion in patients with a symptomatic reflux and patients with esophageal strictures. Ann Surg 1974; 179:119-121.
- 34. Csendes A, González P, Olea E, Díaz JC. Gastric emptying of solids in patients with reflux esophagitis and peptic strictures of the esophagus compared to controls. In: Siewert JR, Holscher AH, eds. Diseases of the Esophagus. Berlin: Springer-Verlag 1987; 1047: 51.
- Csendes A, Oster M, Brodsbay O. Gastroesophageal sphincter pressure and serum gastrin in relation to food intake before and after vagotomy. Scand J Gastroenterol 1978; 13:437-441.
- DeMeester TR, Wang CI, Wernly JA. Technique, indications and clinical use of 24-h intraesophageal pH monitoring. J Thorac Cardiovasc Surg 1980; 79:656-670.
- 37. Csendes A, Alvarez F, Burdiles P, Braghetto I. Magnitude of gastroesophageal reflux measured by 24-h esophageal pH monitoring according to the degree of endoscopic esophagitis. Rev Med Chil 1994; 122:59-67.
- 38. Bechi P, Pucciani F, Baldini F, et al. Long-term ambulatoring enterogastric reflux monitoring. Validation of a new fiberoptic technique. Dig Dis Sci 1993; 38:1297-1306.
- Caldwell MTP, Bryne PJ, Brazil N, et al. An ambulatory bile reflux monitoring system. An in vitro appraisal. Physiol Meas 1994; 15:57-65.
- 40. Csendes A. A modified posterior cardiogastropexy for surgical treatment of gastroesophageal reflux with the adding of highly selective vagotomy and bougie calibration. In: Stipa S, Belsey R, Moraldi A, eds. Medical and Surgical Problems of the Esophagus. Symposium No° 43. London: Academic Press; 1981:91–95.
- 41. Oster MT, Csendes A, Funch-Jensen P, Amdrup E. PCV and modified Hill procedure as surgical treatment of reflux esophagitis: results in 108 patients. World J Surg 1982; 6:412-417.
- Csendes A. Highly selective vagotomy, posterior gastropexy and calibration of the cardia. In: Jamieson GJ, ed. Surgery of the Esophagus. Edinburg: Churchill Livingstone; 1988.
- Csendes A, Braghetto I, Korn O. Combined Operation. In: Pearson FG, ed. Esophageal Surgery. New York: Churchill Livingstone; 1995:361-369.
- 44. DeMeester TR, Attwood SEA, Smyrk TC. Surgical therapy in Barrett's esophagus. Ann Surg 1990; 212:528-542.
- Gastroduodenal function in Barrett's esophagus. Dis Esoph 1995; 8:205-210.
- 46. Fuchs KM, Freys SM, Heimbucher J, et al. Pathophysiologic spectrum in patients with gastroesophageal reflux disease in a surgical GI-function laboratory. Dis Esoph 1995; 8:211-217.
- Stein HJ, Hoeft S, DeMeester TR. Functional foregut abnormalities in Barrett's esophagus. J Thorac Cardiovas Surg 1993; 105:107– 111.
- Clark GNB. Barrett's esophagus; pathophysiological and management. In: Bremmer CG, DeMeester TR, Peracchia A, eds. Modern Approach to Benign Esophageal Disease. St. Louis: Quality Medical Publishing 1995; 69–82.
- Jazraw S, Walsh TN, Byrne PJ, Hill AD. Cholecystectomy and esophageal reflux: a prospective evaluation. Br J Surg 1993; 80:50-53.
- Lorurso D, Misciagna G, Mariquini V, et al. Duodenogastric reflux of bile acids, gastric and parietal cells and gastric acid secretion before and 6 months after cholecystectomy. Am J Surg 1990; 159:575-578.
- 51. Smith RRC, Hamilton SR, Boctwolt JK, Rogus EL. The spectrum

- of carcinoma arising in Barrett's esophagus: a clinicopathological study of 26 patients. Am J Surg Pathol 1984; 8:563-575.
- 52. Starnes VA, Adkins RB, Ballinger JF, Sauyers JC. Barrett's esophagus: a surgical entity. Arch Surg 1984; 119:563-562.
- Cameron AJ, Ott BJ, Payne WS. The incidence of adenocarcinoma in columnar lined (Barrett's) esophagus. N Engl J Med 1985; 313:857-859.
- 54. Haggrilt RC, Tryzelaar J, Ellis FX, Colcher M. Adenocarcinoma complicating columnar lined (Barrett's) esophagus. Am J Clin Pathol 1978; 70:1-5.
- Brand DL, Ylisaker JT, Gelfard M, Pope CE. Regression of columnar esophageal (Barrett's) epithelium after anti-reflux surgery. N Engl J Med 1980; 302:844-848.