Vagal-Sparing Esophagectomy: A More Physiologic Alternative

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Objective
To evaluate the function of the vagal nerves and the gastric reservoir after vagal-sparing esophagectomy.

Summary Background Data
Esophagectomy as currently performed includes division of the vagal nerves and surgical alteration of the stomach, with attendant postoperative dumping, diarrhea, reduced meal capacity, and weight loss. Vagal-sparing esophagectomy has been introduced as a technique for removal of the esophagus while preserving the vagal nerves and gastric reservoir. The procedure is touted as having a low morbidity and is applicable to patients with end-stage benign or early malignant disease.

Methods
A random sample of 15 patients at a median of 20 months after a vagal-sparing esophagectomy was compared to 23 asymptomatic normal subjects; 10 randomly selected patients, 29 months after esophagogastrectomy with colon interposition; and 10 randomly selected patients, 47 months after standard esophagectomy with gastric pull-up. Gastric mucosal acidification was tested with Congo red staining. Vagal secretory function was measured by gastric acid output and pancreatic polypeptide response to sham feeding. Vagal motor function was assessed by a technetium gastric emptying scan and a questionnaire to evaluate dumping and diarrhea. Gastric reservoir function was evaluated by measuring meal capacity and postoperative changes in body mass index.

Results
Vagal-sparing esophagectomy preserved the function of the vagi, as evident by an increase in gastric acid output, a rise in serum pancreatic polypeptide following sham feeding, and preservation of normal postoperative gastric emptying in 70% of the patients. After vagal-sparing esophagectomy, patients were free of dumping and diarrhea and were analogous to normal subjects in meal capacity but had a slight reduction in the speed of eating.

Conclusions
Vagal-sparing esophagectomy preserves gastric secretory, motor, and reservoir function. Postoperatively, patients have normal alimentation, bowel regulation, and no weight loss. It is an ideal procedure for patients with end-stage benign disease, Barrett’s esophagus with high-grade dysplasia, or esophageal carcinoma limited to the lamina propria.

Esophagectomy is often necessary in patients with irreversible esophageal damage from trauma, caustic agents, or chronic inflammation; end-stage motility disorders; Barrett’s esophagus with high-grade dysplasia; or esophageal carcinoma limited to the lamina propria. Benign end-stage and early malignant diseases of the esophagus have traditionally been treated by an esophagogastrectomy with colon interposition1–4 or a standard esophagectomy with gastric pull-up.5 Both procedures interrupt the vagi and alter the stomach, resulting in postoperative complications of dumping, diarrhea, early satiety, and weight loss. The acceptance of esophagectomy in benign and early malignant disease is often resisted because of these morbidities. Consequently,
several new therapeutic approaches have emerged such as mucosal ablation, endoscopic resection, and long-term stenting. The technique of vagal-sparing esophagectomy was developed to avoid the morbidities associated with standard esophagectomy by preserving the vagal nerves and stomach. The intent was to make esophagectomy a more acceptable therapy for end-stage benign and early malignant disease. Although conceptually appealing, preservation of vagal nerve integrity or the gastric reservoir function after vagal-sparing esophagectomy has not been validated, nor has the procedure been compared to esophagogastrectomy with colon interposition or a standard esophagectomy with gastric pull-up. This study evaluates the function of the vagal nerves and gastric reservoir after vagal-sparing esophagectomy. The results were compared to normal subjects and patients who had an esophagogastrectomy reconstructed with a colon interposition or a standard esophagectomy reconstructed with a gastric pull-up.

**METHODS**

**Surgical Technique**

Through an upper midline abdominal incision, the right and left vagal nerves are identified, circled with a tape, and retracted to the right. A limited, highly selective proximal gastric vagotomy is performed along the cephalad 4 cm of the lesser curve. The stomach is divided with a GIA stapler just below the gastroesophageal junction. The colon is prepared to provide an interposed segment as previously described. A neck incision is made along the anterior border of the left sternocleidomastoid muscle and the strap muscles are exposed. The omohyoid muscle is divided at its pulley and the sternohyoid and sternothyroid muscles are divided at

![Figure 1](image1.png)

*Figure 1.* Technique for vagal-sparing esophagectomy according to Akiyama et al. The esophagus is stripped out of the mediastinum and inverted in the process. This results in shearing the nerve fibers off the muscularis propria, leaving the esophageal plexus intact.

![Figure 2](image2.png)

*Figure 2.* Postmortem dissection of the esophagus, showing that the esophageal plexus can be easily freed from the muscular wall of the esophagus. The preservation of the esophageal plexus is the critical part of the vagal sparing procedure. (Courtesy Dr. D. Liebermann-Meffert.)

![Figure 3](image3.png)

*Figure 3.* Esophageal reconstruction with a colon interposition following a vagal-sparing esophagectomy. The high triangular posterior anastomosis of the stomach to the interposed colon is performed with staplers.
their manubrial insertion. The left carotid sheath is retracted laterally and the thyroid and trachea medially. The left inferior thyroid artery is ligated laterally as it passes under the left common carotid artery. The left recurrent laryngeal nerve is identified and protected. The esophagus is dissected out circumferentially in an inferior direction, from the left neck to the apex of the right chest, to avoid injury to the right recurrent laryngeal nerve. The esophagus is divided at the level of the thoracic inlet, leaving about 3 to 4 cm of cervical esophagus. The proximal esophagus is retracted anteriorly and to the right with the use of two sutures to keep saliva and oral contents from contaminating the neck wound.

Returning to the abdomen, the proximal staple line of the gastric division is opened and the esophagus is flushed with Betadine solution. A vein stripper is passed up the esophagus into the neck wound. The distal portion of the esophagus in the neck is secured tightly around the stripping cable with endo-loops and an umbilical tape for a trailer. The tip of the stripper is exchanged for a mushroom head, and the stripper is pulled back into the abdomen, inverting the esophagus as it transverses the posterior mediastinum (Fig. 1). This maneuver strips the branches of the esophageal plexus off the longitudinal muscle of the esophagus, preserving the esophageal plexus along with the proximal vagal nerves and the distal vagal nerve trunks (Fig. 2). In patients with end-stage achalasia only the mucosa is secured around the stripping cable so that it alone is stripped and the dilated muscular wall of the esophagus, with its enriched blood supply, remains. The resulting mediastinal tunnel, or in the case of achalasia muscular tube, is dilated with a Foley catheter containing 90 cc$^3$ of fluid in the balloon. The previously prepared interposed portion of the transverse colon is passed behind the stomach and up through the mediastinal tunnel into the neck. An end-to-end anastomosis is performed to the cervical esophagus using a single-layer technique. The colon is pulled taut and secured to the left crus with four or five interrupted sutures. Five centimeters below the crura, an opening is made in the mesentery adjacent to the colon along its mesenteric border through which a GIA stapler is passed and the colon is divided. The proximal end, which is the distal end of the interposed colon, is anastomosed high on the posterior fundic wall of the stomach using a triangular stapling anastomotic technique. This is done by stapling longitudinally the stomach and colon together with a 75-mm GIA stapler, spreading the base of the incision apart, and closing it with a T-55 stapler. Colonic continuity is reestablished by bringing the proximal

Table 1. UNDERLYING DISEASE IN PATIENTS UNDERGOING ESOPHAGECTOMY

<table>
<thead>
<tr>
<th>Underlying Disease</th>
<th>Vagal-Sparing</th>
<th>Colonic Interposition</th>
<th>Gastric Pull-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-stage GERD</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>End-stage Achalasia</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Giant esophageal diverticulum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extensive lye stricture</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Giant leiomyoma</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Esophageal perforation</td>
<td>7</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Barrett’s high-grade dysplasia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intramucosal carcinoma</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Advanced carcinoma</td>
<td></td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
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right colon to the distal staple line in the left colon and performing an end-to-end anastomosis using a double-layer technique. The reconstruction is shown in Figure 3.

**Study Population**

The study population consisted of 15 patients who had a vagal-sparing esophagectomy, 10 patients who had an esophagogastrectomy with a colon interposition, and 10 patients who had a standard esophagectomy with a gastric pull-up (Fig. 4). They were randomly selected from the patient pool treated between 1992 to 2000 by the Division of Foregut Surgery in the Department of Surgery of the University of Southern California Keck School of Medicine. The underlying disease for which the esophagectomy was performed is shown in Table 1. Patient demographics are shown in Table 2. The median follow-up for vagal-sparing esophagectomy patients was 20 (range 6–79) months, for esophagogastrectomy with colon interposition 29 (range 6–65) months, and for esophagectomy with gastric pull-up 47 (14–72) months. All patients were tested to determine the integrity of the vagal nerves and gastric reservoir function. Twenty-three asymptomatic normal subjects served as controls. The ability of the gastric mucosa to produce acid was determined by Congo red staining. Vagal secretory function was tested by measuring the increase in gastric acid output and the rise in serum pancreatic polypeptide level in response to sham feeding. Vagal motor function was assessed by a technetium gastric emptying scan and a questionnaire to evaluate dumping and diarrhea. Gastric reservoir function was tested by measuring meal capacity and postoperative changes in body mass index (BMI in kg/m²). The study was approved by the Institutional Review Board of the University of Southern California Keck School of Medicine.

**Tests of Secretory Function**

**Upper Endoscopy With Congo Red Staining**

Upper gastrointestinal endoscopy was performed after an overnight fast following 1 day of a liquid diet in all normal

<table>
<thead>
<tr>
<th>Normal Subjects</th>
<th>Vagal-Sparing</th>
<th>Colonic Interposition</th>
<th>Gastric Pull-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male:female</td>
<td>14:9</td>
<td>13:2</td>
<td>6:4</td>
</tr>
<tr>
<td>Median age (range)</td>
<td>35 (20–45)</td>
<td>61 (21–76)</td>
<td>52 (28–72)</td>
</tr>
</tbody>
</table>

Figure 5. Response of gastric acid output to a sham meal. Values presented are the sham/basal acid output ratio.
subjects and patients. Antacids, acid suppression, and anti-
cholinergic medications were discontinued before the study
according to the following schedule: antacids −24 hours,
histamine receptor antagonists −72 hours, proton pump
inhibitors −14 days, and anticholinergic agents −72 hours.
The mucosal lining of the esophagus, stomach, and inter-
posed colon, when applicable, was examined and the ability
of the gastric mucosa to produce acid was determined by
Congo red staining.\textsuperscript{18−20} Congo red is a pH indicator that
changes from red to black in contact with acid. Before the
application of the stain all fluid in the stomach was aspirated
and a spray catheter was passed through the biopsy channel
of the endoscope. One hundred eighty milliliters of a solu-
tion of 0.5% NaHCO\textsubscript{3} and 0.6% Congo red was sprayed
over the gastric mucosa. Residual fluid was then aspirated
and a mucosal map was made of the black-stained areas
representing acid production.

\textit{Basal and Sham Meal-Stimulated Acid Output
Studies}

All normal subjects and patients were assessed for their
ability to increase acid output in response to a sham feeding.

After an overnight fast, a nasogastric tube was placed in the
stomach with endoscopic guidance at the time of endos-
copy. The basal acid output (BAO) was determined by
aspirating gastric contents for one hour before the sham
meal, consisting of a 4-oz hamburger patty and a regular
portion of French fries. Patients were instructed to chew the
food thoroughly and to expectorate it. No water was allowed
during the feeding. Following the sham meal, gastric juice
was aspirated in two separate 30-minute aliquots for a total
of 1 hour. Each aliquot was analyzed separately for the
volume of gastric juice aspirated, pH, and titratable acidity
to pH 7 with 0.1 mol/L NaOH. BAO was expressed as
millimoles per liter. Sham acid output (SAO) was calculated
by the summing the two 30-minute samples following the
sham meal and was also expressed as millimoles per liter.
The ratio of SAO to BAO was calculated.

\textit{Basal and Sham Meal-Stimulated Pancreatic
Polypeptide Response}

The rise in serum pancreatic polypeptide level in re-
sponse to a sham feeding was measured in all normal
subjects and patients by obtaining two blood samples sep-
arated by 30 minutes before and at 15-minute intervals for 1 hour after a sham feeding. The blood samples were collected in chilled tubes containing EDTA and aprotinin. The samples were centrifuged at 3,000 rpm for 10 minutes. The plasma was then decanted and stored frozen at −70°C. Pancreatic polypeptide was measured using a radioimmunoassay as previously described.29

Tests of Motor Function

Gastric Emptying Scan

Gastric emptying was measured in all normal subjects and patients who had a vagal-sparing esophagectomy using a radiolabeled test meal consisting of 1 mCi technetium-99 sulfur colloid readily mixed with a cooked egg. The test was not performed in patients who had an esophagogastrectomy with colon interposition or a standard esophagectomy with a gastric pull-up because the surgical deformity to the stomach added an additional variable to the emptying process. After an overnight fast, the egg, between two slices of bread, was ingested. Upright dynamic planar images of the chest were performed to assess esophageal or conduit emptying and to map the region of the stomach for the analysis of gastric emptying. The subject was then placed in a semirecumbent position and further dynamic anterior planar imaging of the abdomen was performed every 15 minutes for a total of 180 minutes. Activity curves for gastric emptying were generated. Gastric retention was expressed as the percentage of the initial maximum count of the region of interest plotted against time. The half emptying time (t1/2) was calculated as the time in minutes when the gastric activity was reduced to 50% of the initial maximum count.

Symptomatic Assessment of Dumping, Diarrhea, and Early Satiety

A standardized questionnaire was used to evaluate the prevalence of dumping, diarrhea, and early satiety after esophagectomy. Dumping was identified if any of the following symptoms were present after eating and required treatment: palpitations, pallor, abdominal cramps, weakness, sweating, dizziness, and the need to lie down. Diarrhea was defined as postoperative new-onset change in bowel habit resulting in two or more loose or watery stools, with or without postprandial abdominal cramps or urgency. Early satiety was defined by the need to stop eating due to a full sensation in the epigastrium.

Tests of Reservoir Function

Meal Capacity and Speed of Eating

The eating capacity and speed of eating were measured in all normal subjects and patients using a standard test meal consisting of one cup of macaroni and cheese (228 calories), a half-cup of green beans (22 calories), one bread roll (240
calories), one teaspoon of margarine (45 calories), two peach halves (60 calories), and one piece of pound cake (80 calories). The total caloric content of the meal was 675 calories. The patients were instructed to consume the meal in their usual fashion and were allowed to drink water as desired. The total calories consumed and time of consumption were recorded. From these data, the meal capacity, defined as the percentage of total calories consumed, and the speed of eating, defined as calories consumed per minute, were calculated.

BMI
BMI was calculated by kilograms of body weight divided by height in meters squared.

Statistics
Disease characteristics were summarized by medians for the continuous variables and by percentages for the discrete variables. Statistical analysis was performed using the Mann-Whitney test to compare continuous variables and the chi-square or Fisher exact test when appropriate to compare discrete variables. Significance between the values of four independent groups was determined by the Kruskal-Wallis test. *P < .05 was considered to indicate statistical significance. Statistical analyses were made with the help of SPSS for Windows computer software (SPSS Inc., Chicago, IL).

RESULTS
Secretory Function
Gastric mucosal acidification was identified by Congo red staining in 95% of normal subjects, 86% of patients who

| Table 3. PREVALENCE OF DUMPING AND DIARRHEA FOLLOWING VARIOUS FORMS OF ESOPHAGECTOMY |
|---------------------------------|-----------------|-----------------|
|                                  | Vagal-Sparing   | Colon Interposition | Gastric Pull-Up |
| Dumping                         | 1/15            | 3/10*              | 1/10            |
| Diarrhea                        | 0/15            | 8/10*              | 5/10*           |

* *P < .05 versus vagal-sparing esophagectomy.
had a vagal-sparing esophagectomy, 20% of patients who had an esophagogastrectomy with a colon interposition, and 70% of patients who had a standard esophagectomy with a gastric pull-up.

The ability of the gastric mucosa to secrete acid in response to a sham feeding is shown in Figure 5. The distribution of the ratio of SAO over BAO was similar in normal subjects and patients who had a vagal-sparing esophagectomy. With the exception of 2 of 10 patients, gastric acid output was nonexistent after esophagogastrectomy with colon interposition. The rise in gastric acid over BAO was present but diminished in patients who had a standard esophagectomy with gastric pull-up.

The increase in the pancreatic polypeptide serum level following a sham feeding is shown in Figure 6. At 30 minutes after the meal there was a statistical rise ($P < .05$) in the serum pancreatic polypeptide level in normal subjects and patients who had a vagal-sparing esophagectomy. This rise was not seen in the patients who had an esophagogastrectomy with a colon interposition or standard esophagectomy with a gastric pull-up.

Motor Function

Figure 7 shows the individual gastric emptying curves of patients who had a vagal-sparing esophagectomy. Eleven of the 15 patients (73%) had what appeared to be normal gastric emptying and 4 had a nearly flat curve, indicating delayed gastric emptying. One of the four patients with delayed gastric emptying had a previous Nissen fundoplication, another had three photodynamic therapy sessions for high-grade dysplasia in Barrett’s esophagus, and a third had severe mediastinal fibrosis from chronic longstanding reflux disease.

Figure 8 shows the collective gastric emptying curves for normal subjects and patients who had a vagal-sparing esophagectomy. The mean and median t1/2 emptying times in patients who had a vagal-sparing esophagectomy were 52 and 41 minutes, respectively. This was similar to the mean and median t1/2 emptying times of normal subjects (44 and 31 minutes, $P = .141$). However, patients who had a vagal-sparing esophagectomy emptied slower during the latter half of the study. To determine if this was due to the
influence of the 4 patients who had delayed gastric emptying, a second plot was made of the 11 vagal-sparing esophagectomy patients with normal gastric emptying and compared to normal subjects. The two curves were nearly identical.

The prevalence of dumping and diarrhea after esophagectomy is shown in Table 3. Diarrhea did not occur in vagal-sparing esophagectomy patients and was significantly more common in patients who had an esophagogastrectomy with colon interposition or standard esophagectomy with gastric pull-up ($P < 0.005$). Dumping was seen in only one patient following vagal-sparing esophagectomy and was significantly less than in patients who had an esophagogastrectomy with colon interposition ($P = 0.002$) but similar to patients who had a standard esophagectomy and gastric pull-up.

**Reservoir Function**

The meal capacity for normal subjects and that for the esophagectomy patients are shown in Figure 9. Patients with a vagal-sparing esophagectomy had a meal capacity similar to normal subjects and significantly better than patients who had an esophagogastrectomy with colon interposition or a standard esophagectomy with gastric pull-up. Figure 10 shows the speed of eating in calories consumed per minute in normal subjects and esophagectomy patients. All patients ate at a similar rate and were slower than normal subjects.

Figure 11 shows the BMI measured preoperatively and at the last follow-up visit. There was no change in BMI in patients who had a vagal-sparing esophagectomy. The BMI decreased statistically in patients who had an esophagogastrectomy with colon interposition or standard esophagectomy with gastric pull-up. When asked, 3 of 15 patients who had a vagal-sparing esophagectomy, 3 of 10 who had an esophagogastrectomy with colon interposition, and 3 of 10 who had a standard esophagectomy with gastric pull-up reported early satiety.

**DISCUSSION**

This study shows that vagal-sparing esophagectomy preserves gastric secretion, gastric emptying, meal capacity, and BMI when compared to esophagogastrectomy with colon interposition or standard esophagectomy with gastric pull-up.
pull-up. In fact, vagal-sparing esophagectomy patients functioned for the most part similarly to normal subjects; the procedure allowed them to eat a normal meal, free of dumping or diarrhea. These results indicate that the vagal-sparing esophagectomy procedure does indeed preserve the vagal nerves. The strongest evidence for postoperative vagal nerve integrity was the rise in the serum pancreatic polypeptide level following sham feeding. A positive response to this test is independent of end points based on gastric physiology or the capacity of the gastric mucosa to secrete acid. The preservation of an unaltered stomach in patients with vagal-sparing esophagectomy allows for a normal meal capacity that empties properly. This capacity to ingest a normal meal is reflected in the patients’ ability to maintain their BMI. The only difference observed in patients who had vagal-sparing esophagectomy when compared to normal subjects was the speed with which they ate. Eating was slower in all patients who had an esophagectomy, as would be expected with removal of the esophagus.

The clinical benefit of preserving the vagal nerves was the absence of postoperative diarrhea and the presence of dumping in only 1 of 15 patients (7%). This was statistically less than patients who had an esophagogastrectomy with colon interposition but similar to patients who had a standard esophagectomy with gastric pull-p. The 10% rate of dumping seen in our patients who had a standard esophagectomy with gastric pull-up is below the 18% to 50% reported in larger series and probably reflects a selection bias.30–33

The gastric emptying studies showed that four patients had delayed gastric emptying following vagal-sparing esophagectomy. In these patients it is likely that some portion of the vagal nerves was inadvertently removed or damaged at the time of the procedure. This is supported by the observation that when these 4 patients were removed from the cumulative gastric emptying curve, the emptying of the remaining 11 patients was identical to normal subjects. Three of the four patients with delayed gastric emptying had findings that may have compromised the ability to strip the esophagus out of the mediastinum without damaging the vagus nerves. One had a previous Nissen fundoplication that encased the vagal trunks, another had three applications of photodynamic therapy that may have fibrosed the esophageal vagal plexus to the esophagus, and a third had extensive mediastinal fibrosis from long-standing reflux disease. In two of the four patients the delayed gastric emptying required a proximal gastric resection with the anastomosis of the colon to the antrum to alleviate their symptoms. Thus, in some patients with benign disease, it may be difficult to strip the esophageal vagal plexus from the esophageal muscle, and a second procedure may be required to remove the adynamic gastric fundus and body. Consequently, our experience encourages the use of vagal-
sparing esophagectomy earlier in end-stage disease processes, before other multiple therapeutic efforts have compromised the ability to strip the esophagus from the vagal nerves.

The findings in this study make vagal-sparing esophagectomy a particularly applicable surgical procedure for patients with end-stage esophageal disease of motor or inflammatory etiology, Barrett’s esophagus complicated by high dysplasia, or multifocal esophageal carcinoma limited to the lamina propria. The reasons are that no mediastinal dissection is required, the diseased esophagus is removed preserving the vagal nerves, the stomach is unaltered and retained within the abdomen, and no gastric drainage procedure is required. The combination of these factors results in minimal disruption of gastrointestinal function.

Some may question the wisdom of preserving the vagal nerves in patients with early malignant disease. Previous studies performed by us and others have shown that when invasion of an esophageal cancer is limited to the lamina propria, lymph node metastasis is rare,34–36 obviating the need for lymphadenectomy. Ideally this degree of disease can be managed by endoscopic mucosal resection, provided there is only one focus of disease. This approach is less applicable and potentially dangerous when multifocal cancers are present. In this situation vagal-sparing esophagectomy provides a better option. Vagal-sparing esophagectomy is ideally suited in patients with Barrett’s esophagus and high-grade dysplasia. It removes the whole esophagus, as opposed to mucosal ablation procedures that attempt to destroy only the mucosa but are troubled with squamous overgrowth of retained segments of Barrett’s mucosa, requiring continued surveillance of the esophageal mucosa after the procedure. The major impetus for the use of mucosal ablation procedures is the morbidity and mortality of esophagectomy. Our study shows that vagal-sparing esophagectomy has limited morbidity, and in our experience to date with 78 procedures, there has been no mortality. Consequently the decision to perform a vagal-sparing esophagectomy is less arduous than for a standard esophagectomy. This makes vagal-sparing esophagectomy a more acceptable alternative to a mucosal ablation procedure.

In conclusion, the results of this study confirm the ability to perform a vagal-sparing esophagectomy and encourage its use in patients with end-stage benign esophageal disease, Barrett’s esophagus with high-grade dysplasia, or multifocal esophageal cancer limited to the lamina propria. This should be done before other therapeutic efforts have limited the ability to perform the operation. The benefits of a vagal-sparing esophagectomy are removal of the diseased esophagus while maintaining gastrointestinal innervation, normal meal capacity, and unaltered gastric emptying. This allows normal alimentation and normal nutrition. Its only limitation is a slightly reduced speed of eating.

References


31. Dr. RAHIM MOOSA (San Diego, CA): I enjoyed both the presentation and the manuscript. Historically and ironically, both Dr. Tom DeMeester and the late Dr. Dr. DeMeester spent a major part of their careers at the University of Chicago. More than half a century ago, Dr. Dr. DeMeester advocated the liberal use of vagotomy for the relief of various acid-peptic disorders. As a result of this work, he was the first recipient of the Medallion for Scientific Achievement awarded by this Association in 1970.

32. The past 30 years have witnessed a continuous trend of decreasing vagotomy for the relief of various acid-peptic disorders. As a result of this work, he was the first recipient of the Medallion for Scientific Achievement awarded by this Association in 1970.


DISCUSSION

DR. A. RAHIM MOOSA (San Diego, CA): I enjoyed both the presentation and the manuscript. Historically and ironically, both Dr. Tom DeMeester and the late Dr. Dragstedt spent a major part of their careers at the University of Chicago. More than half a century ago, Dr. Dragstedt advocated the liberal use of vagotomy for the relief of various acid-peptic disorders. As a result of this work, he was the first recipient of the Medallion for Scientific Achievement awarded by this Association in 1970.

The past 30 years have witnessed a continuous trend of decreasing vagotomy through the selective and highly selective modifications. The subsequent advent of H2 receptor antagonists and proton pump inhibitors rendered the operation of vagotomy largely obsolete. Until now, the only remaining situation whereby a surgeon could vagotomize a patient with impunity and a clear conscience was a total esophagectomy. Dr. DeMeester’s group have just given the coup de grace to Dragstedt’s operation by the impressive data presented.

I have three questions and one minor disagreement. How many of these vagus-sparing esophagectomies have you now performed, and have you had occasion to inadvertently traumatize the vagal trunks during the blunt esophageal stripping? Have you ever needed to resort to an emergency thoracotomy for intraoperative technical problems such as bleeding or inability to strip the esophagus? Have you ever had to reoperate at a later date for delayed gastric emptying and gastrocolic reflux?

My disagreement centers around the use of the term “sham feeding,” which was coined by Pavlov more than a century ago. He rang a bell each time he fed his dogs and showed that the gastric pouches secreted acid in response to feeding. He later just rang the bell without feeding and the pouches still responded. He referred to this as a response to “sham feeding,” which was subsequently abolished by a vagotomy. But this is just a semantic point.

PRESENTER DR. TOM R. DEMEESTER (Los Angeles, CA): Thank you, Dr. Moossa. As of to date, we have done 78 vagal-sparing esophagectomies, and we feel quite comfortable with the technique that has evolved. We had delayed gastric emptying develop in four patients a year or two after the procedure that required surgical intervention. So there are situations when you are unable to preserve the vagal nerves due to existing mediastinal pathology secondary to the underlying disease process.

We have not had to do an emergency thoracotomy for bleeding. This has not been an issue. On occasion, irrigation of the mediastinal tunnel with a diluted solution of epinephrine may be necessary to stop persistent capillary ooze.

DR. JOHN G. HUNTER (Portland, OR): I really liked the study for many reasons. Generally it was quite convincing that vagal-sparing esophagectomy leaves two functioning vagi intact. This really does make a difference. And that difference can be measured physiologically and clinically. I have several questions.

The first is: How were your patients chosen? Obviously your whole group was not assayed. I gather they were not randomized either. Were they demographically similar between the three groups that you studied?

Second question: Are these groups really comparable? The tests for vagal function are strongly influenced by the status of the stomach, and the stomach was a different size or in a different position in each group. As I understand it, the standard colon in your positioned patients had undergone a near-total gastrectomy as part of the operation, which could explain many of the physiologic changes observed regardless of the status of the vagus.

In the manuscript you described using a Foley catheter to dilate the tunnel. Exactly how was that done? When the vagus is injured during stripping, can you detect it intraoperatively? And if so, should you then do your proximal gastrectomy? Should you do a pyloroplasty?

Lastly, working at Emory with Joe Miller and Kamal Monsour, I became more enamored of the gastric pullup than the colon interposition because of the other GI side effects. You don’t really tell us much about that. Do these patients regurgitate? What about nausea, vomiting, chest pain, bile reflux, and dysphagia? Were they higher in either of these groups than in others?

DR. TOM R. DEMEESTER (Los Angeles, CA): We randomly selected from our data bank a group of patients who had a vagal-sparing esophagectomy, called them, and asked if they would take part in the study. Although there is always a potential for a bias, it is unlikely to have occurred in that four of the patients selected who had a vagal-sparing esophagectomy had delayed gastric emptying.

We concur that test of vagal integrity can be misleading. That is why we performed three tests. The test for acid secretion is the most questionable. Our approach was to demonstrate the presence of acid-secreting mucosa with Congo red staining and then determine if the mucosa would respond to sham feeding. We went a step further to measure the secretion of pancreatic polypeptide in response to sham feeding. This test is not dependent upon having acid-secreting mucosa. Last of all, we evaluated gastric emptying, which in this setting is quite specific for vagal integrity.

Gasric emptying following vagal-sparing esophagectomy was compared only to normal subjects because alterations to the stomach affect gastric emptying with the other two procedures. The evaluation of meal capacity, I think, was applicable to all the patients and shows the effect on reservoir capacity when the stomach is surgically altered or repositioned.

Dilation of the tunnel is done just to make sure there was sufficient room to bring the colon up without compromising its blood supply. A Foley catheter inflated with about 90 cc of fluid was used for dilation.

You may get some suggestion that you failed to preserve the esophageal plexus at the time of the operation by inspecting the specimen for nerve fibers, but you are never completely sure. It is our impression that if in doubt, a clinical trial is reasonable. If after a period of evaluation the stomach doesn’t work, a second remedial operation to remove the proximal stomach and connect the colon to the antrum can be done.

We focused only on the symptoms of dumping and diarrhea because of their association with vagal disruption, but I can state that the patients who have had a vagal-sparing esophagectomy are dramatically different in terms of recovery from surgery and their ability to alimentate after surgery when compared to those who had a standard esophagectomy with a gastric pull-up or an esophagogastrectomy with a colon interposition. They occasionally have regurgitation at night, and most sleep with two pillows or...
Their postoperative recovery is rapid and less eventful, and this has impressed me that taking the vagal nerves does affect the organism response to injury and recovery in ways we have not appreciated.

**DR. ANTOON E. M. R. LERUT (Leuven, Belgium):** I very much enjoyed this very elegant study indeed. I have two remarks.

One relates to the use of the colon. Obviously, it seems that you are systematically using a long-segment colon with anastomosis of the neck. And I have some concern about this. As you know, I had the opportunity to review a couple of years ago, together with JCM, Mr. Banki’s material on coloplasty on the very long-term follow-up of 20 to 30 or more years, and it appears that in the very long-term follow-up there is about 15% of the patients that come up with functional disorders. So that brings me to the question whether you need to resect always, to do always a subtotal hepatectomy or whether you can perform an encaderasic anastomosis and whether you always need to use the colon versus, for instance, what Devis could be doing, the jejuno.

My second remark is, as I had the privilege to read the manuscript, I would like to ask you a question on the development of the vagal nerve in one patient who had had three times photodynamic therapy, where you mentioned that that was possibly a reason for fibrosis. I have had a couple of occasions to operate on such patients, and I was surprised to see how little damage there was during the operation of the external side.

So I wonder whether you have evidence that there is indeed intramural fibrosis and through the intramural fibrosis, the fibrosis of the vagus nerve. Because that might be relevant as to both the difficulty of the follow-up of hidea dysplasia and the technical difficulties if you treat a patient with such a treatment when they have to come for surgery.

**DR. TOM R. DEMEESTER (Los Angeles, CA):** The question was, when do we use the colon for reconstruction? The use of the colon is based on the extent of disease present. If the tumor extends into the stomach beyond where a distal margin can be obtained with confidence, we will use the colon for reconstruction. In patients with the potential for long-term survival, we tend to use the colon to avoid recurrence of the disease in the cervical esophagus. We have an incidence of recurrent Barrett’s in the cervical esophagus in about 14% of patients 3 or more years after standard esophagectomy with gastric pull-up. In one patient, we had a recurrent carcinoma. The anastomosis of acid-secreting gastric mucosa to squamous mucosa of the cervical esophagus sets the stage for recurrent reflux disease. This is most likely to be seen in patients who are blessed with long-term survival. We haven’t seen changes in the cervical esophagus following a colon interposition.

There was a question regarding using a short colon interposition. We have been disappointed with the short colon. It gives more problems in alimentation, requires an intrathoracic anastomosis, less protection against reflux into the retained esophagus, and a shorter surgical margin for a tumor that extends proximally in the submucosal lymphatics.

In regards to the question about photodynamic therapy (PDT), we had one patient who had three PDT sessions to ablate an area of high-grade dysplasia, and all failed. Eventually a vagal-sparing esophagectomy was performed. Despite repetitive PDT treatment, the specimen contained three intramucosal carcinomas. In this patient, the esophageal plexus was adherent to the esophagus. You could see branches of the plexus on the surface of the esophagus when it was removed. This patient developed delayed gastric emptying 6 to 8 months after the operation, but his symptoms are not severe enough to recommend a proximal gastrectomy. So, I suspect that PDT causes inflammation and fibrosis that extends through the wall of the esophagus, causing adherence of the esophageal plexus to the esophageal muscle. Normally when you remove the esophagus, there shouldn’t be any nerve fibers on its surface.

I would like to close by emphasizing that this study could not have been accomplished without the enormous energy that Dr. Farzaneh Banki, our resident, put into the project. She literally spent night and day getting these patients back from all parts of the country and hand-holding them through their studies. It just couldn’t have been done without her, and I appreciate her commitment to the study. Thank you.