GENERAL THORACIC SURGERY

ESOPHAGECTOMY WITH GASTRIC RECONSTRUCTION FOR ACHALASIA

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Purpose: Achalasia is a degenerative esophageal disorder that may result in esophageal failure necessitating resection for restoration of gastrointestinal function. This study evaluates a protocol of esophageal resection and gastric reconstruction for end-stage achalasia. **Methods:** Hospital records, radiographic studies, and resection specimens of patients undergoing esophagectomy and gastric reconstruction were reviewed. Patient outcome was defined by an evaluation of symptoms (early satiety, dysphagia, regurgitation, and reflux), dietary restrictions, and ability to maintain or gain weight. Preoperative, operative, and postoperative variables and pathologic features in the resection specimens were analyzed to determine predictors of outcome. **Results:** In a 10-year period, 32 patients underwent esophagectomy with gastric reconstruction for achalasia; 30 (94%) underwent elective surgery and 2 (6%), emergency surgery. No postoperative deaths occurred. Of 29 patients completing telephone interviews, 24 (83%) had no or mild dysphagia; 21 (72%), no or mild regurgitation; 20 (69%), no or mild reflux; and 19 (66%), no or mild early satiety. Twenty-four (83%) patients had no or minimal dietary restrictions; 26 (90%) had no or minimal social dietary restrictions. Postoperative weight was not different from preoperative weight. Of 30 patients, 26 (87%) felt better after esophagectomy and 25 (83%) would have the operation again. There were few predictors of outcome. Younger patients were more likely to have dysphagia ($P = .03$). **Conclusions:** Esophagectomy with gastric reconstruction relieves preoperative dysphasia and regurgitation in the majority of patients. Dietary function and weight maintenance are excellent, attesting to the durability of the procedure in patients with end-stage achalasia. (J Thorac Cardiovasc Surg 1999;117:1077-85)

Achalasia is a degenerative esophageal disease caused by destruction of the myenteric plexus, resulting in aperistalsis of the esophageal body and

abnormal relaxation of the lower esophageal sphincter. Early disease without marked esophageal dilatation can be treated by reducing lower esophageal sphincter pressure by either pneumatic dilation or surgical myotomy. In the absence of therapy, or if therapy is inadequate or complicated by acid reflux, there is progressive dilatation and eventually marked tortuosity of the esophagus, resulting in the condition known as *end-stage achalasia*. The only solution for end-stage esophageal failure is resection of the obstructed, dilated sigmoid esophagus. Colon interposition traditionally has been used to restore gastrointestinal continuity for benign disease. However, the successful use of the stomach for gastrointestinal reconstruction in malignant esophageal tumors has led to its consideration for benign end-stage esophageal diseases.

The purpose of this study was to evaluate a protocol

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Table I. Symptoms, diet, therapy, and complications before esophagectomy

	No.	%
Preoperative symptoms		
Dysphagia	30	94
Regurgitation	29	91
Chest pain	11	34
Reflux	7	22
Preoperative dietary restrictions		
None	13	43
Selected solids	6	19
No solids	9	28
Nothing by mouth	4	13
Prior therapy		
Pneumatic dilation	25	79
Botox injection	2	6
Chronic self-dilation	1	3
Heller myotomy	16	50
Repeat myotomy	4	12
Fundoplication	3	9
Epiphrenic diverticulectomy	1	3
Gastrostomy or nasogastric feeding	2	6
Total parenteral nutrition	2	6
Preoperative complications		
Weight loss (median 11 kg, range 2-20 kg)	17	53
Aspiration pneumonia	7	22
Esophageal bleeding	3	9
Postoperative leak and abscess	3	9
Pneumatic dilation perforation	1	3
Respiratory failure	2	6

of esophageal resection and gastric reconstruction for end-stage achalasia.

Patients and methods

Protocol and variables. In 1988, a treatment protocol of esophagectomy via the transhiatal route, gastric reconstruction, cervical anastomosis, and pyloromyotomy was adopted as the treatment of choice, where possible, for benign end-stage esophageal diseases. A prospective pathology registry was used to record patients with benign esophageal disease who had esophagectomy. It was used to identify and retrieve for review the medical records of all patients with achalasia undergoing esophagectomy with gastric reconstruction through December 31, 1997. Preoperative variables, date and details of esophagectomy, reconstruction, and postoperative course were abstracted. Preoperative radiographic studies including chest radiogram, barium esophagogram, and chest computed tomographic scan were reviewed and maximum diameter of the esophagus and width of the sigmoid segment were measured.

Pathology review. The esophageal portion of the resection specimens was evaluated and the following features were recorded: (1) presence or absence of ganglion cells and the

number of ganglion cells; (2) presence or absence of myenteric neuritis, defined as inflammation within or immediately around myenteric nerves, graded as mild, moderate, or severe; and (3) presence or absence of myenteric neural fibrosis evaluated by the Masson trichrome stain, graded as mild, moderate, or severe.^{1,2} In addition, the proximal esophageal margin and the gastric cardia were evaluated, and the presence of ganglion cells was recorded.

Outcome. Weight measured at each postoperative visit was obtained from medical records. Patients were contacted by telephone and completed a questionnaire to define outcome and gastrointestinal function. Symptom scores of 0 (absent), 1 (mild), 2 (moderate), 3 (severe), and 4 (incapacitating) were used at the time of follow-up to assess abnormal gastrointestinal function as described by dysphagia, early satiety, regurgitation, and acid reflux. Medications and procedures required to treat these symptoms were recorded. Dietary restrictions and the inability to eat at social events (social dietary restrictions) were also scored from 0 to 4. Patients were asked if they felt better after esophagectomy and whether they would undergo esophagectomy again. Complete symptom follow-up was obtained in all but 3 patients: 1 died, 1 refused to be interviewed, and 1 agreed to a partial interview.

Statistical review. Symptom scores were obtained at only one point in time for each patient. Ideally, these scores would be ascertained at multiple points across time for each patient. Because patients were treated over a 10-year period, we have used longitudinal data analysis of these single assessments to estimate the time-relatedness of symptom scores. This longitudinal data analysis used logistic regression analysis, with time interval since surgery used to assess temporal trends.

For each symptom, a sequential data analysis was done of preoperative variables, pathologic findings, operative techniques, and in-hospital complications. Preoperative variables included gender, age at operation, symptoms, duration of symptoms, dietary restrictions, prior therapy, complications of achalasia or therapy, maximum diameter of the esophagus, and maximum sigmoid width of the esophagus. Operative variables included surgical approach (transhiatal or thoracotomy), conversion of transhiatal approach to thoracotomy, variations from the treatment protocol, and whether the operation was elective or urgent. Postoperative variables included complications, need for blood transfusion, and intensive care unit and hospital lengths of stay. Pathologic variables included presence of ganglion cells, myenteric neuritis, eosinophils and granulomatous inflammation in the resection specimen, and presence of ganglion cells in the proximal esophagus and gastric cardia.

Although the scores were expressed on an ordered scale, few patients had symptom scores above 1. Thus scores were dichotomized in two ways: 0 versus 1 to 4, and 0 and 1 versus 2, 3, and 4 for separate analysis. The two results were consistent and, therefore, only the latter set is presented. Because the data set is small, assessment of symptom status used univariable statistics rather than multivariable statistics. Odds ratios, 95% confidence intervals for the odds ratios, and

Fig 1. **A**, Chest x-ray film shows a mass in the posterior mediastinum (*arrows*). **B**, Barium esophagogram confirms a very dilated sigmoid esophagus. **C**, Computed tomographic scan without oral contrast material again demonstrates the dilated fluid-filled esophagus.

the *P* values were obtained from the logistic regression model. McNemar's test was used to determine whether symptom frequency changed after surgery.

Unlike symptom scores that were available at only one time for each patient, weight was measured before the operation and at each postoperative visit. A regression of weight versus time after the operation was calculated for each patient; slopes were obtained from the regression models. The slopes were averaged, and a 1-sample *t* test was used to determine whether the slopes differed significantly from 0. This is equivalent to a longitudinal data analysis of repeated measures. In addition, change in weight from preoperative to follow-up was calculated. An analysis of variance model was used to determine whether weight change was associated with age at operation, gender, or length of follow-up.

Results

Patients and variables. Over a 10-year period, 32 patients with achalasia were treated by esophagectomy with gastric reconstruction. During this time, 1 additional patient with end-stage achalasia and a previous gastric resection had esophagectomy with colon interposition and is not included in this analysis. Seventeen (53%) patients were women. Median age at operation was 48 years (range 21-78 years). Before esophagectomy, symptoms were present for a median of 13 years (range 1-44 years). Symptoms, diet, complications, and treatment before esophagectomy are listed in Table I. Median preoperative esophageal diameter was 6.2 cm (range 3-10 cm) and median sigmoid width was 10.5 cm (range 3-17 cm) (Fig 1).

Thirty (94%) patients underwent elective surgery and 2 (6%) required emergency esophagectomy for uncontrolled esophageal bleeding. Transhiatal esophagecto-

Table II. Postoperative complications after esophagectomy

Complication	No.	%
Pneumonia	7	22
Temporary total parenteral nutrition	7	22
Aspiration	5	16
Anastomotic leak	4	13
Respiratory failure	3	9
Arrhythmia	3	9
Temporary vocal cord neuropraxia	2	6
Wound infection	2	6
Chylothorax	1	3

Note: Categories are not mutually exclusive.

my was possible in 21 (66%) patients and thoracotomy was necessary in 11 (34%) patients. Five (16%) patients had intraoperative conversion of a planned transhiatal approach to thoracotomy because of dense adhesions preventing transhiatal esophageal resection, and 6 (19%) patients had resection by planned thoracotomy. Cervical esophagogastrostomy was possible in 30 (94%) patients, but previous surgery limited the amount of stomach available in 2 (6%) patients, which made intrathoracic anastomosis necessary. The bed of the esophagus was used for reconstruction in 31 (97%) patients. In 1 (3%) patient who required emergency esophagectomy, staged gastric reconstruction was performed via a substernal route.

Median intensive care unit stay was 2 days (range 1-12 days); median hospitalization was 14 days (range 8-95 days). Seventeen patients required a median of 2 transfusions (range 1-12 units). Postoperative complications are listed in Table II. There were no postopera-

Fig 2. **A**, Myenteric plexus from a control esophagus. The normal myenteric plexus shows easily identifiable ganglion cells (arrow) with essentially no inflammation. **B**, Myenteric plexus from a patient with achalasia showing severe neuritis. There is a marked lymphocytic infiltrate with admixture of eosinophils and plasma cells. **C**, Myenteric plexus from a patient with end-stage achalasia showing a scarred myenteric nerve. No ganglion cells are identified and the degree of lymphocytic inflammation is minimal. **D**, Trichrome-stained section from the same esophagectomy specimen as seen in **C**, showing a scarred nerve and minimal lymphocytic inflammation.

tive deaths. One patient who underwent emergency esophagectomy died 6 months after a complicated postoperative course.

Pathology review. All resection specimens had marked depletion of ganglion cells (Fig 2). In 20 (63%) specimens, no ganglion cells were present. Severe neural fibrosis was seen in all specimens. Severe neuritis was identified in 17 (53%) specimens. The pathologic hallmarks of end-stage achalasia (ie, complete absence of ganglion cells, severe neural fibrosis, and minimal neuritis) were seen in 12 (38%) specimens. In the proximal esophagus, ganglion cells were markedly depleted in 28 (88%) patients, with no ganglion cells present in 16 (50%). In the gastric cardia, ganglion cells were absent in 9 of 29 (31%) specimens.

Outcome. At a median follow-up of 43 months (range 3-115 months), all patients were able to eat without intravenous or enteral nutritional supplementa-

tion. Postoperative weight (median 71 kg, range 46-104 kg) was similar to optimized preoperative weight (69 kg, range 48-120 kg) in both men and women ($P = .2$) of any age ($P = .9$) and did not change significantly with time ($P = .2$).

Symptom scores were available in 29 of 31 patients. The assessment of symptom scores and postoperative diet are listed in Table IIIa. With esophagectomy and gastric reconstruction, dysphagia was reduced significantly from 93% to 17% ($P < .001$) and regurgitation from 90% to 28% ($P < .001$), with minimal change in reflux from 24% to 31% ($P = .5$). Postoperatively, there was a marked reduction of dietary restrictions from 59% to 17% ($P < .001$). Postoperative treatment necessary to obtain these scores is shown in Table IIIb.

There were few correlates of outcome (Table IV). Younger patients were more likely to have dysphagia than older patients (6.1 times more likely to experience

Table IIIa. Prevalence of symptoms and dietary restrictions at follow-up

Symptom†	Symptom score*					Total symptomatic (score >1)
	0	1	2	3	4	
Dysphagia						
No.	24	0	1	4	0	5
%‡	83	0	3	14	0	17
Regurgitation						
No.	21	0	1	7	0	8
%‡	72	0	3	24	0	28
Reflux						
No.	18	2	1	8	0	9
%‡	62	7	3	28	0	31
Early satiety						
No.	16	3	7	3	0	10
%‡	55	10	24	10	0	34
Diet						
Dietary restrictions						
No.	21	3	2	3	0	5
%‡	72	10	7	10	0	17
Social restrictions						
No.	20	6	2	1	0	3
%‡	69	21	7	3	0	10

*Symptom score: 0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = incapacitating.

†These symptoms may have been achieved by medications, dilations (dysphagia), or gastric drainage (N = 4) (early satiety) as indicated by treatment in Table IIIb.

‡Percent of 29 patients with this symptom score.

Table IIIb. Treatment of symptoms

Treatment†	Symptom score*					Total treated
	0	1	2	3	4	
Dysphagia						
No.	15	0	1	4	0	20
%‡	63	0	100	100	0	69
Regurgitation						
No.	0	0	1	4	0	5
%‡	0	0	100	57	0	17
Reflux						
No.	3	1	1	5	0	10
%‡	17	50	100	63	0	34
Early satiety						
No.	3	0	3	2	0	8
%‡	19	0	43	67	0	28

*Symptom score: 0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = incapacitating.

†Treatment includes medications, dilations (dysphagia), or gastric drainage (N = 4) (early satiety).

‡Percent of patients with this symptom score requiring treatment (see Table IIIa).

dysphagia for each 10-year decrease in age, $P = .03$). Patients with a smaller sigmoid width were more likely to have reflux (1.3 times more for each 1-cm decrease, $P = .06$) and regurgitation (1.4 times more for each 1-cm decrease, $P = .07$) than those with greater width. Neither symptoms nor dietary restrictions appeared to increase over time.

Among 30 patients, the condition of 26 (87%) was

improved after esophagectomy, the condition of 2 (7%) was unchanged, and the condition of 2 (7%) was worse. Twenty-five (83%) patients would undergo the operation again, 4 (13%) were undecided, and 1 (3%) would not.

Discussion

End-stage achalasia. Achalasia is the result of T-cell-mediated destruction and fibrous replacement of

Fig 3. Colon interposition for end-stage achalasia. Massive dilatation and tortuosity developed in both the thoracic and abdominal portions of this colon interposition 3 years after esophageal replacement. There was return of preoperative symptoms.

Table IV. *Correlates of outcome*

	OR	95% CI	P value
Preoperative			
Dysphagia			
Age (per 10-year increment)	0.2	0.03-0.8	.03
Sigmoid width (per 1 cm decrease)	1.4	0.97-2.0	.07
Regurgitation			
Sigmoid width (per 1 cm decrease)	1.4	0.99-1.8	.06
Reflux			
Sigmoid width (per 1 cm decrease)	1.3	0.99-1.8	.06
Operative	None		
Postoperative			
Dysphagia			
Anastomotic leak	7.3	0.7-73	.09
Pathology	None		
Assessment interval			
Dysphagia (per year)	0.8	0.5-1.3	.4
Regurgitation (per year)	1.2	0.9-1.7	.3
Reflux (per year)	1.2	0.8-1.7	.3
Early satiety (per year)	0.9	0.7-1.3	.6

OR, Odds ratio; CI, confidence interval.

the esophageal myenteric plexus.³ The inciting mechanism of this inflammatory response is unknown.⁴ By the time disruption of esophageal motility causes most

patients to seek medical treatment, substantial and irreversible damage to the esophageal myenteric plexus has occurred. Many patients respond to pneumatic dilation or myotomy of the lower esophageal sphincter. However, no or delayed treatment, inadequate reduction of lower esophageal sphincter pressure, or peptic stricture (the result of excessive reduction of lower esophageal sphincter pressure and poor esophageal clearance of refluxed gastric contents) may render the esophagus amotile and obstructed.

End-stage achalasia may be characterized clinically, radiographically, or pathologically. The majority of patients have disabling dysphagia (including aphagia) and regurgitation with associated weight loss and profound dietary restrictions. Aggressive treatment and its complications may further aggravate dysphagia caused by the primary disorder. The radiographic findings in end-stage achalasia are massive esophageal dilatation and tortuosity (sigmoid esophagus) in the majority of patients. Pathologically, there is a marked reduction or absence of ganglion cells with fibrous replacement of the myenteric plexus.

Treatment options in end-stage achalasia are limited, and esophagectomy offers the potential for improved swallowing. The principal function of the esophagus is the rapid unidirectional transit of food from the hypopharynx to the stomach. Unlike other segments of

the gastrointestinal tract, the esophagus has no digestive, absorptive, metabolic, or endocrine activities. Despite this seemingly rudimentary task and the deceptively simple arrangement of a muscle pump between two sphincters, it is difficult to replicate esophageal function.

Esophagectomy and colon interposition. Colon interposition is a true esophageal replacement; it spans the thoracic cavity to connect the cervical esophagus to the stomach that remains below the diaphragm. In expert hands, colon replacement offers restoration of gastrointestinal function in patients with end-stage achalasia.⁵ However, most surgeons have abandoned colon interposition when the stomach is adequate for reconstruction.⁶⁻¹⁰ There are many disadvantages to the use of colon as an esophageal replacement,¹¹ including a propensity to late complications and a tendency for the interposition to dilate and form redundant loops (Fig 3).¹²⁻¹⁵ This late complication results in the same problem for patients with end-stage achalasia that initially prompted esophageal resection. Reoperation for a failed colon interposition was reported in 6 of 85 (7%) patients undergoing esophagectomy with colon interposition for benign esophageal diseases.¹⁶

Esophagectomy and gastric reconstruction. The treatment protocol of esophagectomy with gastric reconstruction restored the ability to swallow in all patients, with a marked reduction in the number of patients with dietary restrictions. The improvement in dysphagia and regurgitation apparently was stable over time, and postoperative weight was stable and unchanged from preoperative weight. These findings suggest that gastric reconstruction is durable.

Dysphagia, the hallmark of end-stage achalasia and the primary indication for most operations, was relieved or minimized in the majority of patients. However, patients who were younger reported more persistent dysphagia than older ones. Patients with achalasia have a disordered perception of swallowing. Some of this reflects the disease process and some the social aspects of eating. The fact that younger patients were more likely to report dysphagia than older ones is probably composed of multiple factors: shorter duration of disease, a memory of normal swallowing, social activities that may foster dysphagia (such as active lifestyles, eating fast food, eating rapidly), high expectations of normal swallowing after gastric reconstruction, and more social situations in which eating and drinking are focal points. A patient with an anastomotic leak was more likely to have dysphagia.

A lesser sigmoid width was associated with an increased chance of postoperative dysphagia, regurgi-

tation, and reflux. These unexplained findings were not related to any variable or the pathologic findings in the proximal esophagus or gastric cardia. However, this observation suggests that esophagectomy for end-stage achalasia should be considered only after alternative treatment options have been exhausted and the esophagus is dilated and has obtained a large sigmoid width. With the advent of minimally invasive esophageal surgery, laparoscopic Heller myotomy has been performed generally without fundoplication in patients without prior therapy or inadequate therapy regardless of the sigmoid quality of the esophagus. Esophagectomy with gastric reconstruction is reserved for patients with documented failure of adequate myotomy who have a sigmoid esophagus. This delay may avoid esophagectomy in the younger patient and may decrease the prevalence of dysphagia.

Conclusions

This study is limited by its being a single-institution study of a small consecutive patient group with formal assessment of outcome at only one point in time. However, this work is strengthened by its protocol construction. The use of gastric reconstruction for end-stage achalasia is a procedure with low mortality and morbidity commensurate with that expected for esophagectomy. It relieves the preoperative problems of dysphagia and regurgitation in the majority of patients. There is good dietary function and, after preoperative restoration, weight is easily maintained. These results appear to be durable. Delaying esophagectomy until other treatment options have been exhausted may improve outcome.

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Discussion

Dr. Tom R. DeMeester (*Los Angeles, Calif*). I enjoyed the presentation by Dr Banbury. It was nicely done, well illustrated, and to the point. He has given us an opportunity to review the long-term outcome of our therapy for the disease achalasia. Those who have had 25 years of experience in the management of this disease will attest that patients with achalasia are charmed by anything that is done to improve their ability to swallow. There are 2 reasons for this: First, even though they often deny it, swallowing is so extremely difficult for them that any improvement is often exaggerated; second, eating makes up such a great part of our social lives that any improvement is greatly appreciated. Consequently, it is difficult to know how successful the initial therapy of balloon dilation or surgical myotomy is on symptomatic evaluation alone. For this reason, about 10% will have progressive esophageal dilatation over time and end up with what is called *end-stage achalasia*. At this point, almost everyone agrees, and the authors' data certainly support, that esophageal replacement is the only beneficial therapy.

The focus of Dr Banbury's presentation is that the stomach makes a good esophageal substitute. This is a subject about which I have given much thought, and I have the following comments: First, swallowing after esophageal replacement with either colon or stomach is never like swallowing with a normal esophagus. Consequently, 17% of Dr Banbury's patients have persistent dysphagia, 25% regurgitation, 31% reflux, and 44% early satiety. Yet 83% would elect to have the operation again if that were possible. This tells you how appreciative these patients are for any improvement in their ability to swallow. My question is why 17% have persistent

dysphagia and why the younger patients seem to have more difficulty. Can you explain this?

Dr Banbury. To answer your first question, the advisability of positioning the stomach in the chest is a matter of debate. Many of these patients with dysphagia are younger but they do have the ability to swallow. Perhaps the anastomosis, perhaps the denervated intrathoracic organ is the cause. I cannot explain specifically why there is persistent dysphagia in some, but certainly it is improved from the preoperative status. The use of prokinetic agents helps with clearance of food and stasis. The specific reasons for dysphagia are difficult to pinpoint.

Dr DeMeester. Did any of the patients with dysphagia require dilation?

Dr Banbury. Yes. We are aggressive in dilation. All of these patients are monitored in the postoperative period with endoscopy, and often they are treated by dilation during endoscopy. Sixty percent of patients had postoperative dilation with a median of 5 dilations per patient.

Dr DeMeester. Was the dilation mainly required because of narrowing of their gastric esophageal anastomosis?

Dr Banbury. Any patient who had complaints with regard to the anastomosis was subjected to endoscopy. We routinely perform endoscopy in patients at a point in the postoperative period. If there were problems with the anastomosis that we identified on endoscopy, and in patients who had no complaints, then we dilated the esophagus during endoscopy.

Dr DeMeester. In our experience the need for dilation after a colon interposition is rare, less than 5% (*J Thorac Cardiovasc Surg* 1998;115:1241). Thus there is an issue here that I think needs to be evaluated in greater detail.

My second question deals with your emphasis on attention to detail in regard to the operation. I appreciate that emphasis and I think it is a very valid comment, particularly your emphasizing the need to prevent redundancy of the stomach in the thoracic cavity. Of course, the same admonition can be applied to the colon; you must make every effort to avoid redundancy of either substitute, colon or stomach. Could you explain why preventing redundancy is so important in your experience with the stomach?

Dr Banbury. There is concern about postoperative motor function of the stomach, and redundant stomach could complicate that situation. A straighter gastric tube allows gravity to help the food bolus to pass straight through into the abdominal cavity rather than to be hung up in folds in the chest. That is also true with colon.

Dr DeMeester. Do you make any effort to tube the stomach or reduce it in size, that is, to form a gastric tube as opposed to the whole stomach?

Dr Banbury. The stomach is not specifically tubed, but the proximal aspect is fashioned for the anastomosis.

Dr DeMeester. Do you anchor the stomach to the diaphragm?

Dr Banbury. Yes, we do.

Dr DeMeester. My last question is in regard to an observation we have made, and I would like your thoughts about it. In our experience it is very difficult to place gastric mucosa

next to squamous mucosa without having a problem over the long term. These problems start about 5 years after the operation. Studies have shown that a denervated stomach regains its ability to produce acid similar to preoperative levels. Because of this, 19% of patients will eventually have esophagitis in the cervical esophagus on long-term follow-up, and 12% will have Barrett's esophagus in the cervical esophagus, with the mean time for its development being 7 years. This has not been associated with the use of the colon. My question to you, Dr Banbury, is this: Have you had the opportunity to examine some of your long-term patients with an endoscope? If so, what were the findings? I took note that you have made some adjustments in your operation because of concern over reflux, which suggests that the same thing is happening in your patients.

Dr Banbury. We do routinely examine these patients with an endoscope. We have not found esophagitis or endothelial changes so far, but reflux is a problem in some of our patients. We have changed our operative technique to try to further reduce the complication of reflux.

Dr DeMeester. Do the patients who have symptoms of reflux have irritation of the throat or are they having aspiration?

Dr Banbury. Irritation. We do use acid suppression therapy in any patient who has this problem.

Dr DeMeester. That ends my questions. I do believe that the best organ for long-term replacement of the esophagus is still an open issue.

Dr John R. Benfield (*Sacramento, Calif*). Dr Banbury, this is an important paper. My question is to you and perhaps to Dr DeMeester as well. I would like to hear about the conventional wisdom that you expressed, which is that the stomach remains peristaltic whereas the colon is an aperistaltic conduit. Do you actually have evidence that the stomach remains peristaltic?

Dr Banbury. No, we have not done studies of the stomach to determine specific motility.

Dr Benfield. I doubt that the stomach remains peristaltic, and I was wondering whether you had some evidence with which to dispel my doubt.

Dr DeMeester. Our studies on the stomach show that it does not regain peristaltic function; swallowing becomes pretty much a gravity feed system. Our studies of the colon show that it does provide some peristaltic function, but this occurs several years after the operation. I commonly say to the patients that the longer the colon is in the better it performs, in contrast to the stomach. From my own experience with these operations, about 5 years after a gastric pull-up the patients start having trouble. I will be interested to see Dr Banbury's data as follow-up becomes longer.