EDITORIAL

ANNALS OF SURGERY
Vol. 226, No. 2, 121–122

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

One of the most interesting trends observed in epidemiology of cancer in this country has been the dramatic increase in the incidence of adenocarcinoma of the esophagus. Not more than a few decades ago, the majority of cancers of the esophagus were of the squamous cell type with adenocarcinoma accounting for less than 10% of esophageal malignancies. Since the 1970s, the incidence of adenocarcinoma of the esophagus has increased at a rate surpassing all other cancers, and currently adenocarcinoma accounts for at least one half of the esophageal cancers diagnosed in this country.[1]

The reasons for this change are unclear. Coincident to the increase in incidence in adenocarcinoma of the esophagus was the recognition of the association of this cancer with the acquired condition known as Barrett's esophagus. Currently, Barrett's esophagus remains the only known risk factor for adenocarcinoma of the esophagus. In Barrett's esophagus, squamous epithelium, damaged by chronic esophageal reflux, is replaced by columnar cells that proliferate to form abnormal epithelia resembling both gastric and intestinal mucosa. The repetitive injury leads to mutations during the repair process in the p53 gene, a gene that controls cell death, and results in morphologic changes of dysplasia.[2] The progression of Barrett's esophagus to varying degrees of dysplasia and adenocarcinoma has been clearly defined, supporting the clinical significance of this condition.

Initially, it was assumed that the injury of the esophageal mucosa resulted from excessive exposure to acid reflux. It now is recognized that a number of factors are important to this process including 1) an increase in esophageal exposure to acid and *alkaline* gastric contents; 2) an incompetent lower esophageal sphincter; 3) delayed esophageal body clearance of refluxed material; 4) increased gastric acid secretion; 5) delayed gastric emptying; and 6) excessive duodenogastric reflux. Recently, both experimental and clinical evidence has provided strong support for the theory that it is the reflux of alkaline duodenal contents that plays the most important role in this process.[3–5] The recent introduction of a fiberoptic technique for detection of reflux of bilirubin in the esophageal lumen has provided one more level of support for the theory of the importance of excessive duodenal reflux.[6]

The understanding of the pathophysiology of Barrett's esophagus has the potential for a dramatic effect on the treatment of this condition. The current medical management of reflux esophagitis using agents that decrease gastric acid secretion has no effect on the prevalence of Barrett's esophagus and often allows progression of the disease.[7] Only the creation of an effective barrier to reflux of both gastric and duodenal contents can be expected to halt the progression of the metaplastic changes of Barrett's esophagus.[8] But is a standard antireflux procedure adequate when the stakes are as high as the potential for malignant transformation, as can be seen with Barrett's esophagus?

In 1987, DeMeester et al.[9] introduced a new operation known as the duodenal switch procedure for the treatment of the uncommon entity of primary alkaline gastritis. Now 10 years later, Csendes et al. from The University Hospital in Santiago, Chile, have extended the indications for the duodenal switch procedure to include patients with reflux esophagitis and Barrett's esophagus. In their series published in this issue of *Annals of Surgery,* (pp. 123–133) the authors have combined the duodenal switch procedure, to prevent duodenogastric reflux, with a highly selective vagotomy, to decrease acid secretion, and an antireflux procedure to provide a barrier to esophageal reflux as a "triple assault" in the treatment of Barrett's esophagus.

The authors have prospectively evaluated this procedure in the treatment of 65 patients with documented Barrett's esophagus. There are two valuable components of this study. First, the application of their procedure to a large number of patients with Barrett's esophagus with both short-term evaluation of perioperative morbidity as well as long-term evaluation of subjective and objective results. Second, the authors have evaluated thoroughly the physiologic changes associated with this procedure

with respect to foregut motility, acid secretion, esophageal pH monitoring, and hormonal response.

My brief interpretation of the results is as follows. The procedure was performed with no mortality and minimal morbidity with technical developments during the course of this study having further lowered the morbidity associated with the procedure. Substantial symptomatic improvement was observed in patients with follow-up in excess of 12 months. Although no significant change in the endoscopic location of the squamocolumnar junction was seen, endoscopic evidence of erosive esophagitis was improved markedly, and in three of seven patients, dysplastic changes disappeared. The results of esophageal manometry and 24-hour pH monitoring both before and after the procedure show favorable changes with a substantial decrease in gastric acid secretion without evidence of altered gastric emptying or serum gastrin levels. The reflux of bilirubin into the esophagus as detected by the fiberoptic probe (Bilitec) analysis showed complete elimination of the reflux of duodenal contents. In summary, the operation can be done safely and appears to work based on objective and subjective analyses.

The question now must be answered: should this procedure be done, and if so, on what group of patients would this procedure be indicated? Ten years ago, this question may not have been as complex. But now with the uniformly good results seen with the application of laparoscopic antireflux procedures, the benefits derived from this procedure must be significant to favor it over the laparoscopic alternatives now available. To support the widespread application of this procedure, evidence must be provided that standard antireflux procedures are inadequate to prevent reflux of either acid or alkaline gastric contents and further progression of Barrett's epithelium. I do not believe there is such evidence, and therefore I cannot agree with the authors inference that this procedure should be applied uniformly in the treatment of this disease.

My opinion does not detract from the excellent results reported in this series. I also am impressed greatly with their thorough prospective evaluation of their patients. I would suggest that the authors now apply their energies to a randomized, prospective study comparing their proposed procedure with a standard antireflux operation, likely a laparoscopic Nissen fundoplication, alone. If I am wrong and the clinical outcome and physiologic assessment favor their "triple assault," I look forward to writing another editorial in a few years to acknowledge my mistake. Until that time, I see little chance that this procedure will gain widespread acceptance in the treatment of patients with Barrett's esophagus.

Keith D. Lillemoe, M.D.
Baltimore, Maryland

References

1. Blot WJ, Devesa SS, Kneller RW, Fraumeni JF. Rising incidence of adenocarcinoma of the esophagus and gastric cardia. JAMA 1991; 265:1287–1289.
2. Blount PL, Ramel S, Raskind WH, et al. 17p Allelic deletions and p53 protein overexpression in Barrett's adenocarcinomas. Cancer Res 1991; 51:5482–5486.
3. 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.
4. Waring JP, Legrand J, Chibichian A, Sanowski RA. Duodenogastric reflux in patients with Barrett's esophagus. Dig Dis Sci 1990; 35:759–762.
5. 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.
6. 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.
7. Bremmer CG. Barrett's oesophagus. Br J Surg 1989; 76:995–996.
8. DeMeester TR, Attwood SEA, Smyrk TC, et al. Surgical therapy in Barrett's esophagus. Ann Surg 1990; 212:528–542.
9. 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.
10. Csendes A, Braghetto I, Burdiles P, et al. A new physiological approach for the surgical treatment of patients with Barrett's esophagus. Technical considerations and results in 65 cases. Ann Surg (in press).