The recognition of Barrett’s esophagus has altered the approach to patients with gastroesophageal reflux disease. Endoscopy was formerly reserved for those patients with symptoms of complicated gastroesophageal reflux disease (dysphagia, odynophagia, hematemesis), but is now being widely applied in an attempt to diagnose Barrett’s esophagus. This is advocated due to the known progression of Barrett’s esophagus through more severe degrees of dysplasia to esophageal adenocarcinoma. It is suggested that by careful screening of this metaplastic mucosa, dysplasia can be identified and the esophagus resected while there is either no or localized adenocarcinoma, and therefore provide a cure. In this issue of Digestive Diseases, Clark and DeMeester [1] present an excellent review of the management of dysplasia in Barrett’s esophagus. Their approach is aggressive and should indeed decrease the incidence of adenocarcinoma in Barrett’s esophagus by performing resection of dysplastic tissue. They also advocate antireflux surgery for patients in earlier stages of Barrett’s esophagus. Several points raised in their review bear further discussion.

The first issue of concern is the hypothesis that preventing acid reflux will slow or eliminate the progression from routine Barrett’s epithelium to a dysplastic epithelium and then to carcinoma. Medical therapy using both proton pump inhibitors and histamine receptor antagonists has been able to resolve the inflammatory complications of Barrett’s esophagus, but to this point has failed to result in regression of that epithelium [2]. An important recent paper has also demonstrated that elimination of reflux symptoms does not ensure that acid is controlled in patients with Barrett’s esophagus [3]. Surgical antireflux therapy has been suggested to result in regression of Barrett’s esophagus in both adults and children although these studies have been criticized due to the lack of complete regression and some questions in regard to the actual documentation of the length of Barrett’s esophagus since surgery may alter the usual landmarks [4]. A problem with an aggressive antireflux surgery approach in patients with Barrett’s esophagus is that surgery has not been clearly proven to prevent progression to carcinoma. A second problem with this approach is a consequence of the number of patients in the population with Barrett’s esophagus. The prevalence rate of Barrett’s esophagus may be as high as 12% in patients with reflux symptoms. It has been estimated that the age- and sex-adjusted prevalence of Barrett’s esophagus is therefore somewhere between 23 and 376 cases/100,000 depending on whether these are diagnosed clinically or by autopsy [5]. This number of patients undergoing a fundoplication would further stress the health care system and add considerably to the already high costs associated with Barrett’s esophagus.
Cost issues must certainly be considered when making recommendations in regard to Barrett’s esophagus. It has been estimated that a yearly endoscopic surveillance program could cost as much as USD 62,000 and result in 78 days of work to be lost by patients in order to discover one cancer during Barrett’s follow-up [6]. In addition, it has been suggested that the overall cost of this annual surveillance program could be as high as USD 118,000 for each quality-adjusted life year gained [7]. This is much higher than the cost per life saved for therapy in other chronic diseases. This cost to benefit ratio would be worsened by the addition of a large number of patients with short segment Barrett’s who have an unknown incidence of adenocarcinoma. The cost would also be adversely affected by the addition of antireflux surgeries in many, if not all, patients with Barrett’s esophagus. Further prospective data including cost analysis are critical prior to suggesting a wide application of more aggressive approaches such as routine laparoscopic fundoplication for all patients with Barrett’s esophagus. We are also lacking careful prospective data on patients with short segments of specialized columnar epithelium to determine whether they have the same cancer risks as in the longer forms of Barrett’s esophagus. Although the optimal approach remains unclear, I would suggest the following basic outline for the management of patients with Barrett’s esophagus which varies little from that proposed by Clark and DeMeester [1] and is adapted from Spechler[8].

1. Regular endoscopic surveillance for dysplasia and early carcinoma in patients with known Barrett’s esophagus unless contraindicated by comorbidity. For patients with no dysplasia or cancer, endoscopy with biopsy and cytology can be performed every 2-3 years. If dysplasia is detected it should be confirmed by at least one other expert pathologist. If there is disagreement, further diagnostic material should be obtained. Some advocate doing this only after 6-8 weeks of aggressive acid suppression.

Surgery is advised to resect all of the esophagus lined by columnar epithelium in patients confirmed to have foci of high-grade dysplasia.

For patients with confirmed low-grade dysplasia, aggressive antireflux therapy (medical or surgical) is recommended. More frequent (every 6-12 months) endoscopic and histologic surveillance is needed.

References
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