Barrett’s metaplasia is a common disorder associated with gastroesophageal reflux disease (GERD). There is no doubt that Barrett’s esophagus is caused by complicated GERD since patients with this entity have the same characteristics on diagnostic work-up as patients with severe GERD [1]. Patients with Barrett’s metaplasia have a long history of GERD symptoms, and generally do provide more pronounced pathologic findings on functional esophageal testing such as manometry, pH-monitoring, and bile-testing than GERD patients without Barrett’s. Barrett’s metaplasia carries an increased risk for the development of esophageal adenocarcinoma [2]. Pathophysiologically, this is supported by the fact that reflux of duodenal contents into the esophagus, usually found in Barrett’s patients, enhances the production of oxygen-derived free radicals on a high level [3, 4], inhibits apoptosis in the esophageal mucosa [5] and stimulates esophageal mucosal growth [6].

Due to the complex pathophysiology often found in GERD medical therapy aimed at the suppression of gastric acid production does not prevent the development of Barrett’s esophagus [7] and hence it is also not effective in preventing progression to Barrett’s cancer. Patients with short segment Barrett’s esophagus and patients with long segment Barrett’s were studied separately. In the majority of patients the Nissen fundoplication was applied, a few of these patients had a redo procedure. A small number of patients with ineffective esophageal contractility underwent partial posterior fundoplication. Postoperative analysis included assessment of typical reflux symptoms, endoscopy, esophageal manometry and pH-testing. The length of the Barrett’s epithelium was measured endoscopically. Whereas all patients underwent clinical evaluation of reflux symptoms postoperatively, endoscopy was performed in only 85 % of patients after surgery, and only about half of the patients completed esophageal manometry or 24-hour pH-monitoring.

The postoperative outcome regarding control of reflux symptoms was good with more than 80 % of patients being relieved from heartburn and/or regurgitation. However, data on pH-monitoring were rather poor. The mean postoperative DeMeester score was 17.1 and, although significantly reduced compared with preoperative findings, still in the pathologic range. About 25 % of the patients tested postoperatively had pathologic pH-testing. It has been considered that failure of antireflux surgery was associated with reoperation, paraesophageal herniation and poor esophageal body motility necessitating partial posterior fundoplication. The authors found a significant reduction of the extent of Barrett’s metaplasia with complete regression in about one quarter of patients. However, complete regression of Barrett’s metaplasia was seen only in patients with short-segment Barrett’s but not in those with long-segment Barrett’s. Patients with regression of Barrett’s metaplasia had a significantly lower acid exposure on pH-testing than those in whom no regression was found. One patient developed high grade dysplasia postoperatively. On testing, this patient was found to have recurrence of GERD. Another patient developed adenocarcinoma one year after operation. It is not stated whether this patient had had failed of antireflux surgery.

Conclusions drawn from this study are that antireflux surgery is effective in patients with Barrett’s metaplasia.
regarding clinical outcome. Failure of antireflux surgery in this subset of patients can be ascribed to special situations such as partial posterior fundoplication in the case of deteriorated esophageal peristalsis. Antireflux surgery results in regression of Barrett’s metaplasia and prevents progression to esophageal adenocarcinoma.

Data of this study showing good clinical outcome following antireflux surgery in patients with Barrett’s metaplasia is in accordance with the recent literature [11]. However, there are studies which demonstrate a high rate of failure of antireflux surgery in this subset of patients [12]. In fact as regards pH-monitoring, the present study by Oelschlager et al. also demonstrates poor results following antireflux surgery in Barrett’s. In this context, the presented manometric data on the function of the lower esophageal sphincter (LES) do not add to further understanding since only the resting pressure has been quoted but neither the intra-abdominal sphincter length nor the intra-abdominal sphincter vector volume, which are sensitive parameters of sphincter function [13]. Also no data are given on the recurrence of hiatal hernia. In our experience, control of gastroesophageal reflux following antireflux surgery is effective in Barrett’s patients [14]. Barrett’s metaplasia is often associated with large hiatal hernias [1]. Under these circumstances careful dissection of the herniated stomach far up into the chest with removal of the hernial sac and approximated approximation of the diaphragmatic crura is mandatory in order to achieve good results. If this can be accomplished, postoperative outcome in Barrett’s is as good as in patients with uncomplicated GERD. We also do not feel that partial posterior fundoplication may be a cause of failure in patients with Barrett’s metaplasia [14]. When performing partial posterior fundoplication, technical details are very important enabling the construction of a fundic wrap which provides the same manometric features as the Nissen fundoplication. With good operative techniques, the resting pressure of the LES and the intra-abdominal sphincter length are not significantly different compared with the Nissen fundoplication [15]. However, the authors of the present study do not demonstrate any of these data and therefore they do not prove whether they were able to perform partial posterior fundoplication sufficiently.

Regression of Barrett’s metaplasia after antireflux surgery is a matter of continuing discussion in the literature. Although the data presented in the study of Oelschlager et al. [1] are supported by a recent paper of the DeMeester group, conclusions on the regression of Barrett’s metaplasia following antireflux surgery have to be drawn with caution [11]. In fact in our series of patients, we never found a true regression [14]. Evaluation of the extent of Barrett’s is usually achieved by measuring the length of a Barrett’s segment endoscopically. However, in our opinion, this method is very inaccurate. Postoperatively, the gastroesophageal junction is invaginated into the stomach and therefore a few centimeters of a Barrett’s segment cannot be visualized and a false regression may appear. This is supported by the fact that in the study of Oelschlager et al. [1] complete regression was only found among patients with short-segment Barrett’s.

The conclusion that antireflux surgery may prevent progression of Barrett’s metaplasia to esophageal adenocarcinoma cannot be supported by the data of Oelschlager et al. In this study, one patient developed cancer, giving an incidence of about 0.4% per year which is about the risk seen under medical therapy. However, conducting a meta-analysis on a large number of patients, Bammer et al. [1] demonstrated that the incidence of Barrett’s cancer after antireflux surgery tends to be lower than under medical surveillance [2]. In this study, patients with failed antireflux surgery were also included. Sufficient antireflux surgery, however, seems to entirely inhibit progression to esophageal adenocarcinoma [16].

In conclusion, this study demonstrates a good clinical effect of antireflux surgery in patients with Barrett’s esophagus but fails to demonstrate true regression of Barrett’s postoperatively.

References