Evidence of Association Between GERD and Asthma

The prevalence of GERD increases in asthmatics compared with normal controls. The prevalence of GERD in asthmatics is estimated at between 34% and 89%5,12-16, which probably reflects the variation in GERD definition and populations studied. Although a proportion of patients with GERD have no obvious symptoms, data showed that, among the asthmatics, 77%, 55%, and 24% experienced heartburn, regurgitation, and swallowing difficulties, respectively17. Sontag et al18 performed endoscopy and esophageal biopsy on 186 adult asthmatics, and revealed that 43% of the patients with asthma had esophagitis or Barrett’s esophagus, or both. In healthy volunteers, only 13.8% have abnormal endoscopic findings in esophagus, and 8.5% presented with erosive esophagitis19. Sontag et al5 measured lower esophageal sphincter (LES) pressure and studied gastroesophageal reflux (GER) patterns using 24-h esophageal pH monitoring in 44 controls and 104 adult asthmatics. They found that 82% of adult asthmatics had abnormal gastroesophageal reflux. Compared with the controls, asthmatics had significantly lower LES pressure, greater acid exposure time, more frequent reflux episodes, and longer clearance times in both the upright and supine positions5. Several studies have demonstrated that significant proportion of patients with asthma suffered GERD without classic GERD symptoms of heartburn or regurgitation. Harding et al13 reported that 24-h esophageal pH tests were abnormal in 29% asthmatics without reflux symptoms. Another study performed by Harding et al20 evaluated 26 patients with stable asthma without reflux symptoms using esophageal manometry and 24-h esophageal pH testing, and found that the prevalence of abnormal 24-h esophageal pH tests in asthma...
patients without reflux symptoms was 62%. Al-Asoom et al\textsuperscript{16} found that 36.4\% of asthmatic patients diagnosed by esophageal pH monitoring as having GER did not complain of heartburn and hoarseness of voice; such as the reflux was silent.

GERD may induce or aggravate asthma. A cross-sectional international population survey in 2,661 participants showed that, compared with those without GERD, individuals with GERD had increased risk of pulmonary conditions like wheezing, breathlessness at rest and nocturnal breathlessness\textsuperscript{21}. In this study, association of GERD with physician-diagnosed asthma was marginally significant (OR = 2.2; 95\% CI = 1.04-4.70)\textsuperscript{21}. El-Serag et al\textsuperscript{22} studied 101,366 patients with erosive esophagitis or esophageal stenosis and found that, compared with the controls, patients with reflux esophagitis were at an increased risk of asthma (OR = 1.5; 95\% CI = 1.4-1.6). Recently, El-Serag et al\textsuperscript{23} conducted a large case-control study in 1980 children with GERD and 7920 controls without GERD, and reported GERD was a significant risk factor for asthma (OR = 1.9; 95\% CI = 1.6-2.3). A cross-section study reported that, in the week prior to completing the questionnaire, 41\% of the asthmatics noted reflux-associated respiratory symptoms, including cough, dyspnea, and wheeze and 28\% used their inhalers while experiencing GER symptoms; inhaler use correlated with the severity of heartburn (r = 0.28, p < 0.05) and regurgitation (r = 0.40, p < 0.05)\textsuperscript{17}. Using the Medline 1966-1999 database, Field\textsuperscript{8} reviewed the studies on the relationship between GER and asthma, and concluded that GER caused asthma symptoms but had minimal effects on pulmonary function. Asymptomatic GER did not worsen asthma. Antireflux therapy might have a role in asthma patients with symptomatic GER, possibly being most beneficial for those with reflux-associated respiratory symptoms. Both medical and surgical antireflux therapy could improve asthma symptoms and asthma medication requirements without improving pulmonary function\textsuperscript{8}.

**Mechanisms of GERD Inducing or Aggravating Asthma**

Although accumulated evidences have established that GERD might induce or exacerbate asthma, the mechanism by which GERD might induce or aggravate asthmatic symptoms remains unclear. Two mechanisms have been proposed, as listed below.

1. Acid in the inflamed esophagus acting on exposed receptor causes an increase in bronchial hyper-responsiveness via the vagal reflex\textsuperscript{12}.

   Maybe it is associated with the fact that the esophagus and the bronchial tree share embryological origins in the foregut and have common autonomic innervations. Early report by Mansfield et al\textsuperscript{24} indicated that, using intraesophageal acid provocation test, flow resistance increased when reflux symptoms occurred, and rapidly reversed after relief of reflux symptoms. It demonstrated a reflex mechanism that might produce the observed bronchoconstriction. Schan et al\textsuperscript{25} performed the intraesophageal acid perfusion test in asthmatics with reflux, asthmatics, gastroesophageal refluxers, and normal controls. Peak expiratory flow rate (PEF) decreased with intraesophageal acid in all four groups. Esophageal acid clearance improved PEF in all groups except the asthmatics with reflux group that had a further decrease in PEF. The asthmatics with reflux group also had an increase in specific airway resistance with intraesophageal acid, which continued to increase despite acid clearance. These effects were not dependent on a positive Bernstein test or evidence of proximal reflux\textsuperscript{25}. However, Spaulding et al\textsuperscript{26} reported the response that respiratory resistance increased significantly after intraesophageal acid perfusion only occurred in asthmatics with active esophagitis. This may be associated with the exposure of acid sensitive receptor or the increase of acid receptor sensitivity due to esophagitis.

   The changes in pulmonary function when acid infused into the esophagus are relatively small. The acid infusion mainly causes an increase in bronchial hyper-responsiveness. The severity of asthmatic symptoms is correlated to the level of hyper-responsiveness. Increase in bronchial hyper-responsiveness induced by acid perfusion may be the important cause of asthma aggravation. Herve et al\textsuperscript{27} compared the effects on maximal expiratory flow at 50\% of VC (MEF50) of esophageal perfusion of hydrochloric acid (HCl) and of normal saline (NaCl) in 12 asth-
matics. In all subjects, HCl perfusion did not change MEF50 but potentiated the bronchoconstriction induced by isocapnic hyperventilation of dry air (maximal decrease in MEF50 = 44 ± 7% with HCl versus 22 ± 5% with NaCl; p < 0.001) or methacholine (provocative dose producing a 20% decrease in FEV1 = 349 ± 99 mg with HCl versus 496 ± 119 mg with NaCl; p < 0.01). 7 of the asthmatic subjects were found to have GER on esophageal pH monitoring. In these subjects, HCl alone decreased MEF50 slightly but significantly, possibly reflecting the higher degree of basal bronchial hyperreactivity due to GER27. This bronchial response to esophageal acid perfusion can be blocked by bilateral vagotomy or atropine pretreatment27,28. Thus, acid in the inflamed esophagus acting on exposed receptor and via the vagal reflex to causes an increase in bronchial hyper-reactiveness is considered to be an important mechanism by which GERD induces or aggravates asthma.

2. Microaspiration of gastric contents into the lung damages the bronchial mucosa, which results in inflammation of the mucosa and bronchial hyper-responsiveness29,30. Tuchman et al31 showed that acid inhaled into the airway even in low concentrations caused far greater bronchoconstriction than acid infused in the esophagus, which suggested that microaspiration was a much more likely mechanism for bronchospasm associated with GER. However, microaspiration may be an important mechanism in children asthmatics with GERD, as to adult, aspiration is uncommon.

Scintigraphy was used to demonstrate microaspiration in asthmatics with GERD. Nevertheless, aspiration was proved in only a small number of asthmatics with GERD, which indicated that scintigraphy may lack adequate sensitivity or that aspiration in asthmatics with GERD occurred in only a small subgroup of these patients or at infrequent intervals which was difficult to detect14,32. Proximal esophageal acid exposure was a requirement for microaspiration. Harding et al33 proposed that proximal esophageal acid exposure should be correlated with the changes of PEF and specific airway resistance (SRaw) if microaspiration induces bronchoconstriction. They performed esophageal acid perfusion in asthmatics with GER in supine position and examined the PEF, SRaw and the proximal esophageal acid exposure. They found that the decrease in PEF and increase in SRaw were not associated with the presence of proximal esophageal acid exposure33. This suggests that microaspiration does not play a significant role in esophageal acid-induced bronchoconstriction.

Pathophysiology of Asthma
Facilitate GERD

Asthma predisposes patients to get GERD. With air flow obstruction, negative pleural pressure will increase. And chronic cough or wheezing will increase positive abdominal pressure. This results in the increase of the gradient pressure across the LES – which promotes GER. In addition, over inflation and air trapping may lead to diaphragmatic flattening, potentially impairing the antireflux barrier, and ultimately predisposing to GERD. Bronchodilators (theophylline and β2-agonists) have been reported to adversely affect GER by decreasing the pressure of the LES34-36. Stein et al34 evaluated LES pressure obtained before and after an intravenous dose of aminophylline in 4 normal volunteers and 6 asthmatics with symptoms of GER. All subjects had therapeutic theophylline levels at the time of the second measurement of LES pressure and a significant decrease in LES pressure occurred in normal and asthmatic volunteers34. DiMarino and Cohen36 studied the effect of the oral β-adrenergic agonist on LES pressure in normals and in patients with achalasia. After a 4.0 mg dose of carbuterol, the mean LES pressure significantly decreased from 23.1 ± 6.2 mm Hg to 16.0 ± 5.0 mm Hg in normals and from 50.1 ± 5.1 mm Hg to 22.7 ± 2.4 mm Hg in patients with achalasia36. In addiction, theophylline could increase gastric secretion37. However, the clinical role of theophylline and β2-agonists therapy in causing GERD remains controversial5,34,39. Sontag et al5 reported that there were no differences in LES pressure, esophageal mucosal acid contact time and frequency of reflux episodes between asthmatics who required bronchodilators and those who did not. Recently, Harding et al33 also found that theophylline did not affect esophageal parameters in 24-h esophageal pH tests.
Manifestations of GERD

Manifestations of GERD can be divided into 3 types, including: (1) classic symptoms: heartburn, acid regurgitation, worsen at supine or bending position, often occur after a meal, especially a large meal or high-fat-containing meal; (2) atypical manifestations: noncardiac chest pain, gastrointestinal symptoms (epigastric discomfort, nausea, dysphagia, etc); (3) extraesophageal manifestations: pulmonary manifestations, such as asthma, chronic cough, recurrent pneumonia, chronic bronchitis, idiopathic pulmonary fibrosis and chronic obstructive pulmonary disease; laryngeal and pharyngeal manifestations, such as laryngitis, pharyngitis, sore throat, hoarseness, and choking sensation; other manifestations including halitosis, sinusitis and dental erosion, etc40,41. Although there is a correlation between symptoms of GERD and mucosal damage, they are not parallel. Patients will commonly present with severe mucosal disease, including stricture and Barrett’s esophagus, yet report minimal or no symptoms of reflux disease42.

In asthmatics with factors listed below, the existing of GERD should be suspected. The factors includes: (1) difficult to control asthma, not responding to the treatment without obvious causes; (2) steroid-resistant asthma; (3) symptoms (heartburn, regurgitation) occur when changing position (from sitting, upright to supine or bending); (4) hoarseness; (5) predominance of nocturnal crises; (6) cough or wheezing after acid drink or food; (7) cough or wheezing after a large meal; (8) asthma symptoms worsen after certain foods (e.g. chocolate, alcohol, peppermint, and coffee, etc); (9) asthma symptoms after vomiting6,16,43.

Empiric Therapy

All asthmatics should be carefully questioned about the esophageal and extraesophageal manifestations of GERD. Specific questions should include: (1) whether asthma symptoms occur after a large or a high-fat-containing meal, or with foods that are known to decrease LES pressure; (2) whether cough, wheezing, or dyspnea is associated with a reflux episode, or a patient used inhalers while experiencing GERD symptoms6. If the patient’s history is typical for GERD, no further diagnostic testing is needed and an initial trial of empiric therapy is appropriate. The diagnosis of GERD is established if the symptoms relieve after empiric therapy. Data indicated that a significant proportion of asthmatics with GERD required higher doses than 20 mg/day omeprazole for symptomatic relief10. A common strategy is 20 mg of omeprazole twice per day (or equivalent) for 3 to 6 months40. Johnsson et al44 reported that, if GERD was defined as reflux esophagitis Savary-Miller grades II-III at endoscopy or pH < 4 exceeding 4% of the total time at 24-h esophageal pH-monitoring, good response to 1-week treatment with 20 mg omeprazole twice daily had a sensitivity of 75% and a specificity of 55% in identification of patients with GERD. If the empiric therapy is unsuccessful, or the patient’s symptoms suggests complicated diseases (esophagitis, esophageal stricture, Barrett’s esophagus, or neoplasm), further diagnostic testing is recommended. In addition, patients who have longstanding symptoms or who require continuous therapy may need endoscopy for evaluating esophageal mucosa45.

Diagnosing GERD in Asthmatics

Diagnostic tests for GERD have developed rapidly. Common methods include endoscopy, barium esophagram, ambulatory esophageal pH monitoring, esophageal manometry, and acid perfusion test. And some new techniques are used for the diagnosis of GERD, such as ambulatory esophageal bile monitoring, esophageal scintigraphy. All of the methods have limitations in sensitivity and specificity. There is no perfect “gold standard” for diagnosing GERS at present.

Mucosal Evaluation

Patients in whom empiric therapy is unsuccessful, or who have longstanding symptoms or symptoms suggesting complicated disease, may need mucosal evaluation. The technique for evaluating mucosa includes endoscopy and air-contrast barium esophagram. Endoscopy provides direct visualiza-
tion of the esophageal mucosa and allows biopsy. Endoscopy combined with biopsies has 100% sensitivity and specificity for identifying esophagitis, but only 50% to 70% sensitivity for the presence of GERD. Barium esophagram is an easy, cheap and tolerable technique. Although barium radiography has excellent diagnostic accuracy for esophageal stricture and deep ulcer, it is not sensitive for GERD. The sensitivity for reflux esophagitis is correlated with the severity of the esophagitis, it can detect 22% of patients with mild esophagitis, 83% with moderate esophagitis, and 95% with severe esophagitis.

Sellar et al. reported that, using a “compression” method to provoke reflux, barium radiography had the sensitivity of 71% and accuracy of 72% for GERD. However, the severity of the GERD symptoms is not parallel to the damage of the esophageal mucosa. Patients with typical or atypical manifestation of GERD often have no esophagitis appearance and are negative in endoscopy. These cases must be confirmed with other methods.

Ambulatory Esophageal pH Monitoring

Ambulatory esophageal pH monitoring plays a key role in diagnosing GERD. When the purpose of the test is screening for GERD, it has the highest sensitivity (88% to 95%) compared with the other tests. And the reproducibility is between 84% and 93%. Patients with typical GERD symptoms and documented esophagitis will not benefit from an initial pH monitoring. However, in patients with persistent symptoms without evidence of mucosal damage and with noncardiac chest pain, ambulatory esophageal pH monitoring helps to confirm GERD and allows the identification of the relation of patient’s symptoms and reflux episode. Furthermore, ambulatory esophageal pH monitoring is recommended in patients without classic reflux symptoms or those with difficult to control asthma. Irwin et al. studied 42 patients with difficult to control asthma, and found that GERD was the most common factor that contributed to making asthma difficult to control. GERD was a definite factor in 64% of asthmatics who had a favorable response to medical antireflux therapy. When it was a definite factor, it was clinical “silent” in 24%. Although endoscopy allows for the evaluation of esophageal mucosa, the presence or absence of mucosal injury does not provide proof that the patient’s symptoms are or are not related to GERD. Nevertheless, ambulatory pH monitoring allows not only the identification of patients with excess esophageal acid exposure but also clarification the relation of patient’s symptoms and reflux episodes. Using 24-hour pH monitoring, DeMeester et al. evaluated 77 asthmatics with suspected reflux-related respiratory symptoms (persistent cough, wheezing, and/or recurrent pneumonia) and found that respiratory symptoms occurred during or within 3 minutes after a reflux episode in 22% patients, within 3 minutes before a reflux episode in 16%, and unrelated to a reflux episode in 62%. Ambulatory esophageal pH monitoring also allows the evaluation of the effect of acid suppression in patients with refractory symptoms while taking antireflux therapy.

Esophageal Manometry

Esophageal manometry is insensitive in the diagnosis of GERD. A considerable overlap of LES pressure values exists between GERD patients and normal subjects. Thus, a single pressure has little diagnostic value unless extreme. Richter et al. reviewed 6 studies and indicated that a low LES pressure of less than 10 mm Hg had poor sensitivity (58%) but good specificity (84%) in diagnosing reflux. Recently, esophageal manometry is mainly used to facilitate placement of ambulatory pH probes and to guide antireflux surgery.

Acid Perfusion (Bernstein) Test

The acid perfusion test shows the mucosal sensitivity to acid. The advantage of this test is that it may establish the relation between the patient’s symptoms and GERD. If the patient’s symptoms are provoked by acid and relieved by a normal saline, the test is highly specific for GERD. But unfortunately, the sensitivity of Bernstein test is low. Richter et al. reviewed 7 studies and found an overall sensitivity of 79% and specificity of 82%. In addition, the test can’t exclude reflux and also can’t differentiate between degrees of reflux or esophagitis.
Treatment of Gastroesophageal Reflux Disease in Asthma

Lifestyle Modification
Lifestyle modification should be initiated and continued throughout the course of GERD therapy. Numerous studies have indicated that elevating the head of the bed, decreased fat intake, avoiding of large meal, avoiding recumbent for 3 postprandial hours, cessation of smoking, and weight loss may reduce esophageal reflux. Certain foods (e.g., chocolate, alcohol, peppermint, and coffee, etc.) have been noted to increase esophageal reflux and should be avoided. Expert opinion holds that education of the patient about factors that may precipitate reflux is reasonable, although randomized trials are not available to test the efficacy of lifestyle modification. Patients who are not responsive to lifestyle modification should take medical treatment.

Medical Therapy
Proton pump inhibitors (PPIs) provide rapid symptomatic relief and healing of esophagitis in the highest percentage patients, which will be the first choice for acid suppression in asthmatics with GERD. In patients with GERD, esophageal acid exposure is reduced by up to 80% with H₂-receptor antagonists and up to 95% with PPIs. Treatments with omeprazole in asthmatics with GERD have been reported having different efficacy in relieving asthmatic symptoms, which was due to the difference in effect of acid suppression and course of therapy. In a large study, and the only one to use pH monitoring, Harding et al. evaluated 30 non-smoking adult asthmatics with gastroesophageal reflux before and after 3 months of omeprazole therapy. Gastroesophageal reflux was defined by symptoms and abnormal 24-hour esophageal pH testing. During the 4-week pretherapy phase, patients recorded reflux and asthma symptom scores and PEF upon awakening, 1 hour after dinner, and at bedtime. Patients began 20 mg/d omeprazole, and the dose was titrated until acid suppression was documented by 24-hour pH test. Patients remained on this acid suppressive dose for 3 months. Treatment with omeprazole resulted in improvements in asthma symptoms in 67% of patients, and pulmonary function in 20%. Mean acid suppressive dose of omeprazole was $27 \pm 2.2$ mg/d with 27% patients requiring more than 20 mg/d. The presence of regurgitation or excessive proximal esophageal reflux predicted asthmatics’ good response to acid suppressive treatment with sensitivity of 100%, specificity of 44%. Field et al. reviewed 12 studies of the effect of medical antireflux therapy in asthmatics with GER, and concluded that medical antireflux therapy improves asthma symptoms, may reduce asthma medication use, but has minimal or no effect on lung function. Most of the trials of medical therapy enrolled only small numbers of patients and are thus limited by having insufficient statistical power. Moreover, most of the trials used relative short course of acid suppression therapy (3 months or less). The course may be only sufficient for symptomatic relief but insufficient for pulmonary function improvement, which may take up to 1 year. In addiction, the acid suppression may be inadequate in some trials. The only one trial, which used on-treatment pH monitoring to ensure acid suppression, reported that pulmonary function improved in 20% of patients.

Studies comparing the clinical efficacy of PPIs showed that standard doses of the PPIs (omeprazole, lansoprazole, pantoprazole, and rabeprazole) resulted in comparable rates of healing and remission in erosive esophagitis. For time-to-healing, esomeprazole (40 mg/day) was found to be superior to omeprazole (20 mg/day) or lansoprazole (30 mg/day) for healing erosive esophagitis within 8 weeks, but only in some of the comparative trials; these results were not consistently found and of little clinical importance. There is no evidence supporting the differences in efficacy among PPIs in endoscopy-negative patients. In the light of comparable safety and efficacy, cost may be a factor in choosing PPIs.

There have been many trials evaluating the effect of H₂-receptor antagonists on asthmatics with GERD. Ekstrom et al. enrolled 48 patients with moderate to severe asthma into a double blind crossover study of ranitidine 150 mg twice daily for 4 weeks. There were significant reduction in nocturnal respiratory symptoms and the need for inhaled β₂-agonists. However, there were no significant changes in objective parameters of pul-
monary function. The beneficial effects of therapy occurred in only 56% patients with a history of reflux-associated respiratory symptoms. Bowrey et al\textsuperscript{41} studied 9 trials evaluating the effect of H\textsubscript{2}-receptor antagonists and found that, 7 of the 9 trials reported a beneficial effect on asthma symptoms, with almost half of the treated patients experiencing an improvement in symptoms, despite wide variations in the duration of therapy (range 4 weeks to >1 year). Our study\textsuperscript{41} showed that H\textsubscript{2}-receptor antagonists (ranitidine) could aggrivate airway obstruction and increase bronchial hyper-responsiveness in asthmatics without GERD. In contrast, it could relieve respiratory symptoms and decrease bronchial hyper-responsiveness in asthmatics with GERD. It suggested that H\textsubscript{2}-receptor was expressed in the airway of the asthmatics, which mediated the dilation of the bronchial smooth muscle and inhibited the bronchial hyper-responsiveness\textsuperscript{76}. Thus, antisecretory therapy in asthmatics with GERD prefers PPIs to H\textsubscript{2}-receptor antagonists.

Promotility agents could increase LES pressure, promote esophageal clearance and gastric emptying, which provide control of acid reflux. Promotility agents such as cisapride and metoclopramide have an efficacy similar to standard-dose histamine receptor blockers\textsuperscript{45}. However, there have been reports of fatal cardiac dysrhythmias associated with cisapride. And metoclopramide have frequent central nervous system side effects, such as drowsiness, irritability, extrapyramidal effects. These side effects have appropriately decreased their regular use.

**Surgical Therapy**

Although the majority of GERD can be managed successfully with medical therapy, patients with severe complications, refractory disease, or with a severely incompetent lower esophageal sphincter should be referred for surgical evaluation\textsuperscript{77}. Antireflux surgery attempts to restore sphincter competence by wrapping the gastric fundus around the esophagus, called fundoplication. When performed skillfully, this procedure will restore LES, reduce reflux, and heal peptic esophagitis. The potential advantage of surgery over medical therapy is that the reduction in esophageal acid exposure is greater, at up to 98%\textsuperscript{41}. Harding et al\textsuperscript{43} combined the results of surgical trials and found that, overall, 34% of patients were free of asthma symptoms postoperatively, 42% were improved, and 24% were unchanged. Two randomized controlled trials of surgical versus medical antireflux therapy for asthmatics with GERD have been published. Larrain et al\textsuperscript{11} randomly assigned 81 non-allergic asthmatics with GER to receive cimetidine (300mg four times daily) or placebo or to undergo antireflux surgery (a modified posterior gastropexy). At the end of the 6 months treatment, there was improvement in FEV\textsubscript{1} and midexpiratory flow rate in the cimetidine and surgical groups but not in the placebo group. This improvement didn’t reach statistical significance. Overall, 77% of surgical-treated patients, 74% of cimetidine-treated patients, and 36% of placebo-treated patients had reduced wheezing. Complete relief of respiratory symptoms was reported by 35% of surgical-treated patients, 48% of cimetidine-treated patients, and 4% of placebo-treated patients. After completion of the 6 month study period, 27% of surgical-treated patients, 15% of cimetidine-treated patients, and none of placebo-treated patients were able to discontinue steroids. However, after 6 months trial, discontinuation of cimetidine resulted in prompt return of symptoms. In contrast, 50% of surgical-treated patients were free from symptoms in a long-term follow-up (average, 77 months)\textsuperscript{11}. In another study, Sontag et al\textsuperscript{78} randomized 73 asthmatics with GER to Nissen fundoplication, ranitidine 150 mg three times daily, or antacids on an as-needed basis (controls). Asthma symptom scores and bronchodilator and prednisone requirements were recorded monthly for 1 year. Symptoms were completely relieved or improved in 75% of surgical-treated patients, 9% of ranitidine-treated patients, and 4% of the controls. Asthma worsened in 48% of the controls, 36% of the ranitidine-treated patients, and 6% of surgical-treated patients. During therapy, prednisone could be discontinued in 33%, 11% and 0% of the patients in the surgical-treated, ranitidine-treated and control groups. A beneficial effect on PEF was observed significantly more frequently in patients treated by fundoplication compared with the other two groups. Six months after fundoplication, one third of the surgical-treated patients had >10% increase in maxi-
ormal PEF, and these benefits were still present at 24 months after surgery. The author considered that surgical treatment in asthmatics with GERD may superior to medical therapy in improvement of pulmonary function and decrease of bronchodilator and prednisone. However, these two studies neither used PPIs nor documented control of acid reflux by 24-h esophageal pH testing. In the one trial that is considered optimal antisecretory therapy, the success rate was comparable to that seen after fundoplication. Large-scale studies comparing longer courses of optimal PPIs antisecretory therapy (up to 12 months) with fundoplication are lacking at present. Moreover, side effects (e.g. late dysphagia, lowered quality of life) of antireflux surgery are more serious and widespread than currently believed, which should be taken into consideration before the surgery.

References


