Aspiration Resulting from Gastroesophageal Reflux
A Cause of Chronic Bronchopulmonary Disease

The association of bronchopulmonary disease with gastroesophageal reflux is an old clinical observation. Aspiration of acid gastric content during sleep is the proposed mechanism. Unfortunately, the exact incidence of chronic bronchopulmonary disease due to aspiration associated with gastroesophageal reflux is unknown. This is partly because, until recently, there has been no test to precisely define pulmonary aspiration of gastric content. In addition, secular subspecialty interest has limited the study of the association between bronchopulmonary disease and gastroesophageal reflux. That is, while pulmonologists are interested in diseases caused by aspiration, they do not have a primary research interest in gastroesophageal reflux. In addition, gastroenterologists do not have an investigative interest in the adverse effect of refluxed gastric content beyond that of the esophagus. Fortunately, the advent of scintigraphic techniques that objectively define pulmonary aspiration and gastroesophageal reflux has now afforded a multidisciplinary approach to restudy the old association between bronchopulmonary disease and gastroesophageal reflux.

The article by Crausaz and Favez (Ches 1988;93:376[February]) objectively addresses the subject of bronchopulmonary disease that results from aspiration due to gastroesophageal reflux. In this study, the authors showed that patients with bronchopulmonary disease had a significant increase in the incidence of gastroesophageal reflux when compared to asymptomatic control subjects. Reflux was determined by a scintigraphic technique using an ingested radiolabeled liquid (orange juice) and provocative maneuvers to induce reflux. Importantly, 75 percent of the patient group had evidence of aspiration on pulmonary scintigraphy. These scans were performed in the morning, on another occasion, after the ingestion of a radiolabeled solid meal the previous evening. A third scintigraphic study showed that the patients with bronchopulmonary disease had significant increases in disordered gastroesophageal transit of liquid, thus indicating disordered motility. That the results of the authors' scintigraphic studies were concordant was suggested by one patient who had all scintigraphic studies twice.

A stumbling block in initially accepting the authors' conclusion that aspiration may have caused bronchopulmonary disease in their patients occurs with the finding that both patients and controls had a comparable incidence of vegetal fibers in their sputum the morning after ingesting a bran biscuit the previous evening. This observation appears to be in direct conflict with that of pulmonary scintigraphy. We, as well as the authors, feel that this conflict can be explained by the fact that it is physiologic to aspirate saliva into the trachea and/or upper airway during sleep. Hence, physiologic aspiration of saliva into the trachea and upper airways of both groups would explain the comparable incidence of vegetal fibers found in the early morning sputum of both groups. In contrast, pathologic aspiration of gastric content into the lower airways of the patients would explain their increased incidence of abnormal pulmonary scintiscans. It would also support the authors' hypothesis that aspiration resulting from gastroesophageal reflux caused the patients' bronchopulmonary disease.

Patients in the study by Crausaz and Favez with bronchopulmonary disease and scintigraphic evidence of aspiration also had concomitant "motion" disorders of the esophagus. Esophageal dysmotility along with delayed emptying are known risk factors for aspiration. For instance, patients with untreated achalasia who have esophageal aperistalsis and retained liquid/undigested food in their dilated esophagus can die during sleep from aspiration. Based on this observation, it is conceivable that patients with refluxed acid gastric content in the esophagus at night are also at risk for aspiration. This risk is relevant, especially since some patients with reflux esophagitis may have disordered esophageal motility. This dysmotility has been characterized as a decrease in the amplitude of the esophageal peristaltic contractions, failure of a swallow to either generate a peristaltic contraction wave or the failure of the wave to transit the distal esophagus and nonperistaltic contractions. Therefore, the observation by Crausaz and Favez of "motion" disorders on esophageal scintigraphy in their patients with bronchopulmonary disease supports the old clinical association between esophageal dysmotility and pulmonary aspiration, as well as new evidence concerning motility disorders in patients with reflux esophagitis. In fact, their scintigraphic data concerning "motion" disorders of the esophagus supports the view that delayed clearance of nocturnal acid reflux is a risk factor for respiratory symptoms, and/or pulmonary aspiration, in adults or children with gastroesophageal reflux.

The study by Crausaz and Favez has some deficiencies. While the authors referenced the source of the scintigraphic reflux test, they did not elaborate on any
modifications they may have made nor did they specify their definition of reflux. These variables may account for their observation that 38 percent of asymptomatic control subjects had an abnormal reflux test, a value higher than that originally published.10 These unpublished variables will make it hard for other authors to compare their data for scintigraphy in a comparable population. These deficiencies, however, do not detract from the authors’ basic observation that aspiration from gastroesophageal reflux may lead to chronic bronchopulmonary disease.

In summary, we agree with the authors’ comment that physicians should search for gastroesophageal reflux in patients with chronic bronchopulmonary disease, especially those without an apparent etiology. However, we disagree with their proposal that silent aspiration occurs in 75 percent of patients with chronic bronchopulmonary disease. Many of the patients in their series had clinical evidence suggestive of aspiration such as recurrent cough, noncardiac nocturnal dyspnea, and recurrent pulmonary infections following esophageal surgery. In fact, one could take the position that the authors’ symptomatic patients did not represent silent nocturnal aspiration, but instead, previously unrecognized nocturnal aspiration. Regardless of the incidence of aspiration from gastroesophageal reflux in patients with chronic bronchopulmonary disease or the degree to which it is “silent,” Crausaz and Favez have still made a significant contribution toward documenting aspiration to be a causative factor in some patients with bronchopulmonary disease.

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REFERENCES
2 Andersen HA, Holman CB, Olsen AM. Pulmonary complications of cardiospasm. JAMA 1953; 151:608-12
10 Fisher RS, Malmud LS, Roberts GS, Lobis JR. Gastroesophageal (GE) scintiscanning to detect and quantitate GE reflux. Gastroenterol 1976; 70:301-07