Flow Volume Loops in Diagnosis

To the Editor:

Gelb and associates have described the physiologic characteristics of malignant unilateral mainstem bronchial obstruction in patients treated with laser therapy. In their most recent series, eight patients without a prior history of COPD had a restrictive pattern on flow volume loops, four with associated air flow obstruction. After treatment, all patients had an increase in FVC with a parallel upward shift in MEFV curve. We recently evaluated a patient who had a flow volume loop configuration which is potentially diagnostic for mainstem bronchial obstruction.

This patient was a 76-year-old man who presented with a six-month history of intermittent hemoptysis. Fiberoptic bronchoscopy revealed a polypoid tumor (poorly differentiated carcinoma) located 3 cm from the carina, causing complete obstruction of the bronchus during expiration. Spirometric testing revealed a mixed ventilatory defect with FVC of 2.56 L (63 percent of predicted) and FEV, of 1.09 L (35 percent of predicted). FEV, increased 17 percent post-bronchodilator therapy. There was no difference between FVC and SVC in this patient. Lung volume measurements revealed a normal FRC by both helium dilution and body box methods and a normal TLC. Diffusion capacity was 88 percent of predicted. Flow volume loop revealed a normal upstroke to the peak flow and then a rapid linear fall to an inflection point at approximately 43 percent of forced vital capacity. From this point to residual volume, flow was very low and approached zero (Fig).

This patient's pulmonary function test results indicated that he had a mixed ventilatory defect on spirometry but normal total lung capacity. The flow volume loop suggested that the nonobstructed side of the lung empties rapidly, and the obstructed side empties slowly throughout the forced vital capacity maneuver. Consequently, the expiratory portion of the flow volume loop demonstrates both a restrictive pattern and a severe obstructive pattern in series and is unlike any of the patterns reported by Gelb et al. This pattern is not the airway collapse pattern described by Healy et al and Jayamianne et al. Patients with severe COPD may have the airway collapse pattern in which there is an abrupt decrease in flow from peak to an inflection point less than 50 percent of peak flow which occurs during the first 25 percent of expired vital capacity. These patients typically have very severe obstructive ventilatory defects with low diffusion capacities (mean FEV, 24 percent of predicted, mean Dco 41 percent of predicted in Healy et al). This pattern reflects dynamic compression of the tracheobronchial tree early in expiration and probably develops secondary to reduced central airway support, reduced lung recoil, increased peripheral resistance, and increased intrapleural pressures during forced expiration.

In our patient, the inflection point occurred near mid-vital capacity and the patient had no physiologic evidence of emphysema, at least based on diffusion capacity. In summary, we suggest that this flow volume loop pattern observed in our patient is diagnostic of mainstem endobronchial tumors which cause nearly complete obstruction during forced exhalation maneuvers. This case is similar to one reported by Dull and coworkers.

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FIGURE. Flow volume loop redrawn from original tracing using a MacIntosh computer. This study was done with the Gould 2450 system (dry seal spirometer and Epson printer).
REFERENCES


To the Editor:

With respect to the letter of Lutz and Nugent, we were most interested in their observation. Their description of the maximum expiratory flow volume loop in their patient is somewhat similar to our patients 8 and 9, who had underlying chronic obstructive pulmonary disease in our article in the American Review of Respiratory Disease (1988; 138:1392-85). We have already elaborated upon the possible physiologic explanations for the observed pattern.

We tend to suspect that the characteristics of the curve would be most compatible with unilateral main stem obstruction superimposed upon underlying diffuse obstructive pulmonary disease in the uninvolved lung. Alternatively, severe (but incomplete) main stem bronchial obstruction could result in a similar pattern. While consistent with unilateral main stem bronchial obstruction, this flow volume pattern, however, may not be pathognomonic. We appreciate their interest in this matter.

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Atypical Evolution of Pulmonary Tuberculosis During Treatment

To the Editor:

A recent article by Williams et al (Chest 1988; 93:836-38) presents a paradoxic type of evolution in three patients with tuberculosis primoinfection who, during correct treatment, appear to have experienced a reactivation of the disease accompanied by wheezing and bronchial inflammation. The essential contribution of this paper is that the reactivation appeared as a bronchial inflammation with asthmatic symptoms.

Some years ago we reported eight cases of this kind;4 recently we have encountered eight more.3 This paradoxic type of evolution was first described more than 30 years ago by Choremis et al,4 although without remarking upon the probable bronchial component. Later, this type of evolution was also found in adult pulmonary3 and pleural4 tuberculosis.

Knowledge of this type of evolution of pulmonary tuberculosis is important as it often causes problems for the physician. When faced with a case like this, it is easy to think of the appearance of resistance to drugs or of an initially mistaken diagnosis. All our cases have been cured with continuance of the same treatment. In no case have we used corticoid therapy.

These facts are not easy to interpret. Choremis et al3 and Williams et al4 thought of the possibility of a Herxheimer reaction owing to the liberation of large quantities of bacillary toxins that follows the destruction by chemotherapy of a great number of tuberculous bacilli. However, we believe this interpretation is, at most, doubtful. Herxheimer reactions begin a few minutes or hours after the administration of a chemotherapy drug, whereas in the cases mentioned above and in our cases, the apparent reactivation occurred between the second and sixth weeks after the beginning of the treatment. In the three cases reported by Williams et al, this reactivation began in weeks 12, 8 and 28. We rather think that this type of reaction may be related to the changes in the immune mechanisms of the patient during antituberculous treatment.

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1 Williams DJ, Norbert FJ, Sproude BJ. Endobronchial tuberculosis presenting as asthma. Chest 1988; 93:836-38

To the Editor:

Thank you for the opportunity to respond to Dr. Munoz-Cutierrez and associate's comments on our paper. We appreciate their comparison of our study with theirs. We recognize that endobronchial tuberculosis in childhood presenting with compression, obstruction and perforation were not uncommon before the modern era of antituberculous therapy.14 These studies showed clinical, radiographic and bronchoscopic deterioration two to three weeks after initiation of antituberculous therapy, similar to the description provided by Dr. Munoz-Cutierrez. A comparison of modern day therapy with older studies is difficult because of the differences in antibiotics, dosage and duration of treatment. However, despite appropriate modern antituberculous chemotherapy, early roentgenographic deterioration still occurs.8

Our patients differ from the above reports in presenting much later in the treatment course and requiring emergency hospital...