criteria that we outlined for diagnosing pneumothorax ex vacuo, which are as follows: The onset of pneumothorax is concurrent with the onset of acute lobar collapse secondary to acute bronchial occlusion. This typically, but not always, involves the right upper lobe. The involved lobe usually collapses to a marked degree and is retracted markedly toward the hilum. The pneumothorax is limited to the space between the retracted visceral pleural surface of the collapsed lobe and the overlying chest wall, and does not extend over the visceral pleural surfaces of the adjacent lobe or lobes, which remain fully expanded. These findings, which are typical of pneumothorax ex vacuo, are not seen in other forms of pneumothorax.

Several months after our manuscript was accepted for publication, we encountered a fourth case of pneumothorax ex vacuo. This occurred in a 23-year-old man with severe laryngotracheal papillomatosis who had marked involvement of the trachea, main bronchi, and right upper lobe bronchi. He presented with acute right upper lobe collapse with secondary pneumothorax ex vacuo limited to the space between the visceral pleural edge of the collapsed, retracted right upper lobe and the chest wall. There was no history of trauma and he was not on positive pressure ventilation. Bronchoscopy showed total occlusion of the right upper lobe bronchi by papillomas. Following laser ablation of the endobronchial papillomas, the right upper lobe rapidly re-expanded and the pneumothorax disappeared. No chest tube was inserted.

The exact mechanism of pneumothorax ex vacuo is unknown. A similar phenomenon is well-known to occur in the joints. Traction on a joint may pull gas (presumably to be nitrogen) into the joint space from the surrounding soft tissues. This is not infrequently imaged on routine bone radiographs, especially in children. After a few minutes the gas is gone, presumably having been reabsorbed into the surrounding soft tissues. The exact timeframe over which this occurs is unknown. Berdon et al previously published similar findings in children who developed acute lobar collapse from acute bronchial occlusion. In the cases they reported, pneumothorax occurred concurrently with lobar collapse, was limited to the space between the collapsed lobe and chest wall, and resolved spontaneously after the bronchial occlusion was relieved and the lobe re-expanded. We believe that the same phenomenon (pneumothorax ex vacuo) also occurs in adults.

Drs. Byrd and Roy suggest that the pneumothoraces in our cases moved anteriorly after the collapsed lobe re-expanded, and further suggest that chest tubes should have been inserted. We believe that most physicians would be reluctant to insert chest tubes in patients whose portable chest radiographs showed no pneumothorax such as those illustrated in our article (Fig 1, bottom; 2, bottom left; 3, bottom). Long-term follow-up after lobar re-expansion in our cases showed no evidence of any residual or recurrent pneumothorax. No condition can be diagnosed when the physician is unaware of its existence or when the condition is not considered a possibility.

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REFERENCES

Acute Hyperventilation in the Emergency Department

To the Editor:

I read with interest the article on hyperventilation by Saisch et al. My experience with hyperventilation almost 40 years ago was with a somewhat different population of US Air Force wives who were young and healthy but under the stress of not knowing whether they would be separated from their husbands who might be shipped out. Although Saisch et al minimize the role of anxiety in the hyperventilation syndrome, by their own statistics 16 of 23 patients were diagnosed with anxiety and/or panic syndromes. They site a very high incidence of asthma as a possible predisposing factor for hyperventilation. However, I see no relationship between the two conditions, and they site no evidence for this relationship. If the hyperventilation were the result of asthma, a simple bedside peak flow measurement would determine that airflow was limited. Even if wheezes were not present with a so-called “silent chest” in the late phases of asthma with early respiratory failure, arterial blood gases would provide a clue to this condition. There is no doubt that in an inner-city emergency room the incidence of asthma, alcohol, or marijuana use is higher than in the general population, but the authors site no studies to compare the incidence of these conditions in patients coming to the emergency room without hyperventilation.

Arterial blood gas measurements were not generally available 40 years ago when I researched and wrote my article, but in any condition producing hyperventilation—organic or nervous—an acute respiratory alkalosis with elevated pH and decreased Pco2 will result. If no cardiopulmonary disease is present, the oxygen tension should be normal or high; if Pco2 is low, this is a clue to pulmonary disease.

In the 40 years that I’ve been in practice and have seen hyperventilation as a common manifestation of anxiety, I have found a useful diagnostic test: I have the patient hyperventilate for 90 seconds to see if symptoms can be reproduced. This allows the patient to understand the mechanism for symptoms. In addition to explaining the pathophysiology for certain patients, I supply them with a copy of my article. I do not use rebreathing through a paper bag, but advise the patient to hold his/her breath as long as possible and breathe out through pursed lips to slow respiration. In these days of managed care, a referral to a psychiatrist is rarely necessary. A few minutes spent talking with the patient to find out what stress is bringing on the symptoms is cost-effective treatment.

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To the Editor:

Many of the issues raised by Dr. Singer have been addressed in my recent review on the pathophysiology of hyperventilation in this journal. I don’t think that we intended to imply that anxiety was not important but cited it as just one of a number of joint factors contributing to hyperventilation in these patients. Indeed,