**P carinii** and Pneumothorax

To the Editor:

It was with great interest that we read the report by Sherman et al, "Pneumocystis carinii Pneumonia with Spontaneous Pneumothorax" (Chest 1986; 90:609-10). Coincidental with its publication, we had a similar case at Monmouth Medical Center, Long Branch, New Jersey.

The patient was a 35-year-old man with a 15-year history of intravenous drug abuse and a smoking history of 23 pack-years. He was first admitted to our facility in late September, 1986, with previous positive ABC, HTLV III, ELISA and Western blot test results. He complained of a two-week history of progressively worsening cough productive of gray/brown sputum, hemoptysis, associated dyspnea and malaise. His chest x-ray film showed nodular infiltration in the upper half of the right lung, as well as some nodular infiltration in the apical aspect of the left lung (Fig 1), and was subsequently diagnosed as having *Pneumocystis carinii* pneumonia by both broncholar lavage and transbronchial biopsy sampling of the right lung. He was treated with intravenous Septra therapy and discharged on the tenth day of antibiotic therapy on oral Septra, clinically improved with no further complaints of dyspnea or hemoptysis.

Four days after discharge, the patient presented to the emergency room complaining of abrupt onset of left-sided chest pain after an episode of coughing while working outdoors at his parents' home. Physical examination demonstrated diminished breath sounds on the left side, and AP and lateral chest x-ray examinations revealed a tension pneumothorax on the left side. A chest tube was immediately inserted (Figs 2, 3). Six days later the chest tube was removed after no further air leak was noted. The patient, who had also developed a fever to 104°F and rash during this second admission, was changed to Pentamidine therapy and subsequently had a relatively uncomplicated recuperative period with resolution of his fever and decreased dyspnea and cough. Similar to Dr. Sherman's first case, our patient had a biopsy taken from the right lung and yet developed his pneumothorax on the left. It should also be noted that there were no overt blebs noted on the patient's chest x-ray films during his first admission. For similar reasons expressed by Dr. Sherman, we also suspect a local necrotic process as the etiology of pneumothorax.

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**Oxyhemoglobin Desaturation and Cardiac Arrhythmias**

To the Editor:

The incidence and severity of cardiac arrhythmias that appear during sleep have been studied by several authors in the last years. Possibly, oxyhemoglobin desaturation may play an important role in the origin and development of these arrhythmias. This condition has been demonstrated in healthy subjects and especially in some pathologic conditions: in certain cases, it has been related to sudden death during night sleep.

We studied the incidence and different types of cardiac arrhythmias and their relationship to nocturnal oxyhemoglobin desaturation episodes both in healthy subjects and patients with sleep disorders.

We studied 11 healthy subjects (six men and five women, mean age 62.5 ± 15) and 21 patients with sleep disorders (18 men and three women, mean age 60.1 ± 14). The 21 sleep disorder patients were selected because they had been referred within the previous four years for extreme daytime somnolence, loud snoring, and abnormal behavior during sleep. In each case, during a 24-hour period, we simultaneously analyzed oxyhemoglobin saturation (by means of an ear oximeter [Ohmeda Biot III-3100]) and frequency and types of cardiac arrhythmia by means of a Holter monitor (Reynolds Medical Pathfinder II-B1). Oxyhemoglobin desaturation was estimated by oxygen saturation at 50 percent cumulative time index.