randomized trial comparing the results to conventional ventilator therapy.

As I stated in my editorial, the initial enthusiasm with high frequency ventilation, high levels of positive end-expiratory, and extracorporeal membrane oxygenation ultimately proved to be unfounded once prospective randomized studies were done. Therefore, I support the plan to test pressure controlled inverse ratio ventilation in a prospective randomized trial.

I hope other institutions will not use pressure controlled inverse ratio ventilation until there is solid evidence that it improves outcome in patients with acute respiratory failure or ARDS.

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P Carinii Infection

To the Editor:

Pneumocystis carinii is usually considered an airborne alveolar pathogen. Yet granulomatous1-2 and necrotizing3,4 pneumonias have been related to P carinii infection. We had the opportunity to examine a surgical lung biopsy specimen from a 40-year-old man with acquired immunodeficiency syndrome (AIDS) complicated by bilateral pneumothorax.

The patient was a drug addict with a two-year history of AIDS. He had presented in the previous year with two bouts of P carinii pneumonia (PCP) treated with sulfamethoxazol-trimethoprim, and pulmonary tuberculosis (Tuberculosis hominis) treated for six months with isoniazid, ethambutol, rifampicin, and pyrazinamide. He was on preventive therapy against PCP with aerosolized pentamidine. One month before surgery, he complained of fever, cough and purulent sputum. Treatment with a combination of amoxicillin and clavulanic acid was prescribed. Chest x-ray examination showed bilateral interstitial pulmonary infiltrates. Two bronchial lavage lavages did not yield any specific findings of diagnostic value. The occurrence of a bilateral pneumothorax brought the patient to our intensive care unit. Chest x-ray film and thoracic CT scan showed bilateral nodular infiltrates predominating on the upper left lobe. There was no obvious excavation of the lesions. Surgical abrasion of the pleural cavity with lung biopsy was decided.

On gross examination, the lung specimen was spotted with granulomas. On histologic examination, the lesions were centered on bronchi, septa and pleura (Fig 1). They consisted of necrosis surrounded by foamy alveolitis. In several nodules, giant cell granulomas (Fig 2) and calcified deposits were observed. Some lesions were exclusively necrotic, angio-invasive, and destructive to the septa and pleura. In all these foci methenamine silver stain showed numerous cysts of P carinii in the necrosis, vascular walls and lumen. The patient was treated with trimethoprim and recovered.

Granulomatos and cavitary PCP, as well as pneumothoraces, have been described in patients with AIDS. Few histologic descriptions of the lesions have been reported. In our case, the necrotic and invasive behavior of PCP is unusual as the ability of P carinii to penetrate into the lung parenchyma is not commonly recognized. It would even appear that P carinii can enter the blood vessels and 'metastasize' to the pleura. The pleural necrotic lesions seem to be the cause of pneumothorax. Preventive treatment using aerosolized pentamidine may play a pathogenic role either by damaging the normal lung tissues or enhancing the entry of P carinii into the parenchyma which, if such were the case, could be observed with increasing frequency.

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REFERENCES


The Role of Dipyridamole in Bronchospasm

To the Editor:

In their article entitled, "Severe bronchospasm followed by respiratory arrest during thallium-dipyridamole imaging", Lette et al attempt to find an explanation for this dramatic reaction after dipyridamole. They hold the opinion that inhibition of pulmonary uptake of prostaglandin E1, a bronchodilatory lipid-derived mediator, might be the responsible mechanism. However, there exists a more plausible explanation that is supported by ample pharmacological data.

FIGURE 1. Lung biopsy: necrotic foci (arrow heads) located along the septa and pleura (H and E, × 50).

FIGURE 2. Lung biopsy: granulomas with giant cells (H and E, × 50).

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