AAT levels consistently above 0.8 g/L (13.75 pmol/L); however, their post-bronchodilator FEV₁ has continued to decline at the same rate as before therapy (Fig 1). Thus, our experience with our first two subjects is contrary to that of Barker et al; these subjects have continued to deteriorate at the same rate. The failure of this extremely expensive therapy, at least in some cases, justify the call for a controlled clinical trial of AAT replacement therapy.¹

Rajesh Bhagat, MD,
Veronica A. Sweeney, BSc, and
Donald W. Cockerill, MD,
Department of Medicine,
University of Saskatchewan,
Saskatoon, Canada

REFERENCE

More Cases of Miliary Mesothelioma

To the Editor:

The August 1994 issue (CHEST 1994; 106:605-06) contained the selected report, Miliary Mesothelioma, by Dr. Michael Huncharek. This case report described a patient with miliary pulmonary parenchymal involvement with malignant mesothelioma claiming to be the first such case reported to the knowledge of the author. The patient had a left-sided pleural effusion.

I published a case¹ describing a 44-year-old man with past mining exposure to blue asbestos who presented with supraclavicular lymphadenopathy and miliary shadowing on his chest x-ray film and who was proven with the results of needle aspiration of a cervical node and transbronchial lung biopsy while alive, and also at autopsy, to have miliary mesothelioma. Our case was different from a previously reported case cited in the New England Journal of Medicine² in which the patient with miliary spread of mesothelioma presented with a pleural effusion that was similar to the case reported by Dr. Huncharek.

I believe therefore that his claim to have reported the first such case is in error of fact and that this should be brought to the attention of your readers.

Arthur W. Musk, MD, FCCP,
Sir Charles Gairdner Hospital,
Nedlands, Australia

REFERENCES

Thoracoscopic Resection of Pulmonary Metastases

To the Editor:

I read with interest the Taiwanese experience on resecting pulmonary metastases using the thoracoscopic approach by Liu and colleagues (CHEST 1995; 107:266-68). Although the authors showed that this approach is technically feasible, there were neither follow-up data nor any comparison made between the thoracoscopic vs the conventional sternotomy or thoracotomy approach.

We recently reported our experience on the use of video-assisted thoracic surgery (VATS) in the management of metastatic pulmonary osteosarcoma.¹ From September 1993 to March 1994, 11 cases of therapeutic VAT wedge resection of pulmonary metastases were performed in 7 patients. We found digital palpation useful in localizing nodules intraoperatively.² In 5 out of 11 cases, we were able to detect more nodules during thoracoscopy than were suggested by fine-cut CT scans. All the nodules detected were resected with clear margins. Like Liu et al, we found that the thoracoscopic resections were associated with short postoperative hospital stays (mean 2.3±1.1 d) and good patient acceptance. However, we also found recurrence of pulmonary metastases in three out of seven patients from 4 to 6 months after the initial VATS procedure. This was probably related to an inability to detect intraoperatively all potentially palpable nodules.

The only merit of surgical resection for metastatic disease is the ability to resect all metastases.³ The high recurrence rate in our small series casts doubt on the adequacy of the VATS approach in therapeutic resections of pulmonary metastases. Our current practice is to reserve VAT wedge resections for diagnosis or to supplement an open procedure. However, we do not use the VATS approach alone when complete therapeutic resection is the goal. Until more information is available through prospective randomized study comparing the VATS vs the conventional approach, we believe that the former approach should not be recommended for general use just because it is technically feasible.

Anthony P.C. Yim, MD,
Cardiothoracic Unit,
Department of Surgery,
Prince of Wales Hospital,
Shatin, NT, Hong Kong

REFERENCES

Another Cause of Endobronchial Lesions Found in HIV Patients

To the Editor:

Marc A. Judson and colleagues reviewed endobronchial lesions in patients infected with HIV (CHEST 1994; 105:1314-23) and noted several diagnoses. Infectious diseases were most frequent, involving either opportunistic agents—Pneumocystis carinii, cytomegalovirus, bacillary angiomatosis—or common agents—Mycobacterium tuberculosis, Staphylococcus aureus, Pseudomonas aeruginosa. Other endobronchial lesions were the result of tumors: Kaposi’s sarcoma, nonHodgkin’s lymphoma, lung cancer, or granular cell myoblastoma. We wish to report another case in an HIV-infected man with an endobronchial mass as a result of Rhodococcus equi.

A 32-year-old HIV-seropositive man presented with acute pneumonia. His CT scan showed a dense opacity in the middle lobe, and