that the chest x-ray can be normal, but feel that colonization must be considered in this setting. An abnormal chest x-ray should be confirmed as related to MOTT by applying standard criteria for nontuberculous mycobacterial disease.4 If these criteria are not met, dissemination from a pulmonary isolate alone can be inferred if extrapulmonary disease is documented within one month (arbitrary).4,5 Since we used both 7H11 media (Lowenstein-Jensen) and the BACTEC 12A system, rapidity of initial diagnosis was improved but sensitivity for MOTT colonization was increased.

We could not discern the criteria used by Fournier et al to relate a positive culture for MOTT to either pulmonary disease or dissemination. It is difficult to judge which isolates signify MOTT disease and which might represent MOTT colonization. Without more information to exclude concurrent infectious pathogens, Kaposi’s sarcoma or other causes of “dyspnea, chills, hemoptysis, and chest pain,” it is not possible to ascribe a distinct syndrome to the nontuberculous mycobacteria. The paucity of positive cultures from extrapulmonary sites (seven of 68 isolates)—as the authors suggested—may reflect underutilization of such cultures, but it may also reflect the absence of true pathogenicity for the pulmonary isolates. Stower et al have previously reported that nonpulmonary specimens have an increased yield when MOTT are disseminated. Although expectorated sputum and bronchoalveolar specimens may be an important clue, the ultimate diagnosis of nontuberculous mycobacterial infection in patients with AIDS does not appear so straightforward. Given the potential morbidity and poor response to multidrug MOTT therapy, disease should be clearly differentiated from colonization before a diagnosis is made and treatment begun.

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1 Fournier AM, Dickinson GM, Erdfroich IR, Cleary T, Fischl MA. Tuberculosis and nontuberculous mycobacteriosis in patients with AIDS. Chest 1989;93:772-75

To the Editor:

The findings which Dr. Tenholder and Dr. Moser reported (which were not available for review at the time our publication was being prepared for submission) are complimentary to our own. Many of the differences can be ascribed to study design: ours was a cross-sectional chart review while they followed a cohort from one to 29 months. Our patients were admitted to an acute care hospital with symptoms, were referred for evaluation and were frequently free of pulmonary symptoms. The central issue is whether it is possible—without any degree of accuracy—to distinguish colonization from infection by nontuberculous mycobacteria in patients with the acquired immunodeficiency syndrome. We feel that the presence of immunodeficiency alters the clinical presentation of these infections. Therefore, standard criteria for distinguishing nontuberculous mycobacterial disease from colonization cannot be applied. Other authors consider any presence of these organisms in AIDS patients indicative of infection.1,2

Dr. Tenholder and Moser admit that the assignment of dissemination within one month as criteria for infection is an arbitrary distinction. In fact, in their own series some patients took as long as 24 months to disseminate to other organs.

Our criteria for ascribing the identification of nontuberculous mycobacteria as the cause of infection in this cross-sectional study was dependent on the clinical judgement of the physicians involved in the care of the patients, who ascribed symptoms to nontuberculous mycobacterial infection when no other explanation was found.

We do not feel that our data proves that a distinct syndrome of dyspnea, chills and chest pain is specific for nontuberculous mycobacterial infection. We do feel, however, that the findings of these symptoms in a significant minority of patients with nontuberculous mycobacteriosis but not with tuberculosis is curious and suggests that it may be specific, since other opportunistic infections would be expected to be randomly distributed between both groups. (This was indeed the case with regard to Pneumocystis carinii pneumonia.)

What is still needed is a large cohort of patients with documented infection followed for a long period of time. This would allow us to define the natural history of the disease. Until this is completed, the issue of colonization vs infection will be difficult to resolve.

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REFERENCES

Palliation of Massive Bronchorrhea

To the Editor:

Alveolar cell carcinoma accounts for up to nine percent of all pulmonary neoplasms, and six percent of these people develop characteristically voluminous sputum (bronchorrhea). Bronchorrhea usually occurs late in the course of the disease and with diffuse lung involvement.1 Several therapeutic interventions—including para-sympathetic blocking drugs, antihistamines, adrenocorticotropic hormone (ACTH), corticosteroids, infiltration of the stellate ganglia, chemotherapy, and radiation therapy—have been tried in the past in an attempt to control bronchorrhea, but all have failed to provide lasting benefit.2,3 We present a recent experience in which external beam radiation therapy for bronchorrhea in alveolar cell carcinoma provided both acute and long-term benefit.

A 56-year-old man was admitted to the Cleveland Clinic Hospital with a five-month history of progressive cough producing copious amounts of clear, frothy sputum. He had been unable to sleep for one month because of almost constant bronchorrhea. A chest radiograph showed an alveolar infiltrate in the lingula (Fig 1) and bronchoscopy revealed profuse watery secretions emanating from the lingula. Transbronchial biopsy revealed alveolar cell carcinoma. Multiple bony metastases precluded curative resection. Oral atro-
pine therapy (0.4 mg, three times daily) had no effect on the bronchorrhea. Radiation therapy (3,000 rads) was delivered to the left lung and mediastinum in ten fractions over two weeks and was of striking and immediate benefit in reducing his bronchorrhea; there was a marked reduction in his spatum production by the end of the first week of radiation therapy and almost total amelioration at completion of the course. The patient died five months later from progressive metastatic disease without recurrence of bronchorrhea.

The current literature provides no satisfactory therapy for controlling bronchorrhea that sometimes accompanies alveolar cell carcinoma. Although bronchorrhea may prove refractory because it accompanies diffuse parenchymal involvement by alveolar cell carcinoma, this patient's bronchorrhea accompanied relatively local pulmonary involvement, thus allowing acceptable radiation ports. The immediate and subacute benefits of radiation therapy may reflect the relatively limited extent of his pulmonary involvement. Based on this experience and the absence of response of bronchorrhea to previous therapies, we advocate a palliative course of radiation therapy for patients with inoperable alveolar cell carcinoma complicated by bothersome bronchorrhea.

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REFERENCES

Treatment of Obstructive Sleep Apnea with CPAP and Protriptyline

To the Editor:

Continuous positive airway pressure (CPAP) is a well-documented treatment for obstructive sleep apnea (OSA). It is highly effective but compliance is sometimes low, primarily because of nasopharyngeal irritation. Pressures in excess of 15 cm H2O are necessary in some cases to overcome the upper airway obstruction; this may lead to further dryness in the nose and throat. Reduction of the pressure and/or airflow could increase CPAP tolerance by decreasing nasal irritation and reducing the risk of barotrauma. Because protriptyline has been reported to increase both hypoglossal and recurrent laryngeal nerve discharge activity with no alteration of phrenic nerve discharge in cats1 and may also be a useful treatment for OSA in some patients,3 we hypothesized that combining these two treatments might yield beneficial synergistic effects.

We tested this hypothesis on two male patients. Both patient 1 (179 cm, 109.5 kg) and patient 2 (188 cm, 80.4 kg) had been diagnosed polysomnographically in our clinic as suffering from severe OSA on a baseline night (Table 1). On a subsequent night, (CPAP 2) pressures of up to 17 H2O nasal CPAP were introduced to eliminate apneas and hypopneas (Table 1).

On the next night (CPAP 3), the patients were administered 5 and 2.5 mg of protriptyline h.s., respectively (Table 1). CPAP again produced an improvement in OSA, but at considerably lower CPAP pressures than those used on the CPAP 2 night. We noted reductions in pressures even in REM sleep episodes where the patients were supine.

If further investigation supports these preliminary results of combining CPAP and protriptyline, it may be possible to routinely reduce the CPAP pressures and/or air flows, thereby reducing the risk of barotrauma and increasing patient compliance. Doses of protriptyline used are so small that the anticholinergic side effects may be nonexistent. Such a treatment combination may also be useful for patients with COPD who present with alveolar hypoventilation, reductions in upper airway muscle tone and increases in upper airway resistance during REM sleep. We are currently conducting a prospective, double-blind study to investigate this hypothesis in patients with OSA.

Table 1

<table>
<thead>
<tr>
<th>Test</th>
<th>Patient 1</th>
<th>Patient 2</th>
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<tbody>
<tr>
<td>Baseline TST</td>
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<td>400.4</td>
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<tr>
<td>Average AHI</td>
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<td>29.7</td>
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<tr>
<td>Lower SaO2</td>
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<td>69%</td>
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<tr>
<td>SaO2&lt;90 index</td>
<td>42.1</td>
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<tr>
<td>CPAP 2 TST</td>
<td>283.2</td>
<td>317.8</td>
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<tr>
<td>Average AHI</td>
<td>18.4</td>
<td>21.1</td>
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<tr>
<td>Lower SaO2</td>
<td>87%</td>
<td>86%</td>
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<tr>
<td>SaO2&lt;90 index</td>
<td>17.4</td>
<td>2.6</td>
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<tr>
<td>CPAP 3 TST</td>
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<tr>
<td>Average AHI</td>
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<tr>
<td>Lower SaO2</td>
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<tr>
<td>SaO2&lt;90 index</td>
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</tbody>
</table>

TST = total sleep time (in minutes); AHI = number of apneas and hypopneas per hour of sleep; lowest SaO2 = minimum oxygen saturation observed observed for entire night; SaO2<90 index = number of desaturations <90% per hour of sleep.

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Communications to the Editor