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 spu tum 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. Reduc tion 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,13 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 airflow, 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.

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REFERENCES

Table

<table>
<thead>
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<td>Pressure</td>
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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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Serum Adenosine Deaminase Activity with Mycoplasma pneumoniae

To the Editor:

Adenosine deaminase (ADA) is a predominant T-lymphocyte enzyme, and its plasma activity is high in diseases where cellular immunity is stimulated. Acute pneumonia due to Mycoplasma pneumoniae may be related to T-lymphocytes activity, and is different from bacterial pneumonia in its inflammatory process. It is sometimes difficult to clinically diagnose mycoplasma pneumonia in the early stage, before the rise of antibody titer to Mycoplasma pneumoniae. β-lactam group antibiotic therapy, which is most commonly used in pneumonia, is not effective on mycoplasma pneumonia. If mycoplasma pneumonia is diagnosed in the early stage, antibiotic therapy could be more effective with erythromycin or tetracycline.

In this study, we have investigated 32 cases with pneumonia (12 cases of mycoplasma pneumonia and 20 cases of bacterial pneumonia) to see the usefulness of ADA measurement in the diagnosis of mycoplasma pneumonia.

Twelve patients with mycoplasma pneumonia were diagnosed by rise of a complement fixing (CF) titer of antibody to Mycoplasma pneumoniae. Twenty patients with non-mycoplasma pneumonia did not show elevation of the CF titer, and were treated with β-lactam group antibiotics effectively. ADA activity was tested within ten days from the first symptoms in all cases. Measurements of ADA activity in sera were done by the method of Giusti.1

Serum ADA activity in the group with mycoplasma pneumonia was 30.2 ± 14.0 U/I (37°C), that of non-mycoplasma pneumonia patients 12.5 ± 3.3, and that of normal control subjects 15.2 ± 4.3 (Fig 1). ADA activity of the group with mycoplasma pneumonia was significantly higher than those with non-mycoplasma pneumonia and normal control subjects (p<0.001).

If the inflammatory process of mycoplasma pneumonia is mainly dependent on T-lymphocyte activity, it seems reasonable that serum ADA activity shows high level in this disease but not in bacterial pneumonia. These results suggest that serum ADA activity in patients with acute pneumonia may be useful for the early diagnosis of pneumonia due to Mycoplasma pneumoniae.

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REFERENCES


Geotrichosis: Who is Susceptible?

To the Editor:

As the number of immunocompromised patients increases, a parallel rise in the number of infections from opportunistic invaders seems likely. Bronchopulmonary geotrichosis is a rare disease caused by Geotrichum candidum, a fungus considered to be endogenous normal bronchial flora. Infection caused by this fungus may be either bronchial or pulmonary. Ordinarily, bronchial infection requires no treatment other than routine therapy, whereas pulmonary invasion—which can be fatal—may require specific treatment.1

Few reports of bronchopulmonary infection with G candidum have been published, and the majority of these were in patients suffering from another debilitating disease.4 Recently, the organism was repeatedly isolated in three patients who were admitted to the Veterans Administration Medical Center (VMAc), Memphis, with acute pulmonary distress. Chest radiographs demonstrated nonspecific pulmonary infiltrates which cleared with routine therapy. All patients had pre-existing pulmonary disease and adenocarcinoma of the lung.

To reevaluate the risk of pulmonary infections in immunocompromised patients, we reviewed cultures of patients admitted to two

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