are basically in agreement with his statements, we feel a few comments are in order.

The extremely high rate of specific benign diagnoses reported by Khouri et al from needle biopsies of lung nodules is not the experience of most practitioners, and is probably due to their unique expertise, as well as the fact that in most instances they used an 18 or 20 ga needle to obtain a core of tissue for histologic evaluation, rather than aspiration by a 22 ga needle for cytology, as is the practice in most centers. In less skillful hands one would expect an increased incidence of complications using their technique.

A recent negative prior radiograph increases the likelihood that the lung nodule is malignant, and would be a reason not to do needle aspiration.

One of our patients developed chest wall implantation following needle aspiration biopsy of a completely resected T1 N0 M0 lung cancer. Seyfer et al recently reported a similar occurrence. Chest wall or pleural recurrence from tumor implantation in lesions that are potentially curable certainly is a "significant factor in the decision process" and is a tragic occurrence. We continue to feel strongly that needle aspiration biopsy, with its attendant risks, should be reserved for patients who are not surgical candidates.

We agree that if specific patterns of calcification which indicate benignity can be demonstrated by either standard or high-resolution CT scans, neither needle aspiration nor thorocotomy would be required. Perhaps the use of the phantom scan will be refined enough in the future to discriminate benign from malignant lung nodules.1,4

As surgeons, we are very sensitive to local recurrence following resection of potentially curable lesions. The point of our editorial is that this is a very real possibility following needle aspiration of lung cancers, and should be an important consideration in the decision-making process for the evaluation of solitary pulmonary nodules which have a high likelihood of being malignant.

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L-Tryptophan Induced Cough and Pleural Effusions Associated with the Eosinophilia-Myalgia Syndrome

To the Editor:

I would like to report a patient with cough and pleural effusions associated with the eosinophilia-myalgia syndrome which was caused by the ingestion of L-tryptophan. L-tryptophan is an over-the-counter preparation used to treat a variety of psychiatric disorders.1

A 34-year-old white woman presented with a three-week history of severe tightness in the chest, cough and diffuse myalgias. One week prior to the onset of her symptoms she started taking one gram of tryptophan. On physical examination she appeared in pain with simple movement and was unable to take a deep breath without coughing. Chest x-ray revealed a faint interstitial infiltrate and small bilateral effusions. Her WBC count was 29,000 with 45 percent eosinophils, which dropped to 11,000 with 38 percent eosinophils on day 21. Aldolase was elevated to 15 mu/ml (normal 1.2 to 7.6). The patient improved upon withdrawal of the tryptophan.

The CDC has reported more than 300 cases of a new syndrome called the eosinophilia-myalgia syndrome associated with the ingestion of L-tryptophan products. It is interesting to speculate on the possible role of the eosinophil major basic protein in causing the considerable respiratory findings in this patient.4

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Spontaneous Resolution of Endobronchial Mycobacterium avium-intracellulare Infection in a Patient with AIDS

To the Editor:

We previously reported a case of endobronchial Mycobacterium avium-intracellulare infection (MAI) in a patient with AIDS whom we evaluated for massive hemoptysis.1 Bronchoscopic examination initially disclosed no specific abnormality with the exception of a fresh clot in the posterior segment of the right upper lobe bronchus. A second bronchoscopy performed for evaluation of recurrent massive hemoptysis revealed a clot in the same location. Bronchial artery embolization of the corresponding vessels was therefore performed to control bleeding. One month later a third bronchoscopy performed as a follow-up evaluation disclosed multiple, partially obstructing, polypoid endobronchial lesions. Endobronchial biopsies were performed. Histologic examination revealed necrotizing and non-necrotizing granulomas, and the tissue cultures ultimately grew MAI. Five months later, recurrent hemoptysis, fever, and a chest radiograph typical of bronchiectasis necessitated endobronchial examination. Polypoid masses occluding multiple bronchial segments were partially removed using biopsy forceps, which allowed drainage of purulent material from the distal airways. During the next nine months, the patient’s clinical course was dominated by recurrent maxillary sinusitis resistant to multiple courses of antibiotics (amoxicillin-clavulanate, ciprofloxacin); progressive bilateral, interstitial lower lobe infiltrates; and progressive CMV retinitis. Intravenous DHFG and prophylactic therapy with aerosolized pentamidine and oral zidovudine in addition to cyclic antibiotics (erythromycin, tetracycline, cefadroxil) and monthly gamma globulin injections for control of lower airway infection were utilized over this same period. Bronchoscopic performed for evaluation of the lower lobe interstitial infiltrates 14 months after the initial airway examination demonstrating endobronchial lesions revealed absence of polypoid airway masses (Fig 1). Transbronchial biopsies were unremarkable for infectious organisms or a specific pathologic abnormality. Cytomegalic virus was recovered from
bronchoalveolar lavage fluid cultures, but cultures for MAI organisms were negative. The patient continued to experience frequent bouts of sinusitis and minor episodes of hemoptysis. During September, 1989 his sputum cultures again turned positive for MAI. However, bronchoscopy performed on October 10, 1989 revealed no bleeding source or evidence of recurrence of endobronchial MAI.

To the best of our knowledge this is the first report in the English literature of spontaneous resolution of endobronchial MAI in a patient with AIDS. None of the medications the patient received during the time of evaluation for hemoptysis and parenchymal lung disease have demonstrated clinical bactericidal activity against MAI, although ciprofloxacin in combination with imipenem and amikacin has demonstrated in vitro activity against MAI.\(^*\)

We conclude that, similar to parenchymal MAI, endobronchial MAI may be self-limiting\(^*\) and hypothesize that in this patient it may reflect immunologic reconstitution, perhaps facilitated by zidovudine and gamma globulin.\(^*\) The other medications played a minor role at best in the eradication of this infection.

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Wandering Pacemaker

To the Editor:

An electrocardiographic pattern of irregular, multifocal, supraventricular beats with changing P wave morphology and varying R-R intervals has been referred to as wandering pacemaker. This term has been discouraged by some because it implies a mechanism which is not really known. Most patients with such arrhythmias are asymptomatic and require no treatment.

We wish to describe a highly unusual case of wandering pacemaker which presented with unusual symptoms and required urgent surgical treatment. Perhaps this should be added to the list of possible mechanisms for wandering pacemaker.

A 55-year-old man with a two-year history of frequent syncopal episodes was admitted to the hospital in congestive heart failure. He was diagnosed at that time to have dilated cardiomyopathy with sick sinus syndrome. Temporary pacing was performed immediately and drug therapy was initiated with resolution of symptoms after several days. There was some difficulty establishing steady positioning of the pacing electrode in the dilated right ventricle, and output requirements for the pacing electrode were high. A VVI permanent pacemaker was later fixed to the peritoneum of the upper abdominal wall for epicardial pacing via the subxiphoid approach. The patient was discharged without recurrence of symptoms of congestive heart failure or syncope.

One month later, the patient came to the pacemaker clinic where an examination in the supine position revealed normal pacemaker function. He stated that he had been feeling quite well except for an involuntary, pulsatile twitching of the left leg which had begun several days before. He noted that the twitching disappeared when he was lying flat but returned when he sat up, and particularly bothered him when he was trying to drive his car. He was immediately taken to the X-ray department where a fluoroscopic examination of the upper abdomen revealed no pacemaker! By following the electrode catheter from the heart, the pacemaker was found sitting at the bottom of the pelvic cavity. The pacemaker was apparently shifting position in accordance with bowel motion, and when the table was tilted 90° to supine it "wandered" back to the upper abdomen. There were no signs of ileus, but the patient was informed that surgery would be required to prevent bowel complications.

In the operating room, the pacemaker was easily "fished" out of the abdomen by pulling on the lead, and firmly repositioned on the surface of the upper abdominal muscle. Now, six months later, he enjoys driving without any recurrence of the pulsatile leg movements.

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Nasal CPAP for Severe Hypoxia

To the Editor:

We have used 100 percent oxygen nasal CPAP to avoid endotracheal intubation in seven patients with severe hypoxia and bilateral pulmonary infiltrates. Mechanically, the system employed is identical to the nasal CPAP mask conventionally used for sleep apnea, but provision is made for the driving gas to be 100 percent oxygen. Initially, we were treating AIDS patients with Pneumocystis carinii pneumonia (PCP) who had elected not to undergo aggressive life support. Subsequently, we employed nasal CPAP in non-terminal patients who simply wanted to avoid the discomfort and problems of intubation and mechanical ventilation. In each case, arterial Po2 was less than 55 torr despite the administration of 80 to 90 percent inspired oxygen by nonrebreather mask before initiating nasal CPAP. All were monitored by oximetry.

Clinical data and blood gas changes in severe hypoxia are shown in Table 1. The duration of treatment varied from three to eight days, after which the patients were transferred to conventional mask or nasal oxygen as their respiratory status improved. After the patients became accustomed to the apparatus, eating and conversation in the usual fashion was possible—a distinct advantage over a full face mask. The greatest objection voiced was that the patients looked like "Porky Pig." Remarkably, each of our patients survived to hospital discharge and none deteriorated enough to require intubation, although we do not suggest an intrinsic therapeutic value. We have not attempted the technique in uncooperative or hypoxemic patients. This approach is simply a blending of full face mask technology long used in ARDS with the recognition that human subjects are largely nasal breathers and can even be ventilated via nasal mask with relative comfort.

Nasal CPAP is likely safer than face CPAP in terms of risk of emesis and aspiration, but will not be without anticipated complications.

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