and injury are well-known complications of CO poisoning, possibly more prevalent in patients with pre-existing atherosclerotic coronary artery disease. As Dr. Marius-Nunez points out, hypoxic injury is multifactorial, probably related both to CO binding to hemoglobin and also to direct cellular effects of CO. All patients with carbon monoxide exposure should undergo routine electrocardiography to evaluate the possibility of myocardial toxicity.

It is necessary, however, to clarify the interpretation of laboratory data reported in this case. Quantitative determinations of carboxyhemoglobin saturation are typically performed with a CO-oximeter. CO-oximetry measures total hemoglobin, carboxyhemoglobin (COHb), oxyhemoglobin (HbO2), and methemoglobin by a spectrophotometric method. Arterial hemoglobin saturation with oxygen (SaO2) reported on routine arterial blood gas analysis is calculated from PaO2 and pH. This is relatively reliable for clinical use if there is no significant carboxy- or methemoglobin present. SaO2 is an estimate of the fraction of hemoglobin-binding sites occupied by oxygen of those available for oxygen association.

In a patient with CO poisoning, the calculated value for SaO2 reported by blood gas analysis must be ignored; instead, the oxyhemoglobin value available from CO-oximetry must be used. In this case, initial simultaneous values for COHb of 52 percent and SaO2 of 69 percent were reported, implying that more hemoglobin-binding sites were occupied than were available. More correctly, 52 percent of the sites were occupied by CO molecules and 69 percent of the remaining 48 percent available were occupied by O2. I suspect that the values reported for SaO2 later in the patient’s course are also incorrect estimates of oxyhemoglobin fraction. Instead of O2 saturations of 98 percent with simultaneous COHb of 23 and 13 percent, the likely correct values for HbO2 are 77 and 87 percent. The potential danger of such an interpretation is that the patient will be incorrectly considered to have adequate oxygenation. This patient was estabished from 100 percent oxygen because he was stable and SaO2 “appeared” to be 98 percent. If COHb was indeed 13 percent, HbO2 could have been no higher than 87 percent.

Furthermore, the 87 percent of sites occupied by oxygen would not release the oxygen normally because CO causes the O2 affinity of unoccupied sites to increase (left shift of oxygen dissociation curve). In the face of acute myocardial infarction, longer therapy with oxygen would have been appropriate.

Lastly, it should be noted that serious CO poisoning is treated with hyperbaric oxygen when available. Ischemic EKG changes are a standard indication for hyperbaric treatment in the patient with carbon monoxide poisoning, as are metabolic acidosis, chest pain, and significant neurologic impairment.9,10

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REFERENCES

Needle Aspiration in Lung Cancer

To the Editor:

Several statements in the recent editorial by Drs. Hix and Aaron (Chest 1990; 97:516-517) deserve further comment. The radiologic and literature supports the authors’ statement that a lung needle aspiration which does not reveal malignant cells does not rule out lung cancer. The false negative rate for needle aspiration in patients with lung cancer is relatively high (5 to 10 percent).12,13 However, the radiologic literature does not support the authors’ statement that a specific benign diagnosis of a lung tumor is very seldom made by needle aspiration. A bacterial diagnosis can be made in about 70 percent of solid infectious nodules. Drs. Khouri and Stitik et al made a specific, noninfectious, benign diagnosis in 67 percent (91 of 135 cases) of confirmed benign nodules.5 The diagnosis of noninfectious benign disease is more difficult to establish by needle aspiration than the diagnosis of malignancy, for two reasons. Benign lesions often contain calcification and are difficult to penetrate with the needle tip. A specific benign diagnosis requires a large amount of tissue for histologic analysis.

Drs. Hix and Aaron state that over one-half of lung tumors in smokers over 40 years of age are malignant. This means that nearly half of lung tumors in smokers over 40 must be benign. Which patients should have needle aspiration? Some expert chest radiologists would strongly recommend needle biopsy for all these patients (assuming prior chest radiographs were negative).4 Other expert chest radiologists reserve needle aspiration for those patients who are not candidates for surgery because of age, complicating illness, unresectability of the lesion, or suspicion that the lesion is a metastatic deposit.7 In fact, the decision regarding needle aspiration depends upon many other factors, including the patient’s feelings about the growth in his/her lung, the philosophy of the patient’s attending physician and surgeon, and the track record of the radiologist and pathologist in needle aspiration of benign and malignant lesions.

In my experience, the rare chance of tumor cell implantation in the needle tract is not a significant factor in the decision process.

One noninvasive diagnostic test which may obviate the need for needle aspiration or surgery is high resolution (thin-section) CT scan of the lung tumor. Granulomas account for the majority of benign lung tumors. A specific diagnosis of benign granuloma can be made if dense central calcification, laminated calcification, or diffuse calcification is identified within a lung tumor on chest radiography, spot films, or conventional tomography.6 CT scan is 10 to 20 times more sensitive to density differences than chest radiography. Using thin-section CT, 50 percent of solitary pulmonary nodules not seen to be calcified on chest radiographs or conventional tomograms can be shown to be diffusely calcified,7 thereby avoiding invasive diagnostic procedures.

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REFERENCES
1 Webb WB. Radiologic evaluation of the solitary pulmonary nodule. AJR 1990; 154:701-06
3 Levitt RG. Thin-section CT and the solitary pulmonary nodule. Chest 1986; 89:451-92

To the Editor:

We appreciate Dr. Levitt’s interest in our editorial, and while we
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 al1 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 skilled 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 al2 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.3,4

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.2

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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, ceftadroxil) and monthly gamma globulin injections for control of lower airway infection were utilized over this same period. Bronchoscopy 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...