Diagnosing Cervical Aortic Arch

To the Editor:

In their recent case report, Schiebler et al. stated: "Computed tomography is recommended as the only test necessary to definitively diagnose the presence of a cervical aortic arch." I would like to point out that the noninvasive diagnosis of a cervical aortic arch can also be made by two-dimensional echocardiography. Since a right cervical aortic arch is usually associated with a retroesophageal course of the aorta which then descends on the left of the spine, a barium swallow examination demonstrating a large round posterior impression on the esophageal contour helps in the diagnosis. Two-dimensional echocardiography has an obvious advantage over computed tomography: it is more "noninvasive" in the sense that no bolus contrast injection is required.

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Helium/Oxygen Therapy

To the Editor:

We support the efforts of Dr. Curtis and co-workers ("Helium-oxygen gas therapy: use and availability for the treatment of inoperable airway obstruction," Chest 1986;90:455-57) to bring a neglected therapy, the use of helium-oxygen mixtures in upper airway obstruction, to the awareness of respiratory physicians. We, too, have found these mixtures useful, not only in instances of obstruction of the upper airway by an external tumor, but also in the management of patients with intrinsic tumors of the airway, and in certain patients who develop transient upper airway obstruction at the time of removal of an endotracheal tube. In our hospital, this modality of therapy is routinely available in the intensive care unit, the recovery room, the endoscopy suite and the emergency room.

As Dr. Curtis points out, the clinical response is rapid. This may obviate the need for operation or intubation in difficult circumstances or may convert an emergency procedure into an elective one. This is a safe, effective therapy whose time has passed, but should come again.

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Primary Biliary Cirrhosis in a Patient with Interstitial Lung Fibrosis

To the Editor:

We read with interest the article by Wallaert and colleagues (Chest 1986;90:842) on pulmonary interstitial changes in patients with primary biliary cirrhosis (PBC). The authors point out that the full clinical picture of interstitial lung disease occurs very rarely in the course of PBC. We want to report our own case of PBC combined with severe interstitial fibrosis of the lung.

A white man, aged 60 years, was hospitalized for the first time in 1978 complaining of exertional dyspnea. Physical examination revealed tachypnea and fine rales over both lungs. Liver was palpable 6 cm below the costal margin. Chest x-ray film showed reticular shadowing in middle and lower lobes of both lungs. Pulmonary function tests showed reduction of lung volumes, signs of increased recoil pressure, reduced Dco, and PaO2 fall on exercise. Serum levels of ASAT, ALAT, alkaline phosphatase and immunoglobulin M were elevated. Lung biopsy revealed fibrosis interstitialis progressiva chronicia dispersa sub forma emphysma bronchiolare. Consent for liver biopsy was not obtained.

Due to a history of duodenal ulcer, the patient was treated with azathioprine (200 mg daily). Treatment resulted in normalization of liver tests. There was no improvement in PFT and chest x-ray results. After three months of treatment, the patient developed leucopenia. Azathioprine was replaced by prednisolone (60 mg daily), resulting in alleviation of exertional dyspnea, improvement of PFT results and chest x-ray picture.

Patient stopped taking drugs after seven months of treatment and did not seek medical advice until 1983. At this time, the patient complained of weight loss, itching and severe exertional dyspnea. Slight jaundice, tachypnea, tachycardia, widespread rales in both lungs and enlarged liver were found on physical examination. Chest x-ray examination showed a honeycomb pattern. Serum bilirubin level was 1.8 mg/dl, ASAT 710 IU/L, ALAT 820 IU/L, alkaline phosphatase 1,180 mIU/ml, cholesterol 3.3 g/l, HbA and /+, anty HbA + /±, tissue IF/HbA + /± and anty HbA + /±. Autoantibodies were LE /− /−, SMA /− /−, MIT /++ /−, immunoglobulins G 21.8 g/L, A 4.6 g/L, and M 4.0 g/L. Transduodenal retrograde cholangiography showed normal biliary system. Liver biopsy showed changes consistent with primary biliary cirrhosis III/IV. PFT
results showed a restrictive pattern of ventilation, increased elasticity of the lung (more advanced than in 1978), and reduction of Dco to 30 percent of predicted and resting hypoxemia.

Therapy with prednisolone (60 mg) and azathioprine (100 mg daily) was given. Itching and jaundice disappeared, liver diminished and liver tests improved. Lung changes were progressing despite treatment. After five months of treatment the patient died of respiratory failure. Autopsy confirmed diagnosis of interstitial lung fibrosis and PBC.

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Cocaine and MI

To the Editor:

Myocardial infarction is being reported in alarming numbers as a consequence of cocaine abuse.1 The pathogenesis of cocaine-induced myocardial infarction is as yet unknown, although coronary artery spasm may play a prominent role in some patients.2 This mechanism is suggested particularly in those patients who have nonobstructive coronary artery disease. Recent investigations have noted, however, that the association between coronary spasm and cocaine must be considered a temporal rather than causal one because the effect of cocaine in these patients has not been confirmed by rechallenge with the drug.3 We report an unusual patient with nonobstructive coronary artery disease who suffered two separate episodes of myocardial infarction related to abuse of cocaine.

A previously healthy 31-year-old man developed severe chest discomfort after freebaseing cocaine. He presented to the emergency room, where an initial electrocardiogram showed a right bundle branch block pattern with ST elevation in leads I, AVL, and V1 through V5. Serial electrocardiograms and cardiac enzyme tests were diagnostic of an acute anterior wall myocardial infarction. Maximal serum creatine kinase level was 2,820 IU/L (normal 0 to 125) with an MB fraction of 9.3 percent (normal 0 to 3). Echocardiogram demonstrated extensive anterior wall akinesia with an apical aneurysm. Left ventricular thrombus was not present. Coronary arteriography performed 20 days after admission demonstrated a 30 percent lesion of the proximal left anterior descending artery. The patient was prescribed coumadin with strong advice against further cocaine abuse.

Two weeks after discharge the patient again developed chest pain shortly after intranasal use of cocaine. Elevation of the ST segments in leads I, AVL and V4 through V6 recurred, with new ST elevation in leads II, III, and AVF. Additional myocardial necrosis was documented by cardiac enzyme levels. Peak creatine kinase was 586 IU/L with an MB fraction of 13.8 percent. Repeat echocardiogram showed the development of further segmental motion abnormalities of the inferior and posterior walls of the left ventricle. Diltiazem was added to the patient’s regimen and advice to abstain from cocaine use was reinforced.

Recurrent myocardial infarction has been demonstrated in persons with significant coronary artery disease who repeatedly abuse cocaine.4 In such patients, myocardial ischemia may result from cocaine-induced elevation of heart rate and blood pressure. The mechanism of myocardial infarction in patients without critical coronary narrowing is less clear. We recently reported severe coronary artery spasm with superimposed thrombosis in a 20-year-old male with normal coronary arteries who suffered recurrent myocardial infarction after using cocaine.4 The course of the patient described herein lends support to the hypothesis that use of cocaine may precipitate coronary vasospasm sufficient to produce myocardial infarction in patients with nonobstructive coronary artery disease.

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HpD-PDT for Cancer Treatment in Bronchology

To the Editor:

The recent article by Kato et al1 was of considerable interest to us since it reports the first complete tumor remission with follow-up more than five years after photodynamic therapy (HpD-PDT) for tracheobronchial cancer. In view of the fact that series published in the literature are mainly the work of only two teams, it would seem useful to report our results in a short preliminary series with HpD-PDT.

From May, 1984 to September, 1986, we treated and followed eight patients (seven men, one woman) selected according to the following criteria: tracheobronchial epidermoid carcinoma less than 40 mm in diameter inoperable because of respiratory failure or in a recurrent phase after conventional treatments, and no evidence of metastases as indicated by examinations. The exact sites of the lesions are given in Table 1. Lesion diameter (mean 26.8 mm) was assessed both by endoscopy (opened grip biopsy) and computed tomography. Endoscopically, it appeared as an irregular, vegetating area in three cases and ulcerated and infiltrating in five cases. All the patients (mean age 55.8 years, range 50 to 64 years) presented definite contraindications to surgical treatment: seven had undergone pneumonectomy for cancer, followed in three cases by chemotherapy and supravacular mediastinal radiotherapy, and one had right superior lobectomy followed by chemotherapy.

Endoscopic treatment with argon-pumped dye laser was preceded 72 h before by intravenous infusion of hematoporphyrin derivative (HpD) in doses of 2.5 to 5 mg/kg prepared from hematoporphyrin dichlorhydrate according to the method of Gregorie et al2 and Lipson et al.3 The patients were advised in writing of the risks of photosensitivity relative to HpD. Laser emission power at the tip of the optical fiber ranged from 300 to 400 mW at 632 nm (checked by a JY-20 monochromator). In five cases, irradiation was interstitial (implantation every 8 mm during 5 min), and in three cases only defocused irradiation was performed according to the same para-