young people. Two of the patients whom we described were 18 years of age; one was 20, one was 26, and one was 36 years old. I believe that this is an important point. I think that there is an increased incidence of pneumothorax in people with metastatic sarcomas. Occasionally, as occurred in two of our patients, the pneumothorax preceded the development of the metastatic nodules appearing on the chest x-ray film. I certainly concur with Hyde that pneumothorax should not be considered as a clinical manifestation of primary bronchogenic carcinoma.

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REFERENCES

Pulmonary Eosinophilic Granuloma with Hilar Adenopathy Simulating Sarcoidosis

To the Editor:

Eosinophilic granuloma limited to the lungs has been well described in the literature. Significant hilar or mediastinal adenopathy has not been documented in the absence of disseminated disease. We present the findings in an asymptomatic 17-year-old girl whose initial clinical findings and chest roentgenogram simulated sarcoidosis.

CASE REPORT

We recently evaluated a relatively asymptomatic 17-year-old girl whose chest roentgenogram simulated sarcoidosis and who subsequently was found to have pulmonary eosinophilic granuloma. Our patient sought medical advice because of ill-defined mild discomfort in the left anterior portion of the chest on exertion and a very mild nonproductive cough of several weeks' duration. The findings from physical examination were unremarkable.

A chest roentgenogram showed a diffusely abnormal parenchymal pattern that consisted primarily of tiny nodular lesions but with some increase in linear markings as well. In addition, there was bilateral hilar and right paratracheal adenopathy. Tests of pulmonary function showed a uniform decrease in all pulmonary volumes to about 50 percent of predicted and a single-breath diffusing capacity for carbon monoxide (Dsb) of 35 percent of predicted. The roentgenographic appearance of symmetric bilateral hilar adenopathy with paratracheal adenopathy and a diffuse interstitial pattern in the pulmonary fields in a relatively asymptomatic young girl was suggestive of sarcoidosis.

Transbronchial biopsy of the lung showed nonspecifically inflamed and mildly fibrotic bronchial wall and pulmonary alveolar parenchyma. Mediastinoscopy revealed enlarged lymph nodes, which microscopically showed mixed follicular and sinusoidal hyperplasia in the absence of granulomas or atypical cellular infiltration. Histocytes and eosinophils were not seen.

At thoracotomy, the presence of very hyperplastic mediastinal lymph nodes was confirmed. The pulmonary parenchyma was indurated, with numerous variegated pink and dark red irregular nodes measuring up to 0.3 cm readily visible on gross examination. Microscopically, there was extensive cellular infiltration in a distinctly focal nodular pattern. The nodules consisted primarily of pale-staining histiocytes with vesicular nuclei intermingled with eosinophils, characteristic of eosinophilic granuloma.

The severity of abnormalities on tests of pulmonary function led us to institute therapy with corticosteroids. Administration of prednisone resulted in a significant improvement in pulmonary volumes and diffusing capacity over several months, and our patient seemed to require 20 mg every other day to avert further deterioration of pulmonary function. Most recent measurements of pulmonary volumes are 70 percent of predicted, and the Dsb is 56 percent of predicted. Subsequent chest roentgenograms showed partial resolution of the diffuse parenchymal changes, a further increase in adenopathy, and progressive scarring in the upper pulmonary fields, with loss of volume and honeycombing (Fig 1). Our patient subsequently developed incomplete diabetes insipidus, which did not respond to therapy with either chlorpropamide or hydrochlorothiazide.

DISCUSSION

Eosinophilic granuloma limited to the lungs was first described by Farinacci et al in 1951, and this entity is now included in the long list of possible diagnoses in the patient presenting with a diffusely abnormal chest roent-
Management of Oral Secretions in Bronchoscopy

To the Editor:

I have found a technique which makes fiberoptic bronchoscopic procedures a little easier on both the patient and the bronchoscopist. A problem that I frequently encounter is the management of both the oral secretions and the material coughed into the mouth from the tracheobronchial tree. Admittedly, this problem can be handled in a number of ways, but the easiest way that I have found is simply to place a standard dental saliva ejector into the patient’s mouth at the start of the procedure and connect it to continuous suction, just as a dentist does. With this method the patient does not become agitated when his mouth fills, producing an overwhelming desire to spit; neither is the bronchoscopist nor his assistant required to grow another pair of hands.

There are several brands of dental saliva ejectors available which are disposable and readily obtainable from a local dental supply house.

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Interdependence of Left Ventricular End-Diastolic Volume and Heart Rate

To the Editor:

Dodge and co-workers1 took a very important step in the quantitation of modern cardiologic studies by introducing in the early 1960s a simple practical method for the assessment of left ventricular volume with the aid of biplane films, using the ellipsoid of revolution as an approximation of ventricular shape. Although many more sophisticated methods have been developed since that time, the area-length method is still the most popular.

Kennedy et al2 assessed normal values for left ventricular volume with the subject in the supine position after premedication with pentobarbital in 16 persons without cardiac disease. A mean value of 70 ml/sq m of body surface area (BSA) with a standard deviation of ± 20 ml/sq m of BSA was found. Kennedy and co-workers3 recognized that a significant correlation existed between left ventricular end-diastolic volume and heart rate (r = −0.71); however, this important interdependence has faded from the literature, and the cited normal value (70 ± 20 ml/sq m of BSA) has lived its own life afterwards.

Therefore, we determined the left ventricular end-diastolic volume in 25 normal subjects and correlated these values with heart rate. The method of acquisition of the image and the consequent work-up have already been described4 and do not differ essentially from the procedure of Dodge et al1 and Kennedy et al2 with the exception that images are obtained with a frequency of 50/sec, while Kennedy et al2 used a frequency of 6/sec. The heart rate was calculated from the R-R interval preceding the analyzed beat, determined from a simultaneously recorded electrocardiogram.

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