tetrocardiographic changes and the ventricular dysrhythmias produced by stimulating various areas of the brain.\textsuperscript{14-15} Furthermore, it has been suggested that propranolol modifies the electrocardiographic changes of patients with subarachnoid hemorrhage.\textsuperscript{16} In the case reported herein, the dysrhythmia did not recur after therapy with propranolol was begun. This observation and the evidence noted previously suggest that propranolol may be of benefit in the treatment of ventricular dysrhythmias associated with subarachnoid hemorrhage.

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REFERENCES


5 Estanol B, Marin OSM. Cardiac arrhythmias and sudden death in subarachnoid hemorrhage. Stroke 1975; 6:382-6


7 Parziel G. Life-threatening arrhythmias in subarachnoid hemorrhage. Angiology 1973; 24:17-21

8 Ranquin R, Parziel G. Ventricular fibrillo-flutter ("torsade de pointe"): an established electrocardiographic and clinical entity: report of eight cases. Angiology 1977; 28:115-8


11 Koster RW, Wellens HJJ. Quinidine-induced ventricular flutter and fibrillation without digitalis therapy. Am J Cardiol 1976; 38:519-23


16 Cruickshank JM, Dwyer GN. Electrocadio graphic changes in subarachnoid hemorrhage: role of catecholamines and effects of beta-blockade (abstract). Br Heart J 1974; 36:395

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Major Pulmonary Artery Stenosis Causing Pulmonary Hypertension in Sarcoidosis*

Thomas E. Damuth, M.D.; James S. Bower, M.D., F.C.C.P.; Kyung Cho, M.D.; and David R. Dantzker, M.D., F.C.C.P.

A chest x-ray and evidence of pulmonary hypertension developed in a woman with sarcoidosis. A perfusion lung scan revealed decreased perfusion to the right upper lobe and to the entire left lung, while a ventilation scan was normal. Pulmonary angiography demonstrated multiple bilateral concentric pulmonary artery stenoses most consistent with extrinsic compression by mediastinal granulomatous inflammation and fibrosis. The patient was treated with systemic corticosteroids for one year without improvement. Major pulmonary artery stenosis should be considered in patients with sarcoidosis who developed pulmonary hypertension and are found to have lung scans showing segmental perfusion defects in normally ventilated areas or who develop an unexplained chest pain.

Pulmonary hypertension is uncommon in patients with sarcoidosis with major reviews estimating its incidence...
Occasionally hypertension is usually shown by roentgenogram. A scalene node biopsy specimen revealed noncaseating granulomas. In 1975, the patient admitted to the University of Michigan with right 6th and 7th cranial nerve paresis. She had no pulmonary symptoms and was receiving no medication. Pulmonary function testing revealed only a mild reduction in the single breath DLco and a widened alveolar-arterial oxygen gradient (Table 1). She was felt to have central nervous system sarcoidosis, and her symptoms resolved with corticosteroid therapy.

In 1974, the patient was found to have osteosclerotic bone lesions and noncaseating granulomas were found on bone biopsy specimen. She did well until 1976 when she noted increasing shortness of breath and hoarseness. Pulmonary function testing suggested the presence of extrathoracic upper airway obstruction (Table 1), and direct laryngoscopy revealed edema of the false vocal cords and a 1-mm nodule on each true vocal cord. She was felt to have laryngeal sarcoidosis, and while receiving corticosteroid therapy, her dyspnea resolved. By March 1977, her pulmonary function tests had significantly improved (Table 1).

In January 1976, a chest bruit was heard during a routine clinic visit. The bruit was heard over the entire right hemithorax and became louder with inspiration. Cardiovascular examination revealed a mild right ventricular lift and a widely split second heart sound with accentuation of its pulmonary component. A chest roentgenogram (Fig 1) showed enlarged hilar shadows bilaterally and diffuse interstitial infiltrates. An ECG and echocardiogram were normal. A technetium perfusion lung scan showed markedly decreased flow to the entire left lung and to the right upper lung field while perfusion to

**Figure 1.** Chest roentgenogram obtained in March 1978 showing bilaterally enlarged hilar shadows and diffuse interstitial infiltrates.

**Figure 2A (left).** Normal 133Xe ventilation lung scan. B (right). Technetium perfusion lung scan revealing decreased perfusion to right upper lung field and to entire left lung.
the right lower lung field appeared normal. A 133Xe ventilation lung scan was normal (Fig 2).

Right-sided heart catheterization and pulmonary angiography demonstrated severe stenoses involving the proximal portions of both the left and right pulmonary arteries (Fig 3). There was very little perfusion of the left lung upper lobe and preferential perfusion of the right descending pulmonary artery through a significant stenotic lesion. The pulmonary artery pressure was 86/18 mm Hg, with a mean of 42 mm Hg. The pressure distal to the area of stenosis in the right descending pulmonary artery was measured and found to be 38/12 mm Hg with a mean of 19 mm Hg (Table 2). Hilar tomograms in the 55° oblique projection showed no evidence of adenopathy in the regions of stenosis.

A regimen of 60 mg of prednisone per day was begun. After six months, a repeat catheterization and pulmonary angiogram showed no change in the degree of stenosis or the vascular pressures (Table 2). The prednisone was gradually tapered to 30 mg every other day, and at one year, repeat angiograms and catheterization remained unchanged (Table 2). Pulmonary function tests done prior to right-sided heart catheterization in April 1979 revealed increased hypoxemia at rest and during exercise when compared with studies from 1976 (Table 1). The patient has remained asymptomatic except for minimal dyspnea on climbing two flights of stairs.

**COMMENTS**

Stenosis of large pulmonary arteries has been previously demonstrated in patients with sarcoidosis, but stenoses sufficiently severe to result in pulmonary hypertension have not been well documented. Battesti and colleagues have reported a patient with sarcoidosis who had stenoses of central pulmonary arteries and clinical evidence of pulmonary hypertension, but pulmonary artery pressures were not measured and the

<table>
<thead>
<tr>
<th>Table 2—Cardiac Catheterization Data</th>
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<tr>
<td>Pressure Measurements (mm Hg)</td>
</tr>
<tr>
<td>Right atrium</td>
</tr>
<tr>
<td>Systolic</td>
</tr>
<tr>
<td>Diastolic</td>
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<td>Right ventricle</td>
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<td>Systolic</td>
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<td>Diastolic</td>
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<td>Pulmonary artery</td>
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<td>Systolic</td>
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<tr>
<td>Diastolic</td>
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<tr>
<td>Mean</td>
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<tr>
<td>Pulmonary artery wedge</td>
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<tr>
<td>Right lower lobe pulmonary artery</td>
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<tr>
<td>Distal to stenosis</td>
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<tr>
<td>Systolic</td>
</tr>
<tr>
<td>Diastolic</td>
</tr>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>Cardiac output (L/min)*</td>
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<tr>
<td>Pulmonary vascular resistance (mm Hg/L/min)</td>
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*Measured by dye dilution technique with Lexington cardiac output computer.
relative contributions of central and peripheral vascular resistance to the pulmonary hypertension were not determined.

In our patient, the elevated pulmonary artery pressures resulted from increased flow resistance in the central pulmonary arteries with normal peripheral vascular resistance. We believe that peripheral vascular resistance was normal because the mean pulmonary artery pressure distal to the stenotic area in the right lower lobe was normal despite the high blood flow to this region demonstrated both by pulmonary angiogram and perfusion lung scan. In addition, the wide pulmonary artery pulse pressure with the systolic pressure elevated out of proportion to the diastolic pressure is suggestive of centrally obstructed and noncompliant pulmonary arteries and has been previously noted in two patients with pulmonary artery stenosis secondary to sclerosing mediastinitis.11

The pathologic alteration that led to the pulmonary artery stenosis in our patient cannot be definitely determined. Enlarged hilar nodes have been reported to produce extrinsic pulmonary artery compression,6-8 but in our patient, hilar tomograms did not reveal node enlargement in the area of the stenotic lesions. Exuberant granulomatous inflammation involving the walls of large pulmonary arteries may cause pulmonary artery stenosis in patients with sarcoidosis,9 but the angiographic pattern of concentric arterial narrowing speaks against this possibility. The most likely cause is fibrosing mediastinitis. This process has been reported by Schowengerdt et al10 to occur secondary to sarcoidosis and is known to produce extrinsic pulmonary artery compression which is angiographically similar to that seen in our patient.11

The prevalence of pulmonary artery stenosis in patients with sarcoidosis is not known since most patients with sarcoidosis do not undergo pulmonary angiography. In the previously reported cases, the clinical presentation suggested the presence of acute pulmonary embolism.6-9 Perfusion lung scans in these cases revealed segmental perfusion defects consistent with the presence of pulmonary emboli, but pulmonary angiography demonstrated only pulmonary arterial stenosis. In our patient, the presence of pulmonary artery stenosis was suggested by the appearance of a chest bruit and the development of signs of pulmonary hypertension without significant deterioration in pulmonary function tests.

Appropriate therapy for pulmonary artery stenosis in patients with sarcoidosis is not well defined and may depend upon the pathogenesis of the stenosis. In patients who are asymptomatic and have pulmonary artery stenosis secondary to enlarged hilar nodes, one can expect the process to resolve spontaneously and no treatment is necessary. If the stenosis is due to granulomatous inflammation in large pulmonary arteries or to fibrosing mediastinitis, therapeutic guidelines are not available. Corticosteroid therapy was instituted in our patient in an attempt to decrease granulomatous inflammation in the mediastinum. There was no regression of her disease during a full year of therapy.

**References**

1 Scadding JC. Sarcoidosis, London: Eyre and Spottiswoode, 1967:302
7 Faunce HF, Ramsey GC, Sy W. Protrated yet variable major pulmonary artery compression in sarcoidosis. Radiology 1978; 116:313
12 Goldman JI, Becklake MR. Respiratory function tests: normal values of median altitudes and the prediction of normal results. Am Rev Tuberculo 1959; 79:457
13 Higgins MW, Keller JB. Seven measures of ventilatory lung function: population values and a comparison of their ability to discriminate between persons with and without chronic respiratory symptoms and disease. Tcumset, Michigan. Am Rev Respir Dis 1973; 108:258

**Atropine-Induced Psychosis**

*An Unusual Complication of Therapy with Inhaled Atropine Sulfate*

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An acute organic brain syndrome occurred in a patient being treated with inhaled atropine sulfate. Immediate reversal of the patient's mental status was achieved with intravenous administration of physostigmine.

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