topenia developed. Three days antemortem, yeast-like organisms were grown from the blood. Approximately 36 hours antemortem a Swan-Ganz catheter was placed for hemodynamic monitoring. Despite the addition of amphotericin B to the drug regimen, his respiratory function continued to deteriorate, and he died.

The postmortem examination showed a 0.5-cm, well-circumscribed erosion on the atrial surface of the septal cusp of the tricuspid valve (Fig 2). No pulmonary emboli were present. On microscopic section, the fibrosa of the valve was focally ulcerated and covered by scant thrombotic material. Hematoxylin and eosin, Gram, and Comori methenamine silver stains of permanent sections of the erosion did not show microorganisms or leukocytic infiltrates. Postmortem blood cultures remained sterile.

**DISCUSSION**

In a previous report on Swan-Ganz catheter-induced thrombotic verrucous lesions of the tricuspid valve, Greene and Cummins speculated that thrombi form on endothelial surfaces damaged by the heart's motion against the plastic catheter. No histologic evidence was provided, however, to demonstrate actual injury to the valve. Our microscopic finding of endocardial erosions of the tricuspid valve show that an early lesion predisposing to thrombus formation may occur with a Swan-Ganz catheter. Other histologically demonstrated intimal erosions with overlying thrombi have been documented in the superior vena cava with a central venous pressure catheter and in the right pulmonary artery with Swan-Ganz catheters. Recently an editorial pointed out that the symptomatic and often fatal complications of flow-directed pulmonary artery catheters are well known, but the exact incidence of complications is uncertain.

Our two cases of this clinically silent finding suggest that the overall incidence of valvular damage is indeed higher than that previously expected.

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**REFERENCES**

1 Greene JF, Cummings KC. Aseptic thrombotic endocardial vegetations: a complication of indwelling pulmonary artery catheters. JAMA 1973; 225:1525-26
2 Greene JF, Fitzwater JE, Clemmer TP. Septic endocarditis and indwelling pulmonary artery catheters. JAMA 1974; 233:891-92

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**Bronchial Collapse in Obstructive Lung Disease**

J. Hartley Bowen, M.D.;† Brett H. Woodard, M.D.;‡ and Philip C. Pratt, M.D., F.C.C.P.§

A 57-year-old man who died suddenly with severe bilateral mainstem bronchial collapse is described, and an alteration of the elastic tissue in the membranous portion of the bronchi is identified. The morphologic abnormalities, physiologic dynamics, and potential clinical consequences of such an alteration are discussed.

The motion of the mainstem bronchi during respiration is well known, and cases of increased pliability of the membranous septum have been observed. We describe a severe case of bronchial collapse and demonstrate a specific structural alteration of the membranous septum of the bronchial wall. The potential etiology, physiologic consequences, and clinical significance are discussed.

**CASE REPORT**

In 1972 a 57-year-old man had membranoproliferative glomerulonephritis, which was medically managed for six years until progressive renal failure necessitated regular hemodialysis. He had a 60 pack-year history of cigarette smoking and chronic productive cough. Pulmonary function tests done in 1975 were indicative of obstructive lung disease with hyperinflation (Table 1). In February 1979 treatment for a nonresolving left lower lobe pneumonia included flexible fiberoptic bronchoscopy, which demonstrated easy collapsibility during expiration of both mainstem bronchi in the absence of any endobronchial masses or apparent extrinsic compression. This pneumonia subsequently clinically improved with antimicrobial therapy, but roentgenograms indicated persistent scarring.

*From the Departments of Pathology, Durham Veterans Administration Medical Center and Duke University Medical Center, Durham, N.C.
†Senior Resident in Pathology.
‡Assistant Professor of Pathology.
§Professor and Director of Pathology and Laboratory Services, Durham Veterans Administration Medical Center. Reprint requests: Dr. Bowen, Department of Pathology, Duke University Medical Center, Durham, North Carolina 27710

<table>
<thead>
<tr>
<th>Table 1—Pulmonary Function Tests</th>
<th>% of Predicted Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>Result</td>
</tr>
<tr>
<td>Vital capacity, L</td>
<td>2.37</td>
</tr>
<tr>
<td>Forced vital capacity, L</td>
<td>2.19</td>
</tr>
<tr>
<td>Functional residual capacity, L</td>
<td>4.28</td>
</tr>
<tr>
<td>Residual volume, L</td>
<td>3.78</td>
</tr>
<tr>
<td>Total lung capacity, L</td>
<td>6.15</td>
</tr>
<tr>
<td>One-second forced expiratory volume</td>
<td>1.29</td>
</tr>
<tr>
<td>Maximum mid-expiratory flow, L/sec</td>
<td>0.70</td>
</tr>
</tbody>
</table>

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Following a graft thrombosis, his condition was reevaluated for vascular access in August 1979. However, owing to poor vascular access sites, a program of long-term peritoneal dialysis was begun, during which he had no symptomatic change in his obstructive lung disease. For several hours before his final dialysis session, the patient was resting comfortably without any apparent change in his physical status, and laboratory studies performed one hour before his last dialysis showed the following results: potassium, 4.3 mEq/L; sodium, 136 mEq/L; BUN, 55 mg/dl; glucose, 81 mg/dl; chloride, 105 mEq/L; WBCs, 8,400/cu mm; hemoglobin, 6.7 g/dl; and hematocrit, 23.7 percent. While under observation in the dialysis unit, he suddenly became unresponsive, and cardiopulmonary resuscitation was unsuccessful. Laboratory studies performed at the commencement of the resuscitation showed no significant alteration in serum electrolyte values.

Autopsy findings included end-stage renal disease, hypertrophy of the left ventricle, and severe atherosclerotic coronary artery disease. There were severe fibrous adhesions in the left pleural space associated with fibrous scarring of the left lower lobe, indicative of his persistent pneumonia. Only minimal centrilobular emphysema was evident within the pulmonary parenchyma. The extrapulmonary segments of both mainstem bronchi distal to the tracheal bifurcation were severely collapsed into a fishmouth-shaped orifice (Fig 1). The bronchial cartilage was widely bowed, and the membranous septum was loose and pliable. The distal trachea also showed bowing, but it was not as completely collapsed as the mainstem bronchi. The intrapulmonary bronchial segments were of normal caliber and were not distorted.

Microscopic examination of the lungs showed a mild degree of centrilobular emphysema and severe chronic bronchitis. Sections through the areas of the collapsed mainstem bronchi showed mucous gland hyperplasia and a lymphocyte infiltrate. The membranous septum of the mainstem bronchi of our patient showed fragmentation and loss of elastic fibers (Fig 2) compared with a bronchial section from a patient of similar age without pulmonary symptoms or pathologic alterations (Fig 3). The bronchial cartilaginous rings and smooth muscle were similar in both patients, except for the degree of curvature of the cartilages. The alteration in elastic tissue did not extend distally into the first several orders of bronchi. Examination of elastic tissue in other body sites (bronchioles, pleura, alveolar septa, esophagus, and aorta) did not reveal any destruction or fragmentation.

**Discussion**

The dynamics of the extrapulmonary airway during respiration have been extensively studied in human subjects with and without various lung diseases and in animals. It is well known that the membranous septum connecting the tracheal and bronchial cartilaginous rings invaginates into the airway lumen during expiration and moves in the opposite direction during inspiration. This occurs as a result of the normal increase in intrapleural pressure above atmospheric pressure during expiration. Also, during expiration the lumens of the distal airways are narrowed. Cases of increased pliability of the membranous septum have been reported, and some authors have advocated surgical rein-
This patient had undergone only a single series of pulmonary function tests four years before his death, which did reveal severe expiratory obstruction. Since emphysema was rather mild, the ventilatory impairment was attributable mainly to the inflammatory airway involvement. Forced exhalation, or coughing, in the presence of increased intrapulmonary airway resistance applies excessive transmural pressure on the major extrapulmonary bronchi. Once these bronchi became flattened, this portion of the airway could further enhance the obstructive impairment.

A specific cause of death cannot be stated unequivocally. There were no pulmonary emboli, and electrolyte values were normal immediately preceding death. There were no coronary artery thrombi, but there was severe atherosclerotic coronary artery disease, and in such a setting an acute myocardial infarction or arrhythmia cannot be absolutely excluded. An interesting possibility is that the patient could have taken an exceptionally deep inhalation, perhaps after a cough, so that while the chest was fully inflated, his bronchi became flattened and occluded. The patient then would have been unable either to inhale or to exhale, or to speak and call attention to his problem.

We have described a patient with severe bronchial collapse secondary to demonstrated atrophy and destruction of the elastic tissue of the membranous septum. We suggest that this elastic tissue alteration may develop from the repeated inflammatory insults of chronic bronchitis.

This abnormality may contribute to an obstructive type of pattern on pulmonary function tests, and when identified by bronchoscopy should be considered as a serious complication and potential cause of sudden death.

REFERENCES

3 Herzog H. Expiratory stenosis of the trachea and mainstem bronchi due to a weakened pars membranacea. Ger Med Monthly 1960; 5:73-77
6 Bryant LR, Eiseman B, Gan HK. The significance of tracheobronchial collapse in obstructive emphysema. Med Thorac 1965; 22:244-57
Motion of Pulmonic Valve and Constrictive Pericarditis*

Yoshinori L. Doi, M.D.;† Teturo Sugihara, M.D.; and David H. Spodick, M.D., F.C.C.P.

Extreme respiratory variation in the depth of the “a” wave of the pulmonic valve echo was demonstrated in a patient with constrictive pericarditis; a mechanism for this finding is offered. Disparity in systolic and diastolic ventricular function in constriction is also useful in ruling out restrictive cardiomyopathy.

Echocardiography has contributed greatly to the diagnosis and management of pericardial effusion, yet its value in constrictive pericarditis is not well established, although several echocardiographic features have been reported1–4 (ie, thickened pericardium, flattening of left ventricular posterior wall motion during diastole, abnormal septal motion, and premature opening of the pulmonic valve). We report a case of constrictive pericarditis with echocardiographic findings which were useful in diagnosing this condition and in differentiating it from restrictive cardiomyopathy.

CASE REPORT

A 76-year-old surgeon had coronary bypass grafts to the left anterior descending artery and obtuse marginal branches of the circumflex artery. A week later, he developed pain in the chest, a pericardial rub, and atrial fibrillation, followed gradually by systemic congestion and general weakness. Treatment with prednisolone and diuretics produced no improvement. Three months after surgery, the blood pressure was 132/128/78 mm Hg, and the pulse was small but regular at 70 beats per minute. Jugular venous distention was evident, with a prominent y descent. There was slight accentuation of the pulmonic component of the second heart sound and a dull third sound. The liver was palpable, and there was mild bilateral peripheral edema. The electrocardiogram revealed sinus rhythm, a QRS axis of +90°, low voltage, and nonspecific changes in the S-T segment and T waves. A chest x-ray film was unremarkable.

An echocardiogram (Fig 1 and 2) showed a small left ventricular end-diastolic dimension (18 mm) and end-diastolic dimension (35 mm). The stroke volume was small (37 ml), but with a large ejection fraction (86 percent). Septal motion was increased (8 mm), and left ventricular posterior wall motion was flat in diastole. The epipericardium appeared thickened, but no pericardial effusion was seen. The pulmonic valve showed extreme respiratory variation in the depth of the “a” wave.

Tracings of ventricular pressure (Fig 3) showed the classic “square-root” sign of early dip and plateau. Pulmonary arterial pressure was 35/17 mm Hg, right ventricular pressure was 33/15 mm Hg, and left ventricular pressure was 110/17 mm Hg. The mean right atrial pressure was 16 mm Hg, with the “a” wave equaling 17 mm Hg. A left ventricular angiogram showed excellent ventricular contraction (ejection fraction, 90 percent). The coronary grafts were patent.

DISCUSSION

The distinctive feature of this patient with subacute constrictive pericarditis was extreme respiratory variation in the depth of the “a” wave of the pulmonic valvular echo (Fig 1). It varied from 2 mm to more than 10 mm with inspiration. The only other condition in which such an abnormally deep “a” wave can be observed is valvular pulmonic stenosis;6 however, clinical and hemodynamic evidence excluded pulmonic stenosis.

Extreme variation in the depth of this pulmonic “a” wave may be explained by one of the most important characteristics of constrictive pericarditis, ie, markedly reduced ventricular compliance. Because of this, small changes in ventricular diastolic volume may produce exaggerated increases in ventricular diastolic pressure.6 This may be particularly important in producing an abnormally deep pulmonic “a” wave during inspiration. During inspiration a small increase in right ventricular volume can produce a disproportionate increase in right ventricular diastolic pressure, which may be further increased by atrial contraction at the end of diastole, producing a deep pulmonic “a” wave during inspiration. Although in constrictive pericarditis, respiratory effects on cardiocirculatory flows and pressures may be greatly damped, the degree to which this occurs depends on the severity of constriction. In fact, in our patient a small increase in right ventricular dimension was observed during inspiration, slightly diverting the septum toward the left ventricle.

Patients with constrictive pericarditis often show abnormal septal motion,1–4 although our patient did not. Premature opening of the pulmonic valve, mentioned by Feigenbaum,1 also was not present. Our patient did show a thickened pericardium and flattened posterior

*From the Cardiology Division, Department of Medicine, St. Vincent Hospital, and the University of Massachusetts Medical School, Worcester, Mass.
†Presently at the Third Division, Department of Medicine, Osaka Medical College, Osaka, Japan.
Reprint requests: Dr. Spodick, St. Vincent Hospital, Worcester, Massachusetts 01604

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