view, the duration of symptoms varied from six months to 50 years with a mean of 17 years.

Symptoms leading to diagnosis are paroxysmal coughing and choking sensation after liquids, chronic cough, recurrent pneumonia, sputum production, hemoptysis, chest pains, postural symptoms, intermittent fever, belching and gastric complaints. Bronchiectasis and localized pneumonitis as well as lung abscesses have been found on investigation of some patients. The diagnostic tool which has been found most rewarding is barium swallow. Cine-esophagography with sequential positioning of the patient is the most dependable method of diagnosis. Conventional esophagogram may not show the fistula or demonstrate a spill into the pulmonary tree. It may be pointed out here that since pulmonary symptoms may reflect esophageal disease the esophagus should be investigated more frequently when patients present with respiratory complaints. Esophagoscopy has not been so successful; however, often the bronchial end of the fistula can be seen at bronchoscopy. Bronchoscopy may not show the fistula but more importantly, it will demonstrate the presence of bronchiectasis.

Why this lesion sometimes eludes diagnosis in adults until late is not clear. Various explanations including the initial presence of a membrane which subsequently ruptures, adaptation of the patients to the minimal pulmonary symptoms produced and possibly the obliquity of the tract permitting its closure during swallowing have been offered by previous authors. Perhaps there is not a late onset of symptoms, but rather the initial mild complaints have not been thoroughly investigated until complications appear. There is always a long duration of symptoms, as pointed out. All three patients of Le Roux and Williams had bronchiectasis and in 45 adult patients reviewed by Blackburn and Amoury, 30 had bronchiectasis and 14 had focal interstitial pneumonitis or abscesses. These complications point to a long duration of ignored symptoms.

Once diagnosis has been made, the bronchoesophageal fistula should be treated surgically. Simple excision with repair of both the bronchial and esophageal ends of the tract may be all that is required. If, however, there is additional damage of lung tissue this should be resected. Thus, in several reported cases, segmental resection, lobectomy, bilobectomy, or even a pneumonectomy has been performed in addition to resection and repair of the fistula. It cannot be overemphasized that only damaged tissue need be removed and as much lung parenchyma as possible must be preserved. Results have been uniformly satisfactory with complete remission of symptoms and no major postoperative complications.

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Sudden, Severe Aortic Regurgitation: Reversal of the Abnormal Hemodynamics by Amyl Nitrite Inhalation*

A. G. Adelmann, M.D.; E. D. Wigle, M.D.; N. Ranganathan, M.B.B.S.; and W. G. Grant, M.D.

The effect of amyl nitrite inhalation on the hemodynamics of sudden severe aortic regurgitation was studied during heart catheterization. At rest the left ventricular diastolic pressure equaled aortic pressure and exceeded left atrial pressure closing the mitral valve prematurely. Amyl nitrite inhalation transiently decreased the left ventricular diastolic pressure to the level of the left atrial pressure by reducing the degree of aortic regurgitation. This demonstrates that the severity of the aortic regurgitation is the major factor responsible for the abnormal hemodynamics observed in this condition.

In sudden, severe aortic regurgitation left ventricular diastolic pressure equals aortic diastolic pressure and exceeds left atrial diastolic pressure. The reversed diastolic gradient across the mitral valve closes the mitral valve prematurely producing a diastolic sound and the loss or diminution of the first heart sound. The diastolic murmur due to the regurgitation is usually harsh and short as the regurgitation ends abruptly with the equalization of left ventricular and aortic diastolic pressures. Surgical correction of the aortic leak will reverse these hemodynamic abnormalities. The present study was undertaken to confirm the observations of Fortuin and Craigie and show that the hemodynamic changes could be transiently reversed by the reduction in severity of the aortic regurgitation that occurs after the inhalation of amyl nitrite.

*From the Cardiovascular Unit, Toronto General Hospital; Department of Medicine, University of Toronto, and Department of Medicine, Oshawa General Hospital. Work Supported by the Ontario Heart Foundation.
Reprint requests: Dr. Adelmann, Toronto General Hospital, Toronto, Ontario, Canada

Chest, 66: 2, August, 1974

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CASE REPORT

A 34-year-old man developed a febrile illness and a murmur two months after dental extraction. This was diagnosed and treated as subacute bacterial endocarditis despite the fact that all blood cultures were negative. Over the next six months he began experiencing increasingly severe exertional retrolateral tightness with pain radiating down both arms. He was admitted to the Toronto General Hospital for investigation in July, 1968.

The clinical findings were compatible with the syndrome of sudden, severe aortic insufficiency. He had a blood pressure of 125/55 and all the arterial pulse manifestations of severe aortic regurgitation. A left ventricular heave, a diastolic murmur and a double apex beat were palpable in the anterior axillary line. The first heart sound was absent and a diastolic sound was heard at the apex. A grade 3/4 harsh diastolic murmur that stopped abruptly in late diastole was audible along the left sternal border and at the cardiac apex. There was also a grade 1/4 pansystolic murmur at the apex. The remainder of the physical examination was normal.

The electrocardiogram showed evidence of left atrial and ventricular hypertrophy and there was mild left atrial and moderate left ventricular enlargement on fluoroscopic and x-ray examination of the chest. The other laboratory findings were normal. These included routine blood cultures to rule out active bacterial endocarditis.

A combined retrograde aortic and transseptal left heart catheterization was carried out following which the patient was advised to undergo immediate aortic valve replacement. He elected to defer the operation for several months by which time the chest pain was more severe and pulmonary edema had occurred on several occasions. At the time of surgery the aortic leaflets were noted to be virtually destroyed.

HEMODYNAMIC STUDIES

The left heart hemodynamics were typical of sudden severe aortic regurgitation (Fig 1 and 2, left). The left ventricular end-diastolic pressure, which was 50 mm Hg, equaled aortic pressure at end-diastole (Fig 1, left) and exceeded left atrial pressure earlier in diastole (Fig 2, left). The mean right atrial pressure was 9 mm Hg and the right ventricular pressure was 28/11 mm Hg. Cineangiographic and indicator dilution studies confirmed the presence of severe aortic regurgitation (Fig 3, left) as well as mild mitral regurgitation. The cardiac index, measured by the Fick principle, was 3.4 L/min/M².

The patient inhaled amyl nitrite on three separate occasions. Following the first inhalation, the left ventricular end-diastolic pressure fell below aortic diastolic pressure to 27 mm Hg (Fig 1, right). After the second inhalation of amyl nitrite, the left ventricular end-diastolic pressure fell to 12 mm Hg and no longer exceeded left atrial pressure (Fig 2, right). After the third inhalation of amyl nitrite, repeat indicator dye dilution studies demonstrated a significant decrease in the aortic regurgitation (Fig 3, right) and the left ventricular end-diastolic pressure again fell below the aortic diastolic pressure to 27 mm Hg. The dramatic decrease in the left ventricular end-diastolic pressure following the second amyl nitrite inhalation was believed to be caused by a greater drug effect than on the first and third inhalation.

DISCUSSION

In patients with chronic severe aortic regurgitation the left ventricle undergoes dilatation and becomes more compliant or distensible. It thereby can accept a large diastolic volume without extreme rises in end-diastolic pressure. In sudden severe aortic regurgitation, the left ventricle has not had time to increase its compliance in relation to the diastolic overload. This fact and perhaps the tautness of the pericardium contribute to the extreme elevation of left ventricular end-diastolic pressure found in this condition. Amyl nitrite in this and in one other recent study reversed the abnormal hemodynamics of sudden, severe aortic regurgitation. The vasodilating effects of amyl nitrite reduce the end-systolic volume of the left ventricle by decreasing aortic impedance to ejection, and reduce aortic regurgitation by augmenting peripheral runoff and decreasing aortic diastolic pressure.

It has been suggested that a decreased left ventricular compliance could explain the abnormal hemodynamics observed in this condition. The acute reversal of these abnormalities by reducing the degree of aortic regurgitation suggests that the severity of the aortic leak is the determining factor in the pathophysiology of the observed hemodynamic aberrations. In addition to being severe, the aortic regurgitation must be of sudden onset as no case of chronic severe aortic insufficiency has been reported with these hemodynamic findings and in 55 cases of aortic regurgitation studied echocardiographi-
Tuberculosis in Uremic Pericarditis*

Stanley M. Knoll, M.D.** and Alvin J. Slovin, M.D., F.C.C.P.†

A case of tuberculous pericarditis proved by pericardial fluid culture in a uremic patient is presented. The use of the subxyphoid pericardial window allows for more complete etiologic evaluation of the pericarditis with minimal morbidity.

Pericarditis is a complication of acute and chronic renal failure in from 16 percent to 50 percent of reported cases.2,4 Attempts at establishing a tuberculous etiology in uremic pericarditis have been largely unrewarding. Published series of patients with documented tuberculous pericarditis fail to associate that disease with renal failure.1,3 This report describes a case of tuberculous pericarditis in a patient with chronic renal failure.

CASE REPORT

A 23-year-old Negro woman was admitted on April 9, 1973, with a two week history of chest pain and dyspnea. She had been well until February 21, 1973 (6 weeks prior to present admission), at which time she was evaluated for multiple complaints of one week's duration and was found to be in renal failure. Her BUN at that time was 165 mg percent, creatinine 34 mg percent, potassium 7.2 mEq/L, hemoglobin 5.3 gm percent, hematocrit 15.7 vol percent. Chest x-ray film was normal. Renal biopsy revealed rapidly progressive proliferative glomerulonephritis. Workup for collagen disease was negative. The patient was begun on chronic hemodialysis on February 23, 1973. Dialysis was carried out weekly for several weeks, with the patient being in terminal renal failure at the time of hospitalization. Clinical examination revealed a well developed, well nourished Negro woman in no distress. The blood pressure was 100/60 mm Hg, pulse 88, respirations 16. The chest was clear to auscultation. The heart was not enlarged but showed evidence of right ventricular hypertrophy.

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*From the Department of Surgery, The George Washington University Medical Center, Washington, D.C.

**Instructor in Surgery.

†Assistant Professor of Surgery.

Reprint requests: Dr. Slovin, 2150 Pennsylvania Avenue NW, Washington, D.C. 20037

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