pulmonary parenchymal involvement in the disease. Patterson and co-workers found diffuse lung involvement in nine of 702 rheumatoid patients and noted more common occurrence in men.

Pleuritis with effusion, the most common pulmonary manifestation, is often transient, asymptomatic and develops at any stage of rheumatoid disease. In many instances pleural biopsy reveals only chronic inflammatory changes but occasionally rheumatoid pleural nodules are found. The effusion which often accompanies pleuritis characteristically contains little or no measurable glucose; presumably this is the result of impaired pleural transport of the substance. Elevation of lactic dehydrogenase and lipids, eosinophilia and positive latex fixation tests are also found. Cell counts are rarely helpful although RA cells have been demonstrated in these effusions. Effusion without underlying pleural or parenchymal disease is uncommon.

Other patients may present with diffuse pulmonary fibrosis. In these, nodules are rarely present on roentgenography. Of interest is the frequently positive latex fixation test found in some anarthritic patients with pulmonary fibrosis. The relation of rheumatoid arthritis to conglomerate pulmonary fibrosis remains elusive.

In 1953 Caplan described a syndrome of massive pulmonary fibrosis and rheumatoid arthritis in coal miners. The nodular involvement in this variety tended to be more discrete than the usual variety of pulmonary fibrosis, with frequent coalescence and caviation of the nodules. In some of Caplan and associates' patients rheumatoid factor without arthritis was demonstrated.

Rheumatoid lung nodules may occur in the absence of diffuse parenchymal changes. Although infrequently seen in the absence of pneumoconiosis, these nodules favor corticopleural surfaces and posterior lung segments and frequently cavitate, as do the lesions described by Caplan. Histologically, these necrobiotic nodules contain a central zone of fibrinoid degeneration or necrosis, an immediate zone of proliferating cellular elements and a peripheral zone of inflammation.

Solitary rheumatoid nodules are rarely seen in the lung. In 35 patients with rheumatoid lung lesions reported by Martel and colleagues only one solitary nodule was found. Cavitation of these lesions has been recorded. Solitary lesions in the upper lung fields were confirmed in patients described by Mattingly and Flatley. Although the nature of this solitary nodule was suspected clinically from the time of its discovery, only open biopsy finally could prove the diagnosis. Not uncommonly the clinical syndromes of myopathy or arthropathy secondary to bronchogenic carcinoma might well simulate rheumatoid arthritis. Delegation of such lesions thus becomes increasingly important.

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Isolated Tricuspid Bacterial Endocarditis Resulting in Severe Tricuspid Insufficiency*

D. M. Bramwell-Jones, M.B., Ch.B., F.C.C.P.; W. A. Pocock, M.B.; D. J. Salanti, M.B., B.Ch.; and J. B. Barlow, M.D.

An unusual case of acute staphylococcal endocarditis is reported. Neither the portal of entry for the organism nor septic pulmonary emboli were found. Severe organic tricuspid incompetence developed, the noteworthy features of which included premature opening of the pulmonary valve, a soft pulmonary closure sound and low QRS voltages. The prognosis is probably good and we favor conservative therapy.

*From the Cardiovascular Research Unit, Department of Medicine, University of the Witwatersrand; and the Cardiac Clinic, Johannesburg General Hospital, Johannesburg, South Africa.

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Reprint requests: Dr. Bramwell-Jones, General Hospital, Johannesburg, South Africa
isolated right-sided bacterial endocarditis is an uncommon clinical entity and is reputed to comprise only 5 percent of all cases of bacterial endocarditis. It is a well recognized complication of narcotic addiction and has been observed in 40 percent of addicts presenting with endocarditis. The condition presents as an acute rather than as a subacute infection and, because the infecting organism is frequently a virulent one, normal valves are often affected. Septic pulmonary embolism usually supervenes.

We report an unusual case of acute tricuspid endocarditis in which investigation failed to reveal the portal of entry of the organism. Severe tricuspid incompetence resulted and the more important clinical and hemodynamic features of this rare lesion are described and briefly discussed.

CASE REPORT

A 33-year-old African woman was completely well until she developed a rigor on March 4, 1971. When admitted to hospital on the fifth day of her illness, she was pyrexial with a sinus tachycardia of 180 per minute. A grade 1 blowing systolic murmur and a right-sided gallop rhythm were audible at the lower left sternal border but the jugular venous pressure was not raised, the liver was not enlarged and no clinical evidence of right ventricular hypertrophy was detected. A soft spleen was palpable. The electrocardiogram showed a mean frontal plane QRS axis of +110°, clockwise rotation and inverted T-waves in leads V₁ to V₄. A chest roentgenogram was normal. Laboratory tests revealed a hemoglobin of 10.7 gm percent, white cell count of 19,000, sedimentation rate (Westergren) of 100 mm in the first hour. Coagulase positive Staphylococcus aureus was cultured repeatedly from her blood. In spite of careful investigation, including pelvic examination, no portal of entry for the Staphylococcus was detected. Intravenous therapy of 2 million units of penicillin every four hours and 4 gm of cloxacillin every four hours was started three days after admission and continued for six weeks. The pyrexia settled almost immediately and subsequent blood cultures were negative. Although the septicemia was cured, the signs of right ventricular "decompensation" increased. She had a sustained tachycardia of at least 120 per minute. Prominent systolic waves developed in the jugular venous pulse and the retrosternal impulse became forceful. The systolic murmur increased to grade 3 in intensity, the right-sided gallop persisted and the second heart sound became clinically "single" due to an inaudible pulmonary component. The chest roentgenogram remained normal but the electrocardiogram showed a progressive decrease in QRS voltages.

Cardiac catheterization was carried out on April 14, 1971. The mean pulmonary wedge pressure was 8 mm Hg. The right ventricular systolic pressure was 25 mm Hg and the end-diastolic pressure was raised to 18 mm Hg (Fig. 1). The
prominent "v" waves of 22 mm Hg in the right atrial tracing were virtually diagnostic of severe tricuspid regurgitation (Fig 2). There was no systolic gradient across the pulmonary valve but a noteworthy feature was the slight rise in pulmonary artery diastolic pressure from about 15 mm Hg in early diastole to 18 mm Hg in end diastole (Fig 1). Cineangiography performed from the right atrium failed to reveal any chamber enlargement but prolonged opacification of the right heart was compatible with the considerable tricuspid incompetence. A right ventriculogram was technically impossible because the catheter tip could not be maintained in the ventricular cavity and was repeatedly forced back into the right atrium by the strong regurgitant stream. A pulmonary angiogram demonstrated an entirely normal vascular tree. A phonocardiogram, recorded on April 27, 1971, showed a normal split second sound with a soft pulmonary component. The systolic murmur, right-sided gallop sound and a short mid-diastolic murmur, all of which increased in intensity with inspiration, were demonstrated (Fig 3).

She was discharged on maintenance digoxin and remains well and asymptomatic. Examination ten months after the onset of her illness revealed the liver to be palpable three finger breadths below the right costal margin and pulsatile. The first heart sound was soft and the systolic murmur of tricuspid incompetence was intermittent and barely audible. The second sound was still clinically single and the right-sided gallop has remained loud.

**DISCUSSION**

The presentation and subsequent course of this patient's illness is compatible with acute staphylococccic endocarditis which caused destruction and severe incompetence of the tricuspid valve. It is probable, but unproved, that the valve was previously normal since the valve incompetence was intermittent and barely audible. The second sound was still clinically single and the right-sided gallop has remained loud.

The systolic murmur, right-sided gallop sound and a short mid-diastolic murmur, all of which increased in intensity with inspiration, were demonstrated (Fig 3).

Most of the clinical and cardiac catheterization features in our patient have been detected in other cases of severe isolated tricuspid incompetence. It is noteworthy that her systolic murmur, which was grade 3 in intensity about five weeks after admission, was barely audible at the last examination and this may well relate, as intimated by Brandenburg and associates, to minimal turbulence arising from a grossly incompetent valve between two low pressure chambers with essentially similar pressures. Phonocardiography revealed normal splitting of the second sound but with a soft pulmonary component. Since this soft pulmonary closure sound occurred just after the normal, but relatively much louder, aortic component, it is not surprising that the second sound appeared "single" to clinical auscultation. We have observed that the published* phonocardiograms of other cases of isolated severe tricuspid regurgitation also demonstrate a soft pulmonary component and we believe that this is an important diagnostic sign of this rare condition. Organic rheumatic tricuspid regurgitation very seldom occurs without associated mitral valve involvement, in which instance, as well as in patients with functional tricuspid regurgitation, whether resulting from myocardial disease, chronic rheumatic endocarditis or pulmonary arterial hypertension, the pulmonary vascular resistance must invariably be raised or, at least, normal. The pulmonary closing pressure would thus rarely be low and the intensity of the closure sound is consequently usually normal or increased.

Because our patient's first electrocardiogram was recorded only five days after the onset of her illness and at a time when her tricuspid regurgitation was mild, we were able to observe the decrease in her QRS voltages. Reduced QRS voltages, ascribed to an increase in cardiac volume, have been reported in congestive cardiac failure. Low amplitude QRS complexes have also been recognized in isolated tricuspid regurgitation, without elevation of the right ventricular end diastolic pressure, but have not been explained. We believe that an important factor in the production of low voltages in isolated tricuspid incompetence is low myocardial contractile tension of the left ventricle consequent on its small end

**Figure 3.** Medium frequency (MF) phonocardiogram recorded at the left sternal border (LSB) and pulmonary area (PA). The first heart sound (I) is soft. The second splits 0.04 seconds on inspiration and closes to 0.02 seconds on expiration. The pulmonary component (P) is decreased while the aortic component (A) is of normal intensity. During inspiration the right-sided gallop (G) increases in intensity. The soft systolic murmur (SM) and low frequency vibrations surrounding the gallop sound are also slightly louder on inspiration. Time intervals 0.04 second.
diastolic volume. After replacement of the tricuspid valve an increase in QRS voltages is apparent on several published electrocardiograms. Since the pulmonary artery pressure during late diastole was an unusual finding in our patient. Provided that a pulmonary valve was competent, the usual fall in a pulmonary arterial pressure after pulmonary valve closure must result from forward flow of blood into the pulmonary capillary and venous systems. If the pressure in the pulmonary artery to rise during diastole, both a significant resistance to this forward flow well as a volume displacement into the pulmonary arterial system must be present. The mean pulmonary wedge pressure of 8 mm Hg would constitute resistance to forward flow and we suspect that this level may well have been lower but for the "restrictive" effect of the stretched normal pericardium. A volume displacement could be provided by the billowing of the pulmonary leaflets upwards into the lumen of the main pulmonary artery followed by the forward flow of blood from the right ventricle after the diastolic opening of the valve as the right ventricular diastolic pressure exceeded that in the pulmonary artery. Parry and Abrahams and Somers and co-workers have observed similar pulmonary pressures in severe endomyocardial fibrosis of the right ventricle and appreciated that the pulmonary valve must open in late diastole.

Because of the relatively small number of documented cases of isolated tricuspid incompetence the long-term prognosis is uncertain, but we favor the contention of Morgan and Forker and that of Croxson and colleagues that the condition is relatively benign and that long-term survival without surgery should be possible. Both these groups have reported patients with post-traumatic tricuspid insufficiency who were alive and relatively asymptomatic after more than 30 years. If we were confident that a successful valvuloplasty, as described by Croxson and co-workers, could be performed in our patient, there might be good reason to refer her to surgery now. However, this is unpredictable and we consider that insertion of a prosthetic valve is probably not justified at present.

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Solitary Pulmonary Nodule Due to Phycomycosis (Mucormycosis)*

Adelito M. Gale, M.D., F.C.C.P.** and William P. Kleitsch, M.D., F.C.C.P.†

The case of an asymptomatic, solitary nodule appearing in the lung of a diabetic hospitalized for arteriosclerosis obliterans is presented. The nodule was found to be due to Phycomycoses of the Mucoraceae strain and is the fourth case reported of pulmonary disease due to this organism to recover after treatment. It is the only instance known of this disease appearing as a solitary nodule and emphasizes the fact that the possible etiology of such nodules is endless, and cytologic and bacteriologic identification of them remains important.

The need for surgical excision to establish the diagnosis of solitary pulmonary nodules has been repeatedly emphasized. This suggestion has been received with varying degrees of resistance for several reasons. Some argue that the surgical hazards in a population group in the sixth, seventh and eighth decade with a limited survival potential outweigh the risks of malignancy. Most objections to thoracotomy for diagnosis of solitary nodules revolve about the malignant vs non-malignant controversy. This has generated considerable heat and furor and has practically obscured the fact that solitary pulmonary nodules undiagnosed are still undiagnosed whether benign or malignant. Rational management of disease requires a diagnosis, cytologic and

*From the Thoracic Surgical Service, Veterans Administration Hospital, Phoenix, Arizona.
**Thoracic Surgeon (now in San Diego, California).
†Chief, Surgical Service.
Reprint requests: Dr. Kleitsch, VA Hospital, Phoenix 85012

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