

MYXOMA OF THE LEFT AURICLE WITH DIRECT PRESSURE TRACINGS

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Cardiac myxoma has been reported with increasing frequency in recent years. Pritchard (1951) reviewed some 200 cases then reported. At least twenty-eight further examples have been published in Great Britain and the United States of America; of these nineteen were in women and two-thirds of them died between the ages of thirty and sixty years.

Straus and Merliss (1945) attempted to estimate the true incidence by comparing the statistics of the American Medical Association for the number of autopsies performed in the United States of America with the number of cases of primary cardiac tumour reported in the same five-year period, 1938-42. The autopsies numbered 480,331 and 8 cases of primary cardiac tumour were found, giving an estimate of 17 per million. This may be regarded as a minimum figure and is equivalent to about 10 patients per annum in Great Britain.

We report a case that exhibited most of the characteristic clinical features. Few of the published accounts have included intra-cardiac pressure measurements, and none has been found in which, as in this instance, direct measurement of the left auricular pressure was made by means of a needle passed through the left bronchus.

Case Report

The patient, a Jew, was aged 45. He was first admitted to the General Infirmary at Leeds in April, 1951, with a vague history of intermittent dizziness of several months duration, followed by the sudden onset of right frontal headache and impaired vision three days previously. There was no history of rheumatic fever and no cardiac symptoms were present, but an apical presystolic murmur was queried. The blood pressure was 120/80. He was found to have a homonymous, roughly quadrantic defect of the upper left visual field which afterwards persisted.

Effort dyspnoea began early in 1954 and in June of that year there were two episodes of sudden dyspnoea followed by cough and white frothy sputum. He was examined then and found to have pre-systolic and systolic murmurs at the apex. He had sinus rhythm, a blood pressure of 120/85, and fine râles at both bases but no œdema. He was admitted to hospital for a month and improved readily on routine treatment. At that time fluoroscopy showed considerable left auricular enlargement with some right ventricular hypertrophy and pulmonary congestion, and the X-ray examination was considered to be in keeping with the diagnosis of mitral stenosis. The electrocardiogram showed sinus rhythm and right axis deviation only.

From this time dyspnoea became progressively worse. Œdema appeared in July, 1955, and increased steadily. In that month he lost consciousness suddenly when sitting in a chair, fell to the floor, and was unconscious for a quarter of an hour. A similar but very short loss of consciousness occurred in December, 1955. He was admitted to the Herzl Moser Hospital at Leeds in November, 1955, for treatment of congestive heart failure, and discharged nine weeks later somewhat improved, but still in failure.

In January, 1956, he was admitted to the General Infirmary at Leeds for further investigation and possible valvotomy. The physical examination showed sinus rhythm with multiple extrasystoles and a blood pressure of 115/70. The apex was in the fifth space in the anterior axillary line with a tapping cardiac impulse. The first sound was accentuated at the apex and a third sound was present in early diastole. There was a moderate, grade 3, apical systolic murmur and soft rumbling mid-diastolic and presystolic

murmurs. The sounds in other areas were normal. There were a few basal crepitations. The jugular pressure was not raised, but there was a little sacral œdema, and a smooth firm hepatic enlargement to three fingers breadth below the costal margin.

Fluoroscopy showed much the same general contour as in 1954 but with a considerable increase in left auricular and right ventricular enlargement, and it was thought that the left ventricle might also be enlarged. Again the picture was thought to be in keeping with rheumatic mitral disease. Electrocardiography now showed marked right ventricular hypertrophy. Auricular fibrillation developed in February, 1956. Left auricular and pulmonary artery pressure tracings were taken in March (Fig. 1 and 2), and showed a pulmonary artery pressure of 106/54 mm. Hg and a left auricular pressure of 61/20 mm. Hg. The left auricular tracing appeared to show considerable incompetence although the shape of the curve was unusual. He died suddenly two weeks later.



FIG. 1.—Left auricular pressure tracing taken in a case of myxoma of the left auricle, showing pressure of 61/20 mm. Hg.

Necropsy Findings

The heart weighed 560 g. The left atrium was enlarged and occupied by a large polypoid tumour mass, 9.5 × 6 × 5.5 cm., attached by a short narrow pedicle to the inter-atrial septum just below the fossa ovalis (Fig. 3). The tumour was greyish yellow, smooth, and elastic, with a transverse annular groove corresponding to the mitral ring. Old and recent hæmorrhages were apparent in the substance of the tumour, the latter being particularly notable in the apex of the tumour which projected through the mitral valve. There was no evidence of rheumatic heart disease. The right ventricle was moderately dilated and hypertrophied. The right coronary artery was completely occluded 4 cm. from its origin, and the other coronary vessels displayed patchy atheroma.

A number of small pale infarcts were present in both kidneys. There were old areas of softening in both occipital lobes of the cerebrum and in the left lobe of the cerebellum.

Discussion

Successful removal of a cardiac myxoma has only recently been reported, (Chin and Ross, 1957). A number of attempted removals had previously been described but had been unsuccessful, in

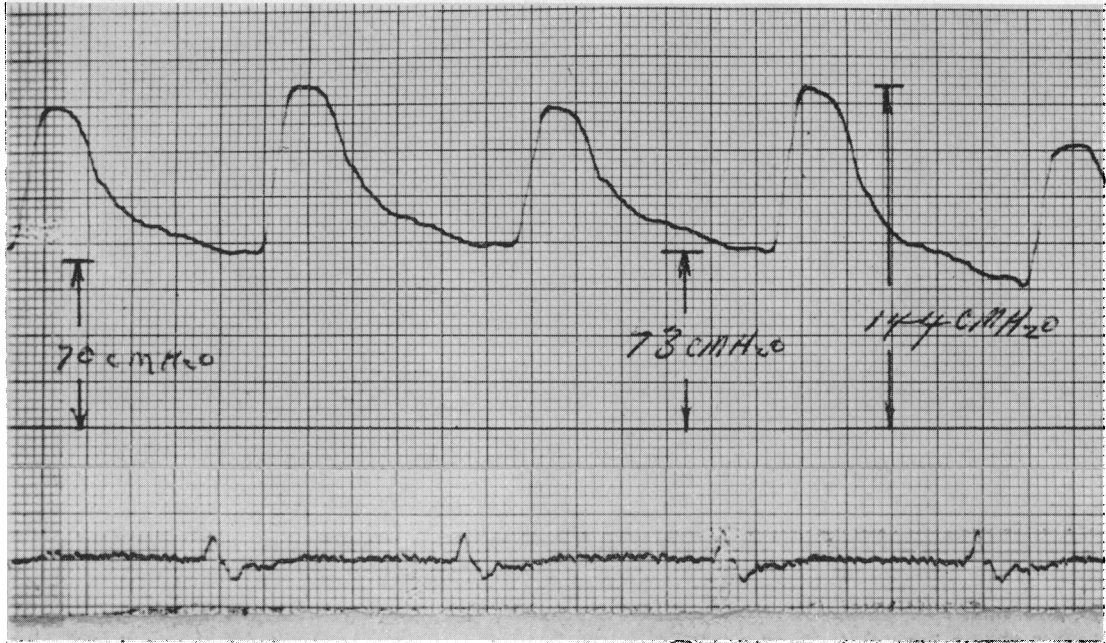


FIG. 2.—Pulmonary artery pressure tracing, showing pressure of 106/54 mm. Hg.

some cases owing to the friability of the tumour and the production of massive embolism. Nevertheless, treatment is clearly surgical as the natural history is uniformly gloomy, survival rarely exceeding eighteen months from the onset of dyspnoea.

Myxoma of the left auricle clinically simulates mitral valvular disease very closely and there are no distinguishing features upon which complete reliance may be placed. The possibility should be considered when the history is short, the course is rapid and relentless, and the response to medical treatment poor. Arrhythmias are uncommon and late. Radiologically left auricular enlargement remains slight or moderate when failure is severe. Alteration in physical signs with posture is observed in about half the cases, and sudden attacks of dyspnoea, cyanosis, præcordial pain, and tachycardia may occur. Embolic phenomena are frequent and may, as in this case, precede other evidence of cardiovascular disease.

Clinical diagnosis is, however, very rare; probably because the possibility is not considered. Kirkeby and Leren (1952) were able to make the diagnosis in their case on the basis of changing murmurs, but only in the last forty-eight hours of life.

The electrocardiogram gives little assistance in the diagnosis although the comparatively rapid development of the electrocardiographic pattern of right ventricular hypertrophy was notable in this case. In the left auricular pressure tracing several features were of interest. The rise of systolic pressure was very rapid, the *c* wave was higher than the *v* wave, and the *x* descent was well preserved; in these respects the tracing differed from those commonly seen in rheumatic mitral regurgitation and in retrospect may have been produced by a ball-valve action of the protruding portion of the tumour in the mitral orifice. In addition, the pressure in the left auricle was very high but enlargement of the left auricle was only moderate, unlike rheumatic mitral regurgitation where the left auricle is usually considerably enlarged and the pulmonary artery and left auricular pressures are not extreme. Some of these features may prove to be of diagnostic value.

When the diagnosis has been suspected, angiocardiology has usually been successful in demonstrating the tumour (Bahnon and Newman, 1953; and Steinberg *et al.*, 1953).

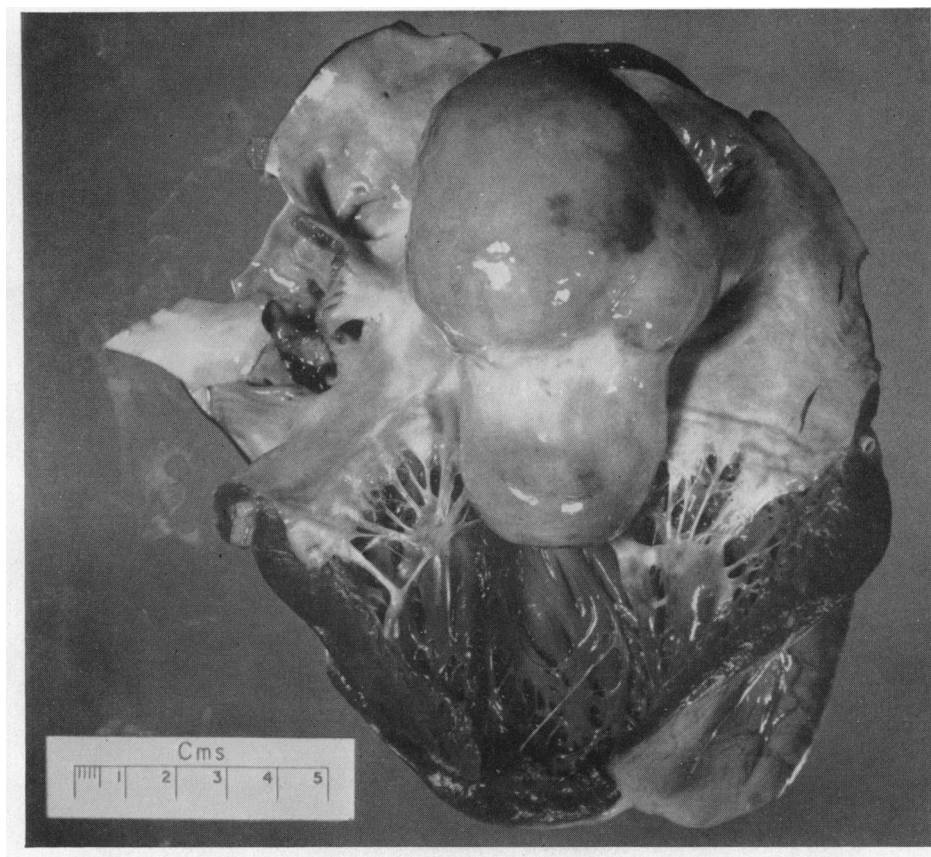


FIG. 3.—The enlarged left auricle is laid open to show a large polypoid myxoma attached by short pedicle to the inter-auricular septum.

Summary

We report a case of myxoma of the left auricle in which there were features of special interest.

The presenting symptoms and signs were those of embolism, preceding by three years the onset of dyspnoea. Recurrent dyspnoea of great severity eighteen months prior to death was followed by progressive failure, little influenced by therapy. Auricular fibrillation was delayed until two months before death.

Left auricular pressure and pulmonary artery pressures were measured by the transbronchial route, and were very high, the figure in the pulmonary artery being 106/54 mm. Hg, and in the left auricle 61/21 mm. Hg.

The tumour contained recent hæmorrhages especially in that part which projected into the left ventricle, possibly resulting in complete occlusion of the mitral orifice.

We are indebted to Mr. G. H. Wooler for the transbronchial pressure measurements, and to Dr. H. Thompson for the report on the necropsy.

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