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Dr J R Hobbs (*Royal Postgraduate Medical School of London*) said that the typical skin lesions brought on by exposure to cold indicated the need to examine serum and oxalated plasma separated at 37°C so that cryoglobulins or cryofibrinogen could be detected. Serum cryoglobulins might be mixed (polyclonal) or monoclonal (Meltzer *et al.* 1966). The formation of a crystalline precipitate in this patient's serum, its narrow electrophoretic mobility and the finding of only one class of heavy chain (γ) and only one type of light chain(λ) confirmed this as a monoclonal cryoglobulin.

Monoclonal cryoglobulins were either of γG or γM class and about 95% were associated with malignant neoplasia within the reticuloendothelial system: about 2% of γG-myelomatosis had symptomatic cryoglobulinæmia. That malignancy had been developing, albeit slowly, in this patient seemed probable from the findings of March 1967 which established (1) Bence-Jones proteinuria, (2) reduction of normal immunoglobulins, and (3) that the serum level of M-protein had risen to 1 g/100 ml. In several similar patients cytotoxic treatment had reduced the serum level of M-protein and relieved symptoms.

Calcified Right Atrial Myxoma Producing Tricuspid Incompetence

D C Fluck MB MRCP
and L Lopez-Bescos MB
(for C G Baker OBE MD FRCP)
(*Guy's Hospital, London*)

Mr F C, aged 41. Charge-nurse

History: In 1957 this patient fainted while sawing wood and, later on, while walking to work. He saw his general practitioner who heard a murmur. He remained well until June 1966 when he began to notice some breathlessness on exertion. In December 1966 he started to have occasional episodes of rapid palpitations associated with a feeling of emptying in the chest. These attacks would last about 20 seconds and afterwards he would feel ill for about one hour. In October 1967 he was admitted to the Royal Sussex County Hospital, Brighton (Dr R Kemball Price) with a five-day history of melæna. The gastrointestinal hæmorrhage was not severe, the stools quickly became negative for occult blood and the Hb only fell to 76%. A barium meal was normal. On screening of the chest, a calcified mass was seen in the heart which swung with a free range of movement during cardiac contractions. 1.11.67: Transferred to Guy's Hospital (Dr C G Baker).

On examination: Cardiovascular system: Jugular venous pressure – marked 'a' and systolic waves; pulse normal; sinus rhythm, 90/min; moderate right ventricular hypertrophy; no left ventricular hypertrophy; first and second heart sounds normal; systolic murmur, louder and full length pansystolic on inspiration, maximum left sternal edge; early diastolic sound internal to apex. Respiratory system normal. Abdomen normal. Central nervous system normal.

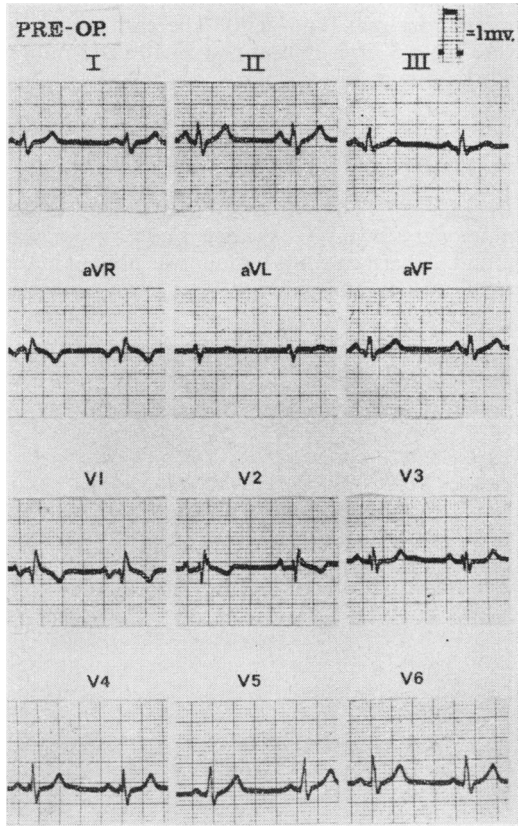

Fig 1 *Electrocardiogram prior to operation showing sinus rhythm and right bundle branch block*

Investigations: ECG (Fig 1): sinus rhythm, right bundle branch block. Chest X-ray (Figs 2, 3A, B): cardiothoracic ratio 16·5/29 cm; calcified lesions right apex, calcified mass within heart shadow. Screening of heart: calcified mass 5 cm in diameter in the position of right atrium; it appeared to prolapse through the tricuspid valve into the right ventricle. Ciné venous angiocardiography (Dr John Dow): a large calcified atrial myxoma is seen, which during atrial systole is prolapsing partly into the right ventricle through the tricuspid valve.

Plasma proteins normal. ESR 3 mm in 1 hour (Westergren). Hb 15·4 g/100 ml. Normal film.

Right heart catheterization: right atrial pressure showed marked systolic wave (a 17, x 10, cv 25, y 5 mmHg). Right ventricular pressure 28/6 mmHg (right ventricular end diastolic pressure 11 mmHg). Pulmonary vascular resistance 2·3 units. Cardiac index 2·2 litres/min/m^2.

Exercise test: physical working capacity at a heart rate of 170 beats/min 640, 760 kilopondmetres per min (kpm/min). The cardiac output rose from 3 litres/min at rest to 10 litres/min at 400 kpm/min.

The patient was seen by Lord Brock, who advised removal of the right atrial myxoma.

Operation (7.12.67, Mr A K Yates) (under cardiopulmonary bypass): A large right atrium was found. A hard calcified tumour was present in the right atrium and inferior vena cava and was

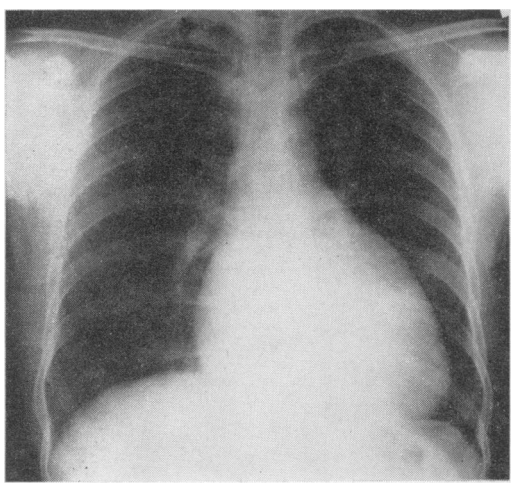

Fig 2 *Chest X-ray 20.10.67, prior to operation. The heart is enlarged and a calcified lesion is present at the right apex*

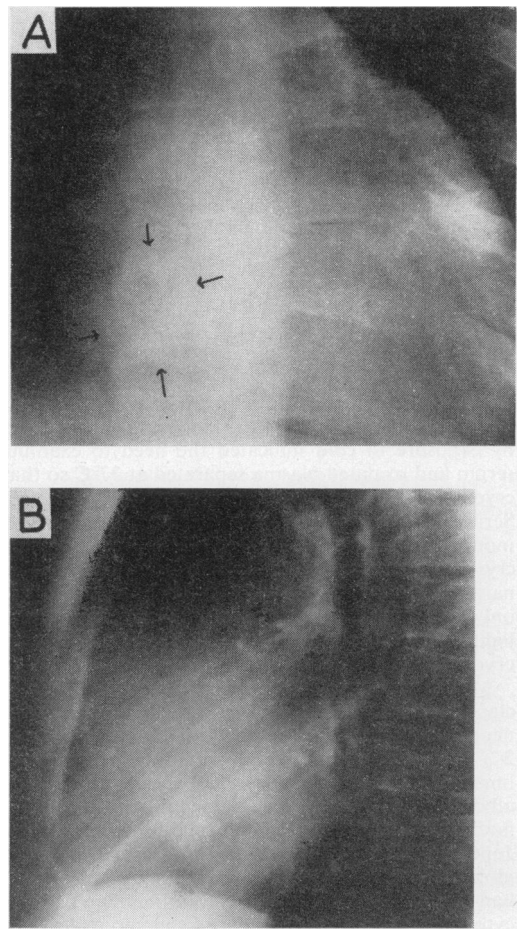

Fig 3 *Chest X-ray.* A, *enlarged postero-anterior view.* B, *enlarged lateral view. A calcified mass is visible within the heart shadow*

attached by a pedicle to the posterior margin of the fossa ovalis. The tricuspid valve had a large ring, the cusps being thickened and fibrosed. The chordæ appeared intact, except for a single ruptured chorda of the posterior margin on the septal cusp. The myxoma was removed. Tricuspid regurgitation was observed to be present following removal of the tumour. The tumour was pear-shaped with a narrow pedicle; it weighed 34 g and measured about $57 \times 38 \times 30$ mm (Fig 4A); it was heavily calcified (Fig 4B). Histology (Dr G A K Missen): the tumour showed the features of a necrotic myxoma with a viable capsule. Following the operation, the patient's course was unsatisfactory, with clinical evidence of a low cardiac output.

Second operation (19.12.67, Mr A K Yates) (under cardiopulmonary bypass): Starr valve inserted

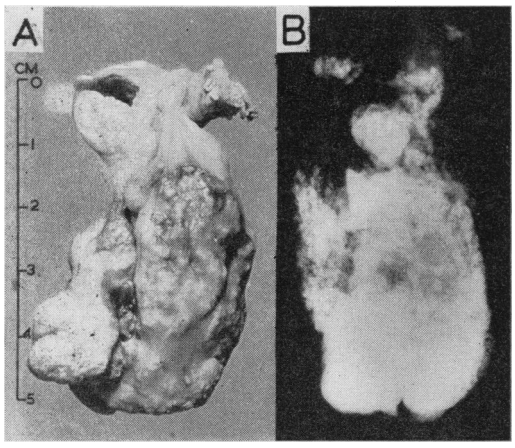

Fig 4 A, *the right atrial myxoma.* B, *X-ray of the right atrial myxoma showing heavy calcification*

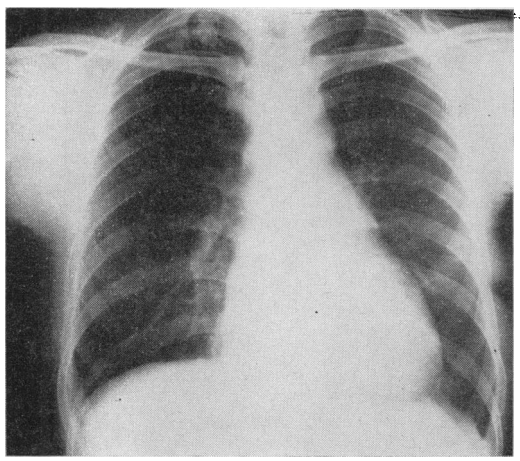

Fig 5 *Chest X-ray 28.12.67, nineteen days after second operation, showing that size of heart has returned to normal*

into tricuspid ring, after an unsuccessful attempt to render the tricuspid valve competent by annuloplasty. Following this operation, the patient improved and made a slow but complete recovery, the post-operative chest X-ray showing a return to a normal heart size (Fig 5).

Discussion

The patient's symptoms were probably related to the tumour. The faints in 1957 may have been due to temporary occlusion of the tricuspid valve and the episodes of palpitations may have been related to right atrial distension as a consequence of the tricuspid regurgitation. The systolic murmur became louder and full length pansystolic on inspiration suggesting it was due to tricuspid regurgitation. It is probable that the early diastolic sound found in this patient (Fig 6) had a similar cause to that previously described in some patients with a left atrial myxoma; this has been attributed to the tumour hitting the cardiac wall (tumour plop) (Abbott *et al.* 1962).

The systolic wave in the right atrial pressure tracing (Fig 7) was probably due not only to tumour movement (spurious tricuspid regurgitation) but also to actual regurgitation since this was seen to be present at the operation following removal of the myxoma. It was also confirmed by the presence of a marked systolic wave in the post-operative right atrial pressure tracing (Fig 8). The tricuspid regurgitation was presumably due to the calcified tumour damaging the tricuspid ring and valve.

The notch in the upstroke in the pre-operative right ventricular and pulmonary artery pressure tracings (Fig 7) was similar to that which has been described in the left ventricular pressure of patients with a left atrial myxoma and attributed to movement of the tumour out of the ventricle (Penny *et al.* 1967). The absence of this notch following removal of the myxoma would tend to support this explanation.

The slight increase in pulmonary vascular resistance was similar to that seen in the case of calcified right atrial myxoma described by Oliver & Missen (1966). In their patient histological examination of the lungs showed the presence of multiple calcific emboli. Pre-operatively our patient appeared to be tolerating the tricuspid regurgitation satisfactorily, as shown by the cardiac output response to exercise. However, following the first operation there was marked deterioration in his condition with clinical evidence of a low cardiac output. This deterioration may have been related to the atriotomy, causing loss of atrial function, which is probably

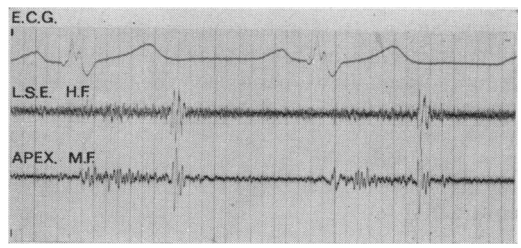

Fig 6 *Phonocardiogram. Recording at left sternal edge* (LSE) *obscured by respiratory noise. Recording at apex shows pansystolic murmur and early diastolic sound.* HF, *high frequency.* MF, *medium frequency*

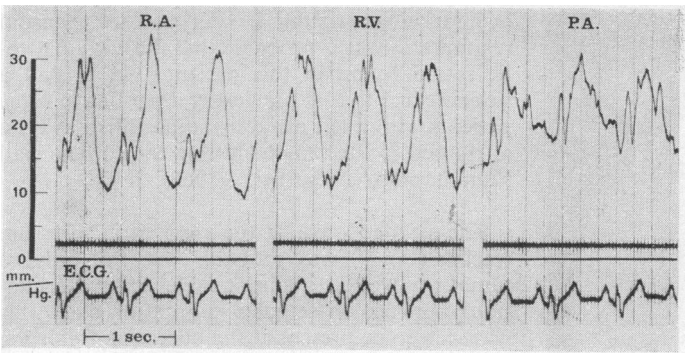

Fig 7 *Pressure tracings in right atrium* (RA), *right ventricle* (RV) *and pulmonary artery* (PA) *prior to operation. The RA pressure shows a prominent 'a' wave and large systolic wave. A notch is present on the upstroke of the RV and PA pressures*

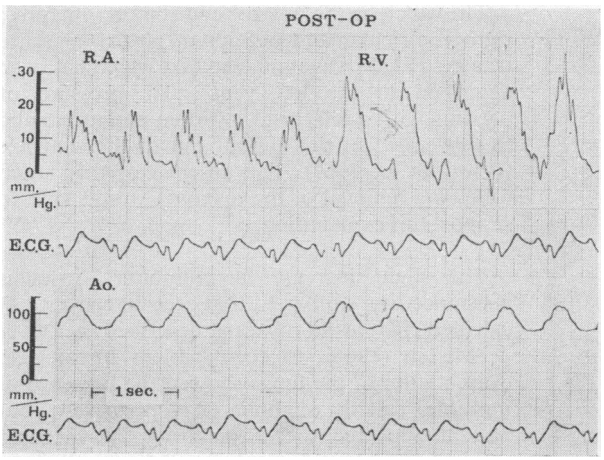

Fig 8 *Pressure tracing in right atrium* (RA), *right ventricle* (RV) *and aorta* (Ao) *recorded immediately after the first operation. The RA shows a small 'a' wave and a large systolic wave (although smaller than pre-operatively). The notch on the upstroke of the RV pressure present pre-operatively has disappeared. The aortic pressure is well maintained.*

necessary for the maintenance of a normal cardiac output in the presence of tricuspid regurgitation. It is also even possible that the tumour itself was minimizing the amount of regurgitation. Post-operatively the right atrial pressure tracing (Fig 8) showed only a small 'a' wave, as compared with a prominent 'a' wave pre-operatively (Fig 7), a finding which supports the view that there had been a reduction in atrial contraction.

This case, which closely resembles that described by Oliver & Missen (1966), illustrates that a calcified right atrial myxoma is usually associated with organic damage to the tricuspid valve. It also showed that following removal of the tumour the patient may be unable to tolerate the tricuspid regurgitation in the immediate post-operative phase. It suggests that after the tumour has been removed an attempt should always be made to render the tricuspid valve competent either by repair or by valve replacement.

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