Case II.—Miss C., aged 28, had rheumatic fever at the ages of 13, 18 and 20 years. Following the last attack she was told that her heart was affected. Now she has no symptoms. The pulse is moderately collapsing in quality and the blood pressure is 165/60. At the lower left sternal edge there is a loud high-pitched diastolic murmur separated from the second sound by a gap. The phonocardiogram (Fig. 4) confirms that the main diastolic murmur is late though some vibrations begin immediately after the second sound. The presence of aortic valve disease is supported by left ventricular preponderance in the electrocardiogram and slight left ventricular enlargement on cardioscopy. There is no clinical or radiological evidence of mitral valve disease.

The phonocardiogram (Fig. 5) of a man aged 43 with mitral stenosis is shown for comparison with the records of aortic incompetence and unusual diastolic murmurs. At the apex (MA) there is an obvious mid-diastolic murmur; here and at the lower left sternal edge (LSE) there is a loud sound (X) following the second sound, the so-called "opening snap" of the mitral valve. It precedes the mid-diastolic murmur by a short interval. Between the second sound and the "opening snap" are some vibrations which probably represent the early diastolic murmur of aortic incompetence which was just audible on auscultation.

In conclusion, it is well known that aortic diastolic murmurs begin immediately after the second sound but graphically this murmur may be shown to be maximal later. In rare cases the late crescendo may create the auscultatory impression that the murmur is separated from the second sound by a gap. This may cause difficulty in diagnosis though the murmur may be otherwise typical of aortic incompetence in its location and high pitch.

My thanks are due to Dr. William Evans for referring both cases to me.

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Constrictive Pericarditis and Mitral Stenosis.—FREDERIC JACKSON, M.R.C.P.

The patient, a male aged 52, had rheumatic fever three times in childhood. He had no known contact with tuberculosis. All his life he had been a little short of breath on exertion but had worked as a farmer until the age of 47.

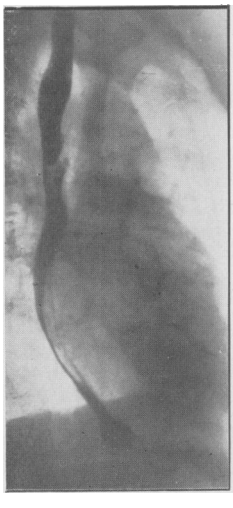

FIG. 1.

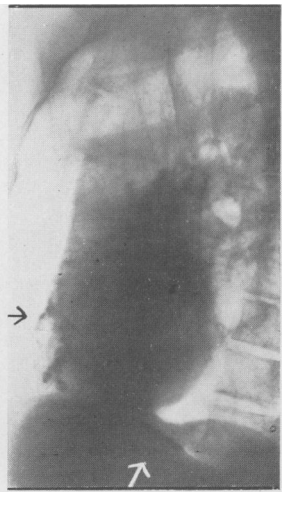

FIG. 2.

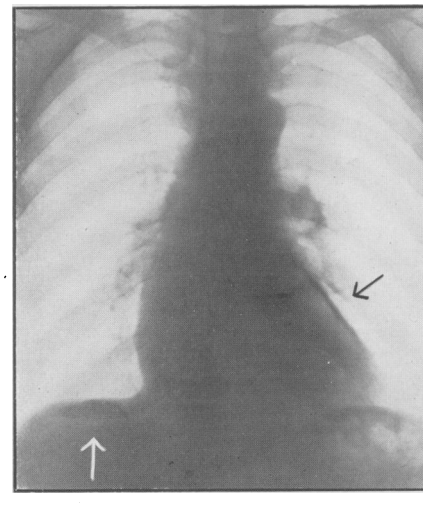

FIG. 3.

FIGS. 1 and 2.—Anterior and left oblique views. Slight enlargment of the heart. Calcium in the pericardium and in the dome of the right diaphragm.

FIG. 3.—Right oblique view. Left auricular impression on the barium-filled œsophagus.

In 1946 he was seen with symptoms of heart failure of two years' duration. His pulse (70–80 a minute) was irregular from auricular fibrillation; the venous pressure was raised (13 cm. of water); the liver was enlarged, but there was neither ascites nor œdema. A moderately loud systolic murmur was heard at the apex, but no diastolic murmur, and a third heart sound followed closely after the second sound.

On radiological examination the heart was slightly enlarged, calcium was seen in the pericardium (Figs. 1 and 2), and the left auricular impression on the barium-filled œsophagus was a little prominent (Fig. 3). In addition there was a plaque of calcium in the pleura over the right diaphragm (Figs. 1 and 2).

A diagnosis of mitral stenosis was considered but could not be proved. The venous distension and liver enlargement without other signs of failure in a man with moderately slow auricular fibrillation and clear triple rhythm suggested pericardial constriction. Calcium in the pericardium with minimal enlargement of the heart supported this view, and a diagnosis was made of constrictive pericarditis with probable rheumatic heart disease.

Pericardiectomy was performed by the late Mr. Tudor Edwards. The pericardium was thickened, densely fibrous and contained plaques of calcium; it was closely adherent to the heart, and it was stripped from both ventricles and the apex.

Since the operation the venous pressure has remained normal and the liver edge has receded, but symptomatic improvement has not been as favourable as expected. This may be due to additional mitral stenosis which can now be recognized by hearing a mid-diastolic murmur. The disappointing clinical improvement leads one to question the advisability of the operation in this case, despite the relief of the constriction and the lowering of the venous pressure.

Constrictive pericarditis is believed by many to be tuberculous in origin though evidence of the infection is frequently lacking. This case is likely to be an association of the effects of tuberculosis and rheumatic fever, two common diseases, existing together in the same patient. The difficulty remains, however, as to how far the symptoms must be ascribed to each.

Dr. A. Elkeles: Dr. Jackson's case is of special interest, since it shows that rheumatic fever can be an ætiological factor in the production of chronic adherent calcified pericarditis. I had the opportunity of investigating radiologically two cases of mitral stenosis with auricular fibrillation and signs of constrictive pericarditis. Oblique and lateral X-rays of the heart showed diffuse calcification of the pericardium. There was no evidence of tuberculosis. Mitral stenosis and tuberculosis exclude each other to some extent. In the past calcified opacities in the lungs sometimes met with in cases of mitral stenosis have been interpreted, probably wrongly, as evidence of pulmonary tuberculosis. In recent investigations Elkeles and Glynn (1946) have proved that some lesions can be due to intra-alveolar bone formations with no evidence of tuberculosis. Harkavy (1941) drew attention to the occurrence of constrictive pericarditis and even polyserositis in cases of severe vascular allergy with eosinophilia, in which the chief precipitating factor was bacterial allergy, resulting from chronic infection of the paranasal sinuses. In my opinion, the widely held view that tuberculosis is the most common ætiological factor in chronic adherent pericarditis has to be revised.

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Xanthomatosis Peritonei. ? Carcinoma of the Bronchus.—J. S. STAFFURTH, M.D., M.R.C.P. (for REGINALD HILTON, M.D.).

G. P., a retired police sergeant, aged 64.

1942: Skin lesions first noticed. W.R. ++.

1947: At an operation for right inguinal hernia free fluid was found in the peritoneal cavity. The peritoneum itself was thickened and white, and on section showed chronic inflammatory change with a large number of foam cells. Blood cholesterol 97 mg.%.

April 1949: A hard irreducible lump appeared at the site of the previous hernia. At operation this was found to be a yellow tumour at the apex of a hernial sac. Free fluid was present in the peritoneal cavity.

Since then he has lost weight, the ascites has become more troublesome and for three months he has complained of hoarseness of voice. There is only slight cough.

Family history is not relevant.

On examination.—The patient is thin. Xanthomatous deposits are present in both axillæ, elbows and in the inguinal folds; also along the margins of the eyelids and in the inner canthus of both eyes. There is diminished air entry of the left side of the chest with impaired percussion at the left base; considerable ascites; palpable liver; large spleen.

Investigations.—X-ray of chest shows a mass at the left hilum. Screening shows a paralysed left diaphragm. Blood cholesterol 70 mg.%. W.R. negative. Plasma proteins 4·4 grammes% (albumin 2·5, globulin 1·9 grammes%). Bronchoscopy: Immobile left cord; bronchial tree normal.

He has just completed a course of radiotherapy to the left hilar region, but as yet there is no improvement in symptoms and only slight change in the radiological appearance.

POSTSCRIPT (14.12.49).—Dr. G. B. Dowling's report reads: "There are large patches of superficial xanthomatosis in the axillæ, in the elbows, on the upper eyelids, in the post-auricular folds and in the inguino-crural areas." He stated that he had not seen lesions like this in xanthomatosis before.