DISCUSSION

Though the diagnosis was made firmly by the finding of exfoliated malignant cells and by operative biopsy, the deliberate introduction of air into the pericardium in the course of aspiration of effusion for the relief of tamponade gave the first clue to diagnosis in this case, as in that of Lübschitz *et al.* (1949). Though there are few observations on the relative pericardial thickness in different diseases, the thinness of the membrane weighed against a tuberculous aetiology. Angiography, a useful procedure in the investigation of suspected atrial myxoma (Griffiths, 1962), would not have been helpful in the absence of encroachment of the growth on either atrial cavity; electrocardiographic changes in malignant disease of the heart are variable and non-specific (Friedberg, 1956).

Though the sarcoma in this case arose in the left atrium, superior vena caval obstruction, a common finding in the more usual right-sided tumours, eventually occurred despite treatment.

In theory, early diagnosis of primary malignant disease of the heart raises the possibility of surgical removal, such as has been successful in cases of myxoma of the left atrium (Goodwin *et al.*, 1962). In most of the cases reported so far, however, the growth has been too far advanced to allow of anything other than palliative treatment, including fenestration to prevent recurrence of tamponade. There is little information on the results of any attempt at radical treatment, though radiotherapy and nitrogen mustards, radioactive phosphorus, and gold have been used in secondary neoplasms of the myocardium.

In one case of sarcoma local resection and nitrogen-mustard therapy result in temporary benefit (Abbott *et al.*, 1962). In our

case radiotherapy appeared to have no effect whatever on the inexorable course of the disease.

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Allergic Pericarditis

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We wish to record a case of idiopathic giant urticaria with pericarditis as a complication. Rogen and Richardson (1961) have recorded the association of chest pain and non-specific electrocardiographic changes with urticaria, and we believe that this may constitute a definite form of "benign pericarditis."

CASE REPORT

A 58-year-old foundry worker had suffered from infrequent attacks of bronchial asthma for the past 10 years. He was seen by one of us (P.F.) on several occasions, and treatment with anti-spasmodics was always effective. A chest x-ray film in 1957 and an electrocardiogram in 1959 were normal.

On 26 February 1961 he was gardening, in contact with numerous weeds. His diet had been his usual one, and he had taken no medication for some months. The next day he developed giant urticaria of the whole body. Three days later he sought medical advice.

He had extensive urticaria and mild asthma. Temperature was 101° F. (38.3° C.). Investigation that day showed a haemoglobin of 15.2 g./100 ml., W.B.C. 26,700/c.mm. (90% neutrophils, 7% lymphocytes, 3% monocytes). A Casoni test was negative. Treatment with antihistamines (chlorpheniramine ("chlor-trimeton") 4 mg. b.d.) was started, and he was admitted to Napier Hospital for observation. White cells were 13,000/c.mm. (80% neutrophils, 13% lymphocytes, 5% monocytes, 1% eosinophils). Skin tests to common antigens were negative. His temperature subsided.

On the fourth day after admission he developed increasingly severe central chest pain, not affected by posture or respiration. A loud to-and-fro pericardial friction rub was present, and, apart from

urticaria, nothing significant was noted. Pulsus paradoxus was not evident then, nor at any subsequent stage. An electrocardiogram (Fig. 1) was regarded as consistent with pericarditis or early myocardial infarction. A chest x-ray film (Fig. 2) taken next morning showed significant cardiac enlargement as compared with the film taken four years earlier, and was in keeping with a diagnosis of pericardial effusion. Serum glutamic pyruvic transaminase showed a transient rise to 76 Sigma-Frankel units; the serum glutamic oxaloacetic transaminase was 54 Sigma-Frankel units on the third day of this illness, and subsequently returned to normal. The urine was microscopically normal: there was no albumin and no growth on culture. Serial W.B.C.s varied between 9,000 and 13,000/c.mm., with a normal differential count. The serum electro-

FIG. 1.—Electrocardiogram taken on 7 March 1961.

phoretic pattern showed a slight rise in α_2 -globulin. Several examinations for L.E. cells were negative. A muscle biopsy two weeks after the onset of illness showed no abnormality. Viral investigations on three specimens of faeces were negative on both culture and animal inoculation.

The patient experienced continuous chest pain for 48 hours, and a loud pericardial friction rub persisted for two weeks, then gradually disappeared over the subsequent week. Thereafter his only inconvenience was urticaria and occasional mild asthma. Subsequent electrocardiograms over the next month showed return of the S-T segment, which originally had shown saddle-shaped elevation in leads II, avF, V5, and V6, to isoelectric levels, with flattening of the T waves in these leads. After a month the T waves became elevated again. An electrocardiogram taken in October 1962 was normal.

FIG. 2.—Radiograph taken on 8 March 1961.

FIG. 3.—Radiograph taken on 25 October 1962.

Chest x-ray films showed persisting enlargement of the cardiac shadow for one month, and this later returned to normal (Fig. 3).

He received two doses of phenindione at the beginning of his pericarditis, and a few injections of pethidine. The same antihistamine was administered periodically for the next three months, and he had an occasional compound preparation of ephedrine, aminophylline, and amylobarbitone for asthma.

He was discharged from hospital on 30 March 1961 and resumed work one month later.

Urticaria persisted moderately until October 1961, and he has since then enjoyed excellent health. He was last seen in March 1963.

DISCUSSION

Chest pain, electrocardiographic changes, a pericardial rub, and fever in a middle-aged man are often sufficient to make one highly suspicious of acute ischaemic heart disease. If such a diagnosis is accepted it will usually lead to "medical treatment": bed rest, restriction of activities, and possibly anticoagulants. If the diagnosis is in any doubt, it can lead to a feeling of insecurity both by the patient and by the physician.

To find an alternative distinct "benign" condition in such cases constitutes considerable relief. Unfortunately, pericarditis,

if accepted as a disease, often defies further diagnostic elucidation, and our experience is similar to that of Nemser and Donoso (1962)—namely, that about half the cases so diagnosed remain "idiopathic."

Wolff and Grunfeld (1963), in a further discussion of pericarditis, state in their introduction that "the pericardium is one of the structures in which allergic phenomena occur and produce diagnostic and therapeutic problems of considerable magnitude." No mention is made, however, of the phenomenon as encountered in this case.

Rogen and Richardson (1961) had experience of three cases, all showing features similar to our case, but not exhibiting a pericardial friction rub or cardiac enlargement, and they

speculated on whether the pathological process lay in the myocardium or the coronary vessels.

Our purpose in drawing attention to this recent experience and to quote the experience of others is to try to ascertain whether any definite connexion does in fact exist in cases of idiopathic urticaria which develop chest pain and signs and symptoms of pericarditis, or even abnormal electrocardiographic changes only. We believe that our case would support such an association.

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