In 1972 at this centre the incidence of postoperative fatal pulmonary embolism in patients over the age of 50 was 1.35% (calculated from 1 800 necropsy reports). The incidence of fatal pulmonary embolism in this series was higher (2%), but this was because the patients constituted a high-risk group and included both the heparinized cases and the controls.

The dosage regimen had no apparent effect on clotting times. There was a variation of 5 to 20 seconds in the thrombin clotting time, which is within normal limits. Thromboelastographic recordings were well within the normal range. The peak plasma levels of heparin after the subcutaneous injection of 5 000 units were less than 0.013 IU/ml. (With conventional therapy the peak plasma level of heparin is 1.3 IU/ml; Kakkar *et al.*, 1971). Thus the circulating heparin levels were subclinical—that is, there was no evidence of increased operative or postoperative bleeding or wound haematoma formation—and thus safe.

When asked to comment on the degree of bleeding at operation the surgeons were unable to distinguish between the two groups. There was no increase in postoperative bleeding or wound haematoma formation. Two patients were withdrawn at the request of the surgeon because of a suspected increase in postoperative bleeding but coagulation studies showed nothing abnormal. Drainage measurements after abdominoperineal resections and thoracotomies did not show any significant difference between the two groups.

The mucous heparin sodium used in the trial is manufactured in the U.K. and is readily available. In previous studies calcium and sodium salts of heparin (lung) have been used (Sharnoff and De Blasio, 1970; Kakkar *et al.*, 1971; Kakkar *et al.*, 1972; Nicolaidis *et al.*, 1972) and the advantages claimed were fewer local haematomas, and possibly longer-sustained levels in plasma. Haematoma formation at the site of injection was not a problem in this series and the results confirm that the sodium salt of mucous heparin works as effectively as calcium heparin and is much cheaper. The potential errors in a study of this kind arise mainly from the use of unstandardized methods—namely, the matching of patients

and variable factors such as surgical technique—and the use of other thromboembolic precautions. This is, however, a controlled random trial and statistically these factors will cancel one another out as the number of patients increases.

The clinical diagnosis of pulmonary embolism is often difficult and unreliable. Aids such as scanning, angiography, etc., are not without hazards or foolproof (Oakley 1970; Poulouse *et al.*, 1970). Hence evaluation of the efficacy of heparin prophylaxis is made at necropsy.

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MEDICAL MEMORANDA

Myocarditis Complicating Q Fever

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Cardiac involvement in Q fever is well recognized, the lesion being usually endocarditis (Evans *et al.*, 1959; Robson and Shimmin, 1959; Marmion *et al.*, 1960; Smith and Evans, 1960). Myocarditis complicating Q fever though mentioned in the European literature (Giroud and Goulon, 1953; Busila *et al.*, 1971), does not appear to be recorded in the English literature and is not listed in recent editions of standard textbooks. We describe here two such cases in which myocarditis was a prominent feature.

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Case 1

In April 1972 a 42-year-old motor mechanic was admitted to the Royal Devon and Exeter Hospital as an emergency case with chest pain and a provisional diagnosis of myocardial infarction. He lived and worked in the city. He drank pasteurized milk and rarely visited the countryside. His illness had begun four days previously with shivering and muscular aches. There were no respiratory symptoms and he had no headache. On the day of admission he experienced sudden, tight pain across the front of his chest radiating to the jaw and left scapular region. He sweated profusely. The pain was not related to posture or inspiration and lasted two hours. Temperature 39.5°C; pulse 90/min, regular; blood pressure 120/70 mm Hg. Examination of the heart and chest showed nothing abnormal.

In view of the prodromal symptoms steps were taken to exclude viral myocarditis. Investigations were: haemoglobin 13.6 g/100 ml; W.B.C. 8,600/mm³ (81% polymorphs); E.S.R. 91 mm in the first hour (Westergren); serum aspartate transferase 250 mU/ml (normal 10-50 mU/ml); and lactic acid dehydrogenase 430 mU/ml (normal 30-85 mU/ml). Chest x-ray changes were compatible with pneumonitis at the left base. Serial E.C.G. showed progressive changes compatible with acute myocardial damage inferolaterally (fig. 1). No virus was isolated from throat swab or faeces but *Coxiella burnetii* phase II complement fixation titres rose from 1/10 on the fourth day of the illness to 1/160 on the eighth day. Phase I titres were negative throughout.

The fever and symptoms settled after five days without treatment. The haematological and biochemical findings were normal by the sixth week. A year later he was in good health, having had no recurrence, and an E.C.G. was normal.

FIG. 1—Case 1. E.C.G. on fifth day of illness showing changes in leads II, III, and aVF.

Case 2

In April 1968 a 28-year-old accountant was admitted to the North Devon Infirmary with a two-week history of mild fever, shivering, and malaise. For 10 days he had had cough, slight haemoptysis, and vague central chest pains. Two days before admission the dyspnoea increased and he had a nosebleed. Examination showed an ill young man with a fever of 38.1°C, dyspnoea, cyanosis, and a pulse of 120/min. An atrial fourth heart sound was heard and there were widespread crepitations over both lungs. Blood pressure 130/70 mm Hg. Investigations were: haemoglobin 15.5 g/100 ml; W.B.C. 10,700/mm³ (84% polymorphs); E.S.R. 30 mm in the first hour (Westergren); serum aspartate transferase 33 IU (normal 0-16 IU); A.S.O. titre less than 200 U; and Widal and Brucella agglutination tests negative. Chest x-ray film showed patchy opacities over both lungs. E.C.G. showed sinus tachycardia and S-T change, indicating acute myocardial injury anteriorly (fig. 2). *C. burneti* titre on the 28th day of the illness was 1/2,560 in phase II.

FIG. 2—Case 2. E.C.G. on 15th day of illness showing Q waves in V2 with S-T elevation and early T-wave inversion in V2 and V3.

Treatment with tetracycline, digoxin, diuretics, and oxygen resulted in improvement and subsequent full recovery. The E.C.G. reverted to normal within six weeks. The antibody titre fell to 1/160 after 14 months; five and a half years after the onset of the illness it was 1/40, which is unusual. Phase I antibody was not detected at any time.

Comment

The rising or very high titres of antibody to *C. burneti* confirmed the diagnosis of acute Q fever in these patients. The E.C.G. changes showed unequivocal evidence of myocardial injury. It is interesting that these changes were in the distribution of the right coronary artery in case 1 and in the distribution of the left coronary artery in case 2. Fatal cases of louse-borne typhus and tick-borne typhus, also rickettsial illnesses, have shown necrosis of the vessels of the myocardium (Sachs, 1946) and it is tempting to speculate that such lesions might have occurred in these two cases.

Endocarditis complicating Q fever is a grave condition with a high mortality and merits prolonged chemotherapy and often valve replacement (Kristinsson and Bentall, 1967). Antibodies to both phases of *C. burneti* are found in the serum, usually in high titre. Neither of our cases showed evidence of endocarditis or produced phase I antibodies. Myocarditis appears to be much less common and in case 1 was a relatively mild illness which resolved without therapy. The other patient was more severely affected, as reflected in his clinical state, E.C.G.s, and *C. burneti* titre, and required general treatment and specific antibiotic therapy.

In case 1 there was no obvious reason for the illness. On the industrial estate where the patient worked there was a weekly cattle market and an abattoir, and though he never visited either place airborne infection may well be implicated. Marmion and Stoker (1958) recorded 10 cases of Q fever in a small town in Kent. The source of these infections was obscure but may well have been by airborne dissemination of *C. burneti* from a fertilizer factory where infected offal was processed. In case 2 the likely source was thought to be sheep at the bottom of the patient's garden which had recently lambed, but serological examination of these for antibodies to *C. burneti* gave negative results; however, he drank unpasteurized milk from the farm. Many herds of cattle in Devon are infected with *C. burneti* (Hart, 1973).

Though the prodromal illness in case 2 was typical of Q fever that in case 1 was mild and the cardiac pain was the prominent feature. By establishment of the diagnosis of Q fever the label of ischaemic heart disease, with its emotional, social, and prognostic implications, was avoided. We do not believe that the incidence of Q fever myocarditis is high but suggest that the diagnosis should be considered in such patients.

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