1951-8 is reported. The results observed in this group are compared with a control series previously reported from the same hospital.

The expectation of life of the treated patients was increased by a factor of six to eight times that expected from the control series. Prolonged survival was particularly seen in those patients whose renal function was normal or only slightly impaired at the start of treatment. Death was most frequently due to uraemia; heart failure as a cause of death was considerably diminished in the treated group.

The benefits of treatment were most pronounced on retinitis and in heart failure. Improvement in the electrocardiogram was seen in one-third of cases; reduction in cardiac size occurred somewhat less frequently.

Renal function tended to remain stationary or to improve slightly in patients with an initial blood urea level below 80 mg. per 100 ml.; where renal impairment was more severe than this progressive deterioration was the rule.

Patients with neurological complications did not derive much benefit from treatment. Accompanying atherosclerosis was a limiting factor in the success of treatment, particularly in older patients.

A survey of the histological lesions in the kidneys showed that after treatment with hypotensive drugs the main changes were a conversion of cellular intimal hyperplasia in the interlobular arteries to fibrous intimal thickening and a "healing" of fibrinoid degeneration to hyaline and fibrous tissue. Some correlation was likely between the degree of narrowing of the interlobular arteries at the start of treatment and prognosis.

The clinical observations have been documented through the years, especially by Drs. Brenda Morrison, Helen Mellanby, and Edmond Murphy. We are indebted to our clinical and pathologist colleagues who co-operated in the study. We are deeply grateful to Messrs. May and Baker Ltd., who contributed generously to the support of this research irrespective of the source of drugs under trial.

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CHRONIC Q FEVER

1. CLINICAL ASPECTS OF A PATIENT WITH ENDOCARDITIS

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The tendency of rickettsiae to persist in the tissues of human or animal hosts after an initial infection is well recognized. Perhaps the most striking example is that of Brill's disease, in which the rickettsia of epidemic typhus has been isolated during a recrudescence of illness occurring in persons first exposed to infection up to twenty or more years previously (Murray and Snyder, 1951). Other rickettsiae—namely, those of scrub typhus (Smadel *et al.*, 1952) and of Rocky Mountain spotted fever (Parker *et al.*, 1954)—have been recovered from lymphoid tissue taken from patients one to two years after the original illness.

Although the lengthy persistence of *Rickettsia burneti* (the causative organism of Q fever) in the tissues of experimentally infected animals has been demonstrated on a number of occasions, relatively little is known about its persistence—either as a latent infection or as the cause of a chronic illness—in human beings.

This paper describes the clinical aspects of a patient with chronic Q fever who had the symptoms and signs of endocarditis; it also briefly reviews the literature of the problem in general. A second paper by our colleagues, P. S. Andrews and B. P. Marmion (1959), gives details of the pathological and bacteriological observations which were made on the patient.

Case History

The Acute Illness

The patient, a geophysicist aged 47, first became ill in December, 1955, while his yacht was being refitted in Panama after damage at sea. The illness began suddenly with fever, rigors, severe headache, low back pain, and a dry cough. On examination in hospital he had a temperature of 102° F. (38.9° C.) and a scanty maculo-papular rash on the trunk. No other abnormal signs were described. A chest radiograph showed an area of consolidation in the right lower zone and a normal cardiac silhouette (cardio-thoracic ratio, 47%). The urine contained a few pus cells. All other investigations, including at least one blood culture and search for malarial parasites, were negative. He was given a course of sulphafurazole ("gantrisin"), and 12 days later (December 15) was afebrile and symptom-free. He returned to England, where in March, 1956, a chest radiograph showed nothing abnormal.

Retrospectively, the description of this illness is suggestive of Q fever, especially as Panama is a known endemic area and the patient had removed ticks from his dog and had also drunk raw milk. Unfortunately, however, no serological investigations for Q fever were undertaken at the time.

The Chronic Illness

In May, 1956, he fell ill again, with fever reaching 103° F. (39.4° C.), lassitude, and a severe occipital headache. He was treated with consecutive five-day courses of a sulphonamide and chlortetracycline without response. He was admitted to the Middlesex Hospital on June 15, with general malaise, fever, joint pains, and myalgia. His history revealed that, as a potential conscript, he had failed a Polish Army medical examination in 1929 because of a cardiac murmur, but he denied having had rheumatic fever. Examination showed a temperature of 100° F. (37.8° C.), a grade 2 systolic murmur, and a soft immediate aortic diastolic murmur. There was early finger and toe clubbing. There was no evidence of emboli and the liver and spleen were not palpable. A provisional diagnosis of subacute bacterial endocarditis was made.

A series of clinical and laboratory investigations were carried out. These showed a total white-cell count of 5,900 cells per c.mm. of blood, with a normal differential distribution, haemoglobin 13.4 g./100 ml. and an E.S.R. (Westergren) of 40 mm. in the first hour. Twelve blood cultures were sterile. L.E. cells were not seen on one examination. Centrifuged deposits of urine repeatedly failed to show leucocytes or red blood cells. The Wassermann reaction was negative. The antistreptolysin "O" level was 200 units/ml. of serum. Liver-function tests and plasma proteins were within normal limits, but paper electrophoresis of the serum revealed increased α_2 - and γ -globulin components. A chest radiograph showed normal lung fields and a cardio-thoracic ratio of 51%. An intravenous pyelogram showed healthy kidneys. Radiographs of the forearm revealed a subperiosteal reaction. A Widal reaction did not show serum agglutinins for the enteric fever group of organisms or for *Brucella abortus*, but tests carried out at the Virus Reference Laboratory, Colindale, did show a titre of complement-fixing antibody to Q fever antigen of at least 1/128 (the highest dilution tested). The significance of this finding in relation to the illness was discussed with Dr. B. P. Marmion, of the Public Health Laboratory, Leeds, and arrangements were made with him for further investigations into the possibility of chronic Q fever infection. The results of these investigations are described in detail in the paper by Andrews and Marmion (1959), but, briefly, a high titre (1/2,000) of Q fever complement-fixing antibody was demonstrated in the blood and *R. burneti* was isolated from the blood stream on two occasions during the chronic illness and from various organs and the aortic valve vegetations after death.

Subsequent Course of the Illness and Treatment

Initially the patient was given tetracycline for two weeks (45 g.), with depression of fever to 99° F. (37.2° C.) and improvement in the arthralgia, but the drug had to be discontinued because of severe diarrhoea. Subsequently, during a three-weeks course of penicillin (100 mega units) and one week on streptomycin (16 g.), the symptoms and fever recurred. Sodium salicylate (80–160 gr.—5.2–10.4 g.—daily) caused no improvement. Phenylbutazone (200–400 mg. daily) brought symptomatic relief of joint pains and depression of the temperature, but was stopped after two months when the haemoglobin fell to 7.5 g. (52% Haldane). The anaemia responded to transfusion and iron.

In December a five-weeks course of chlortetracycline (1–1.75 g. daily) produced only slight remission, and the administration of oleandomycin (1 g. daily) was followed by a relapse of fever to 101° F. (38.3° C.).

From January to June, 1957, the patient had chlortetracycline (1–5 g. daily) and remained fairly well. Prednisone suppressed the fever but was stopped because of abdominal pain. Colchicine controlled the arthralgia of small joints for a time. In November a course of oxytetracycline (28 g.) had no effect.

During the entire period from May, 1956, to December, 1957, the temperature fluctuated from 99° F. (37.2° C.) to 103° F. (39.4° C.)—a typical sequence is shown in Fig. 1.

Arthralgia and swelling of the small joints persisted and the clubbing of fingers and toes progressed. The heart enlarged progressively, a cardio-thoracic ratio of 60% was found in an x-ray film taken on December 19, 1957, and the cardiac murmurs became more prominent. The weight, however, remained steady.

In December, 1957, the patient developed a harsh systolic murmur and left ventricular failure, and, despite symptomatic

FIG. 1.—Typical temperature chart in early part of chronic illness, showing depression of fever with tetracycline and no response to penicillin. Drugs were given six-hourly in doses indicated.

FIG. 2.—Chart summarizing course of illness and significant findings.

treatment, died with acute pulmonary oedema on January 2, 1958. Two days previously there had been a marked fatalistic personality change.

The post-mortem examination, which is described in detail by Andrews and Marmion (1959), revealed a deformed and fibrosed aortic valve with perforations of the cusps and a cerebral haemorrhage in the left frontal area.

The course of the entire illness, from the first acute episode in Panama to the final and fatal outcome, is shown diagrammatically in Fig. 2. Important investigations during the period of chronic illness can be summarized as follows. Twenty-four blood cultures were bacteriologically sterile. The urinary deposit never showed pus cells or erythrocytes, although albumin was present in the terminal phase of the illness. Haemoglobin ranged from 13.1 g. (88%) to 10.4 g. (70%)—apart from the anaemia during phenylbutazone therapy. The leucocyte count remained normal and the E.S.R. was raised, varying between 30 and 50 mm./hour (Westergren). Serial chest radiographs showed progressive increase in cardiac diameter and the lung fields remained clear. In May, 1956, the E.C.G. was normal, and in December, 1956, showed changes indicating left ventricular hypertrophy. Plasma proteins were normal. The α_2 - and γ -globulins were consistently raised. Liver-function tests on January 23, 1957 showed: thymol turbidity, 16 units; colloidal gold, 5 units; zinc sulphate, 21 units. Blood uric acid was 0.7 mg./100 ml., and alkaline phosphatase 9.5 King-Armstrong units. These results were thought to be due to the non-specific effects of a chronic infection rather than indicative of hepatic damage.

Comment

At first sight this appeared to be a case of subacute bacterial endocarditis, but, if so, the illness had many atypical features. Except during phenylbutazone therapy, there was no significant anaemia and the leucocyte count was not raised. There were no embolic phenomena, the urinary deposit never showed red blood

cells or pus cells, and repeated blood cultures were sterile. Arthralgia and arthritis are found only as fleeting symptoms in bacterial endocarditis, but in this patient they were constant and progressive features. Joint pains have occasionally been described in Q fever (Huebner *et al.*, 1949). A diagnosis of rheumatic fever was considered, but the course of the illness and the pathological findings did not support this.

In retrospect, the initial illness in Panama seemed typical of Q fever. Serological evidence of infection with *R. burneti* was found at the beginning of the chronic illness, and this organism was isolated from the blood during the chronic illness and from the tissues after death. The diagnosis therefore seemed to be one of chronic Q fever infection with endocarditis.

Discussion

Q fever has a world-wide distribution, although it is of interest that it has not so far been shown to be endemic in Ireland, New Zealand, the Netherlands, or Scandinavia (Kaplan and Bertagna, 1955).

Typically, Q fever is an acute febrile illness of sudden onset with severe headache, rigors, and, in a variable proportion of patients, evidence of pneumonitis. Generally the fever does not last more than two to three weeks. The lung lesions have attracted much attention, but it should be emphasized that Q fever is a generalized systemic infection. Less common manifestations, or complications of the disease, include hepatitis (first described by Derrick in 1937), thrombophlebitis and pulmonary infarction, pleural effusion, oesophagitis, pericarditis, pancreatitis, epididymo-orchitis and meningo-encephalitis (Denlinger, 1949; Huebner *et al.*, 1949; Moeschlin and Koszewski, 1950; Clark *et al.*, 1951; Marmion *et al.*, 1953; Germer and Schaubert, 1953).

Fortunately these manifestations are rare and Q fever is usually a benign illness—for example, Robbins and Ragan (1946) found no complications in more than 350 cases of Q fever in men of military age. Even so, an increasing number of cases are being recorded in which the illness has run a protracted course. Thus the Commission on Acute Respiratory Diseases (1946) found four patients who relapsed on the 18th, 26th, 55th, and 66th days of the illness respectively. Deparis *et al.* (1956) reported a probable case of Q fever in which the fever persisted for more than 30 days, and of the 180 cases described by Clark *et al.* (1951) 30 had a fever lasting more than 28 days. The liver was more often enlarged or tender in patients with protracted illness; and Ludwig (1956) and Wespi (1957) also describe cases of Q fever hepatitis which ran a protracted course. Bertrand and Roux (1955) reported the isolation of *R. burneti* from a patient who suffered a relapse a month or so after an attack of Q fever. Beck *et al.* (1949), in a survey of 300 cases in southern California, mention one patient who had a recurrent fever for about five years and several other patients with a chronic, febrile illness after a proved attack of Q fever.

Somewhat different evidence of prolonged but latent infection with *R. burneti* is provided by the observations of Babudieri (1953) and Syruček *et al.* (1958), who isolated the rickettsia from placentas collected at the birth of children taking place at varying intervals from six months to three years after an attack of Q fever in the mothers.

Marmion *et al.* (1953) emphasized the concept of a chronic form of Q fever and suggested that this might

occur when the infection was superimposed on a pre-existing cardiac deformity or a disease of another system. In support of this, one of Denlinger's (1949) patients with pneumoconiosis had a prolonged illness, but, on the other hand, another patient with rheumatic heart disease was unaffected. Marmion *et al.* (1953) discuss some of their atypical cases, one of which was a man with a 10-years history of bouts of fever, cough, and dyspnoea. This patient had valvular heart disease and developed the clinical picture of subacute bacterial endocarditis, but repeated blood cultures were bacteriologically sterile. The serum titre of Q fever complement-fixing antibody was 1/1,024. Eventually the patient died, and necropsy revealed mitral stenosis and pulmonary infarction. *R. burneti* was not isolated during the illness or from organs taken at necropsy. Marmion *et al.* (1953) also give some information about two patients with a chronic illness who had been investigated by Dr. R. J. Huebner, National Institutes of Health, Bethesda, U.S.A. (Dr. Huebner had kindly allowed the reproduction of his unpublished data.) The first of the two cases was that mentioned briefly by Beck *et al.* (1949). This man had five years' illness with recurrent fever. About three months before he died from a stroke he had been shown to have a raised titre of Q fever complement-fixing antibodies and *R. burneti* had been isolated from his blood. At necropsy there was a massive cerebral haemorrhage, bronchopneumonia, and a fibrosed mitral valve with vegetations. *R. burneti* was isolated from various tissues, but the brain and mitral vegetations were not available for culture. The second patient was a woman with a history of rheumatic fever who had an illness resembling subacute bacterial endocarditis after an attack of Q fever. Numerous blood cultures were negative for bacteria, but *R. burneti* was isolated from the blood and urine more than a year after the original attack of Q fever.

Baylon *et al.* (1952) describe the case of a patient who was suffering from uncompensated mitral stenosis and an arterial embolus in the right leg. He had finger-clubbing, but no fever or splenomegaly. His heart failure was treated successfully, but he returned 18 months later with signs suggestive of either an embolism in the left radial artery or an acute arteritis. The serum titre of Q fever agglutinins, measured by the Giroud micro-agglutination method, was 1/1,280. Fever then developed and the clubbing increased. Blood cultures were bacteriologically sterile and the urinary deposit did not contain pus cells or erythrocytes. The Q fever agglutination titre had now reached 1/3,220. The patient was treated with successive courses of broad-spectrum antibiotics with improvement and the Q fever agglutinin titre decreased to 1/20.

The evidence for a chronic form of Q fever, sometimes with endocarditis, is therefore suggestive, but it appears that before the present investigation *R. burneti* had not been isolated from heart valve vegetations.

It is of interest that a number of these patients suffered either from pulmonary infarction or cerebral haemorrhage, or, possibly, from acute arteritis, and also that two other patients of Marmion *et al.* (1953) died either from pulmonary infarction or, probably, from a vascular catastrophe. Huebner *et al.* (1949) mention a patient who had severe thrombophlebitis, recurrent fever, and leg swelling which lasted for two years after an attack of Q fever, and two other elderly patients with leg pains resembling those of intermittent claudication which occurred as sequelae to Q fever.

Q fever is generally regarded as being different from other rickettsial infections in that the primary lesion is not in the endothelial cells of small vessels and in other parts of the cardiovascular system (Wolbach, 1948), but it is possible that vasculitis is a feature of the chronic rather than of the acute disease. It is of interest in this general context that a number of French workers (see Donzelot *et al.* (1950) and Giroud and Pfister (1957) for description of cases and references) have described cases with the symptoms and signs of subacute endocarditis following typhus or other rickettsial infections, but the exact part played by the rickettsiae in producing the cardiac damage is hard to assess.

With our present patient we have no means of knowing for certain whether the aortic valvulitis was due entirely to *R. burneti* or whether there had been previous damage due to rheumatic fever which had allowed the valve to be colonized by *R. burneti* in the manner suggested by Marmion *et al.* (1953). In spite of the detection of a cardiac murmur in 1929, the patient led an extremely active life; nor do we know the timing of this murmur. No cardiac abnormality was noted at the time of his illness in Panama. When he first attended the Middlesex Hospital the diastolic murmur was only just audible, and it might well be argued that the lesion had originated at the beginning of the chronic illness.

The importance of the case reported here lies in the proof that *R. burneti* can cause a chronic illness with endocarditis. Such cases are probably rare, although the clinical picture differs so much from the classical case of Q fever that the diagnosis might easily be missed. The possibility of Q fever should be considered in patients with subacute endocarditis from whom no bacteria have been isolated. So far the prognosis for such established cases of chronic Q fever appears to be grave.

Summary

A case is described of a prolonged and fatal illness which at first resembled subacute bacterial endocarditis with some atypical features. On further investigation the illness was found to be due to a chronic infection with *Rickettsia burneti*, the causative agent of Q fever.

The patient, a middle-aged man, had an acute pneumonia resembling Q fever; he recovered and was apparently well for six months. He then became ill with a swinging fever and signs of aortic valve incompetence which lasted until his death from cerebral haemorrhage and acute pulmonary oedema some 18 months later.

Serological evidence of infection with *R. burneti* was found at the beginning of the chronic illness, and the rickettsia was isolated from the blood stream during life and from various organs, including the aortic valves, after death.

The literature concerning similar cases is briefly reviewed, and we have concluded that Q fever may pursue a chronic course and that on occasion the site of infection may be in the heart valves.

[Note.—Since these two papers were submitted for publication Evans, Powell, and Burrell (*Lancet*, 1959, 1, 864) report the isolation of *R. burneti* from the aortic valve of a patient with endocarditis who died five months after an illness resembling Q fever.]

We thank Dr. F. H. Scadding, under whose care the patient was admitted, for allowing us to publish this case, and for his advice and criticism. We are grateful to Dr. B. P. Marmion for his advice about the problem and for his help in the preparation of this paper.

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CHRONIC Q FEVER

2. MORBID ANATOMICAL AND BACTERIOLOGICAL FINDINGS IN A PATIENT WITH ENDOCARDITIS

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[WITH SPECIAL PLATE]

The purpose of this paper is to give an account of the pathological and bacteriological investigations which were made on the patient with endocarditis described from the clinical viewpoint by Robson and Shimmin (1959). The findings at necropsy and on histological examination are given first of all, then the serological results and animal inoculation experiments—carried out with specimens taken either during the chronic illness or after death—which established that the patient was infected with *Rickettsia burneti*.

Pathological Investigations

Post-mortem Examination (Middlesex Hospital)

Because of the risk attached to the post-mortem examination of cases of Q fever (see Marmion and Stoker, 1950) full precautions were taken. Only the pathologist (P. S. A.) and one assistant were present in the post-mortem room; both wore caps, masks, gowns, aprons, arm-length rubber gloves, and rubber boots. A large supply of sterile universal containers for collecting specimens for virological and bacteriological studies was