diverticulae was thought unlikely to cause folate deficiency but may have contributed to the B12 deficiency. The coeliac disease (case 2) had been present for several years, was stable, and had not previously caused macrocytosis. The poor diet (case 3) was long standing and had not previously caused macrocytosis. A mild haemolysis does occur with sulphasalazine treatment (case 4).

We would emphasise that treatment with sulphasalazine has an experimentally proved mechanism for inducing folate deficiency, which is dose dependent. The paper by Grindulis and McConkey cited by Dr Greaves and coworkers concluded that "caution is therefore necessary if more than 2 grams of sulphasalazine daily is given in rheumatoid arthritis; in such circumstances clinical or haematological evidence of deficiency is more likely to develop because the mechanism by which the drug antagonises folate metabolism is dose dependent."¹ All but one of our patients received a higher dose than this.

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Social future of elderly admitted to acute care hospital

SIR,—In the study by Dr Françoise Roudot-Thoraval and colleagues (7 March, p 608) 32 of the 39 patients discharged to long term care had a negative view of it at the start of their acute admission, and no patient who was free of family pressure wanted to be transferred to long term care. The factor that predicted whether a patient would go into long term care was the view of the family, not that of the patient.

Secondly, though it is difficult to find a comparable British series, a transfer rate from acute to long term care of 31% seems very high. Might this high transfer rate itself be responsible for the strong correlation between being transferred to a long term bed and staying longer in the acute one? Perhaps the local long stay sector is unable to cope with this transfer rate and the system is dealing with the overload by allowing a backup of patients in the acute sector.

I have not seen the cited papers by Kane *et al* but presume that they report American experience. As to the importance of the age and mental state of the patient, several British studies have shown them to be valuable in predicting the outcome of hospital admissions.¹⁻³

In this unit we emphasise the need for very early involvement of the family in an old person's care. In the difficult balance between taking risks and preserving independence, however, we believe that British families tend to opt for safety at the expense of freedom. We would therefore advise against any movement to give families the predominant voice in determining placement. It would also be unwise to ignore the financial consequences of non-essential use of long term facilities.

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What contribution has cardiac surgery made to the decline in mortality from coronary heart disease?

SIR,—As Drs John M Neutze and Harvey D White point out (14 February, p 405), a major problem in assessing the contribution of cardiac surgery to the decline in coronary heart disease mortality is in estimating the potential mortality of the surgical patients had they not had surgery. The earlier, and much lower, estimates are a result of different assessments of the likely benefit of surgery. Drs Goldman and Cook¹ and I² prefer an estimate based on a randomised clinical trial, with all its limitations. Drs Neutze and White prefer estimates based on a variety of case series from North America (which I used in estimating an upper limit of benefit). It seems, however, that because of the lack of appropriate data from Auckland on the survival of a comparable group of medically treated patients Drs Neutze and White are guilty of the same "conceptual fallacy" as previous investigators.

It might be useful to take a broader look at the potential impact of cardiac surgery. In New Zealand coronary heart disease mortality has declined in all age, sex, and race groups. In general, however, coronary surgery has been restricted to people under 70 years of age. Moreover, the decline in mortality began well before coronary artery bypass surgery was introduced in New Zealand. To suggest that surgery alone was responsible for a major part of the decline (26-42%) requires a complex explanation of the overall decline as other factors must therefore be operating in the population subgroups not exposed to surgery.

Another explanation would be that the major factors in the decline must be those to which the whole population is exposed. Evidence from New Zealand shows, in fact, that dietary changes and, to a lesser extent, a reduction in tobacco consumption are likely to be the major factors in the decline in coronary heart disease mortality.³

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Eczema herpeticum: a potentially fatal disease

SIR,—As Dr I R Sanderson and colleagues (14 March, p 693) rightly point out, eczema herpeticum—and eczema vaccinatum before smallpox vaccination was stopped—is a potentially fatal disease. During 1950-5, 16 cases of eczema herpeticum were seen at the Hospital for Sick Children, Great Ormond Street.¹ Of these, 11 were considered to be severe, with fever lasting more than 12 days. In September 1953 an outbreak of eczema herpeticum occurred in the skin ward of the hospital. Six severe cases developed, and in one of these the patient died on the tenth day of the illness.² The outbreak probably occurred because of incorrect diagnosis in the first case,^{2,3} and herpes virus infection was spread by two nurses who had cutaneous vesicular lesions on the hands. All cases of herpes infection were confirmed by virus isolation. In the patient who died diffuse necrotic

lesions were found in the adrenals and also in the liver and lungs.² The presence of herpetic type inclusion bodies and isolation of virus from these organs indicated that herpes virus was responsible for these typical and atypical necrotic lesions. In two subsequent fatal cases necrotic lesions were found confined to the adrenals.

One of the patients affected by the ward outbreak subsequently suffered four separate attacks of eczema herpeticum with thrombocytopenia and died at the age of 4 years with diffuse eczema herpeticum, chronic interstitial pneumonitis, and advanced glomerulonephritis, complicated by an overwhelming infection with *Pseudomonas pyocyanea*.³ Some years later, when this case was reviewed, it was realised that this patient was probably suffering from the Wiskott-Aldrich syndrome.

I agree with Dr Sanderson and colleagues that early identification and treatment may be life saving, as is recognition of the mode of spread. Unfortunately, when these cases were seen modern methods of treatment of eczema were not then available.

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Outbreak of Weil's disease in a food fad commune in India

SIR,—In response to the queries made by Dr Singh (14 February, p 443), we would like to reiterate that in our report the patient in the index case, which alerted us to the fact that rat meat was the source of infection, did not belong to the Mussher commune and did not participate in the catching, handling, or cooking of the rats.

Musshers are basically wandering communes and are considered to be untouchable. A few such communes, however, have undergone socio-cultural changes. After independence considerable efforts were made to bring about agricultural reform, and some landowners started to employ members of Mussher communes as agricultural workers and allowed them to settle permanently outside the main village. Nevertheless, they are still at the mercy of the landowners, and because they are considered to be untouchable they have never been allowed to live inside the villages.

One group of the tribe still wanders from one village to another, its members earning their living by performing various plays and tricks.

As we stated in our article, the rats were grilled hastily over a makeshift bonfire, without the use of cooking utensils or the addition of salt during cooking. Salt was smeared on the meat only at the time of consumption, and it is therefore highly unlikely that salt destroyed leptospira, especially at the centre of the rat meat. Dr Singh should also be aware of the fact that in most of rural India the villagers believe that the sprinkling of salt over fire invites a catastrophe like leprosy.

It is true that leptospirae usually fail to survive in gastric acid, but they can easily enter abraded skin or mucosa.¹ In the index case the leptospirae in the contaminated food may have entered through the upper gastrointestinal mucosa or escaped destruction by gastric hydrochloric acid.

Though some of the Musshers' habits might