I escaped the syndrome first described in my tribe by Dr Cicely Williams¹ was simply because my educated parents took the same steps that Professor Marsden described from Brazil: I was fed with beans, eggs, milk, minced meat, and Ovaltine. Less fortunate relatives of my age whose parents also had three children in less than three years, and of whom it was also said "afor ese kwasiorkor," went on to develop kwashiorkor because they could only afford to be fed with maize products such as akasa and kenky.

The sibling positional word became almost invariably associated with the syndrome Cicely Williams was investigating, and although the term kwasiorkor was occasionally attached to a child who never went on to get the disease, whenever the syndrome kwashiorkor was seen in a child there was, more likely than not, very close proximity to a younger sibling. Exceptions occurred when children were orphaned, regardless of sibling positioning, or in multiple births.

A further argument in support of Professor Marsden relates to what has come to be known as the adult kwashiorkor syndrome, in which diarrhoea, pitting oedema, hepatomegaly, dermatopathy, mental apathy, and hair changes result from protein energy malnutrition as, for example, in the creatorrhoea and steatorrhoea in severe alcoholic chronic pancreatitis. It is known that the syndrome in the adult is completely reversible with appropriate treatment and protein energy replacement.

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Ambulance response times

SIR,—We were concerned to read Mr John Warden's report that the time allowed for ambulances to respond to urgent calls from general practitioners is to be relaxed.¹

We have reviewed one week of emergency work by South Glamorgan Ambulance Service. The 999 system was used to initiate 318 journeys. The service transported 121 trauma patients, 36 of whom were admitted, and 195 non-trauma patients, 105 of whom were admitted. Urgent calls from general practitioners resulted in 234 ambulance journeys: 22 trauma patients, eight of whom were admitted, and 212 non-trauma patients, 209 of whom were admitted. General practitioners thus generally perform an effective filtering mechanism for acute medical care, leading to a more efficient use of the emergency ambulance service.

There were 20 cases of acute myocardial infarction during the study. The nine patients who used the 999 system were attended to on average 12 minutes later whereas the 11 patients who called their general practitioner first had a significantly slower mean activation and response time of 53.8 minutes ($p < 0.02$; unpaired t test). To this time must be added the delay between calling a doctor and the doctor's arrival.

The reason for the prolonged ambulance response time is that attending doctors do not always stipulate an immediate response for patients with chest pain. In a separate study of patients admitted to our hospital with a diagnosis of cardiac chest pain from general practitioners during November 1989 we found that only 23% of patients had an immediate response; general practitioners stipulated ambulance transfer within 30 minutes for 40% of patients and within 30-60 minutes for 28%. Nine per cent of patients arrived by their own transport.

For cardiac patients at least, a relaxation in response times should not be entertained.

Given that British patients are still most likely to call a doctor if they have chest pain,² general practitioners should be encouraged to ask for an immediate ambulance response. The ambulance service should give as much priority to these calls as to 999 calls. "The best system is one in which both the general practitioner and the ambulance service respond without delay."³

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Response of older women to invitations for breast screening

SIR,—Dr P Hobbs and colleagues¹ comment on anecdotal evidence that the general practitioner's letter that accompanied the screening invitation had an important influence on attendance, probably because older women tend to hold their general practitioner in high esteem.

In a recent survey in the North Western region I found that the doctor's letter did have an influence,² particularly for certain types of women in the age range 50-64. Of the women who attended, 43% said that the doctor's letter had helped them make up their mind to go to be screened and 57% said that they would have gone anyway. Women who were anxious about the invitation who had not previously heard of the breast screening unit or were nervous about mammography were more likely to say that the doctor's letter had helped them make up their minds to go, with 60% of each group endorsing this statement.

Factors against using general practitioners' letters include the cost of the stationery and the extra work entailed in arranging for the letters' preparation. As the uptake of breast screening is of great concern the positive effect of the letter is important especially as it seems to influence those women who may not have attended otherwise.

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Chronic fatigue syndrome

SIR,—Dr Anthony David and colleagues¹ cite our paper² as one that makes inflated claims about the chronic fatigue syndrome.

We first reported retrospectively an association between antibodies to coxsackie virus B and a group of symptoms similar to those previously described as myalgic encephalomyelitis.³ We were faced with an ever increasing clinical problem of which we had little understanding, and the prospective investigation of coxsackie virus B antibody titres in these patients seemed a reasonable step forward. No widely accepted definition of the chronic fatigue syndrome existed in 1983, and we did not attempt to define it. We approached the problem from the opposite direction in that we had a definable test and we tried to show what happened to the results of this test in a group of ill patients.

Since 1983 much research into this syndrome has been carried out. It has taken a long time for a consensus to be agreed defining the syndrome. We believe that today's definition that the syndrome cannot be diagnosed before six months has elapsed is acceptable. In our study 72% of our patients were still unwell six months into the illness.

The comparison made by Dr David and colleagues of their paper with ours is invalid. They questioned 611 general practice attenders whereas we reported on a group of 140 patients presenting over six months with what we believe to be the same illness.

In retrospect we think that what we observed was the slow spread of an infective agent through our town in 1983 and through neighbouring towns in our district in 1984 and 1985. The clinical syndrome coincided with a rise in the prevalence of coxsackie virus B antibodies in the general population from 10-12% in 1973-84 (we found 25% in 1983) to 55% in 1985-6.⁴ Since then our clinical impression has been one of a return to normal; we see occasional new cases but not as many as in 1983.

The prevalence of this condition seems to depend on the activity of an infective agent of some kind, be it viral or otherwise, in the area of study at the time, and further research is made difficult by the wide fluctuations of prevalence that will be found from place to place and from time to time.

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Do streptococci cause toxic shock?

SIR,—Drs Sunil Shaunak and A M Gordon emphasise the role of both toxin production and septicaemia in group A streptococcus related toxic shock syndrome,¹ but toxin elaboration alone may explain the features of this illness, as we have previously reported,² and this is the case with staphylococcal toxic shock syndrome.³ Given the similarity of the two conditions, it would be prudent to start treatment with antibiotics effective against both organisms in case of doubt.

We disagree that 12 million units of intravenous benzylpenicillin given daily, on which Drs Shaunak and Gordon saw one treatment failure, is standard "high dose." We routinely give 16-24 million units daily to all our adult patients with known or suspected group A streptococcal infection and, when improvement is unsatisfactory, increase the dose up to 48 million units daily (8 MU four hourly). No treatment failures or severe side effects have been seen with such treatment despite a heavy caseload of serious streptococcal infections in this unit.

The authors suggest erythromycin or clindamycin as alternative treatments. Erythromycin resistance was found in 13% of group A streptococci referred to the streptococcal reference laboratory at Colindale (PHLS, personal communication) but this is an overestimate of the situation nationwide. There is also an appreciable risk of pseudo-membranous colitis with clindamycin.⁴ As an adequate response may depend critically on dosage even though the organism is highly sensitive to the antibiotic in vitro we recommend high dose benzylpenicillin as used at this centre for serious group A streptococcal infection. Flucloxacillin may be