pessimism of Mead and Peter C Rubin, who comments on the controversy, be refuted. Moreover, to achieve this it will be necessary to improve the education of all doctors and medical students about the availability of treatments and clinical trials that are suitable for their patients.

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Prophylaxis against malaria

EDITOR,—Santanu Chatterjee expresses concern about the advice given on prophylaxis against malaria, especially to young people travelling for long periods on low budgets.¹ Almost a quarter of 452 foreign travellers studied by Chatterjee in Calcutta were not taking any prophylaxis against malaria. The reasons for this are not reported.

Regulations amending the NHS (General Medical Services) Regulations 1992, which came into effect in England and Wales on 6 February this year, gave general practitioners the power to charge patients for prescribing or providing drugs for prophylaxis against malaria for travel abroad.² This means that private prescription forms will now have to be used to prescribe drugs for prophylaxis against malaria if the drugs cannot be obtained over the counter. The table gives the costs to the patient of a six week course of each of the recommended drug regimens for prophylaxis.

The cost of chemoprophylaxis against malaria for travellers, especially those with low budgets, may be a barrier to their taking any or the most appropriate chemoprophylaxis. For example, the increasing prevalence of strains of *Plasmodium falciparum* resistant to chloroquine and proguanil, the cheaper antimalarials, has resulted in maffoquine, which is much more costly, being recommended as the drug of choice for travel to east and central Africa.³ Of the 3551 cases of malaria reported in the United Kingdom during 1992 and 1993, 1983 were due to *P falciparum*. Fourteen of these 1983 patients died, 12 of whom had contracted the disease in Africa and two in India.⁴ Eight of the 14 patients who died had taken no prophylaxis at all.

If the cost to travellers of chemoprophylaxis against malaria has been a barrier to any or the most appropriate chemoprophylaxis being taken in

the past then the recent changes to regulations may result in an increased number of cases of falciparum malaria being imported into England and Wales, with a resultant increase in the number of deaths from the disease. The short term savings to the NHS resulting from the change in the regulations may be short sighted.

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Profiting from the NHS

EDITOR,—Chris Ham seems gloomy about private finance for NHS trusts for building projects.¹ This could, however, herald a new era, providing modern health service building without the restrictions of the public sector borrowing requirement.

A crisis is developing in the management of acute care through the reduction of trainees' hours of duty, the introduction of higher standards² and new treatments, and, in my specialty, an acute shortage of candidates for consultant posts. It is essential, therefore, to concentrate emergency services on as few sites as practicable; this may mean rebuilding, which would also introduce an opportunity to provide the facilities required for the appropriate level of care.³

In the United States "not for profit hospitals" (analogous to trusts) raise money at interest rates that depend on their credit rating and are based on their history of repayment of loans, the status of the institution, and inspection of the project. NHS institutions have no such history, so that capital for whole hospital projects may either be subject to unsustainable interest rates or be unavailable. Fundholders will certainly, as Ham writes, have to give long term commitments to NHS trusts but cannot cover a pay back period of 20 years or more. Some unquantifiable risk will remain. Perhaps optimum terms could be arranged with a minority contribution of capital (but also repayable) from government funds. This subsidy would make schemes viable, producing a more reasonable residual value in the case of total loss (which must remain a possibility). This may seem impure but be a practical solution.

Ham also seems to be criticising private sector involvement in the control of trusts that borrow. The history of NHS capital expenditure is grim (for example, at the University Hospital of Wales, Cardiff, millions were spent on decaying facades and fire precautions), and there is widespread evidence of great waste in running costs.⁴ There is room for improvement in the management of both

the capital project (which should be completed in time and at the cost stated in the contract) and subsequent recurrent expenditure. More careful scrutiny would therefore be welcome. It is up to purchasers to ensure that their objectives and standards are being met and that short term financial considerations do not receive undue weight. Instead of being looked on as selling off the silver this should be looked on as a golden opportunity for the health service to make rapid progress in updating building for the next century. But it will need the profession's and government action to make it happen.

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Community psychiatric nurses and drug regimens

EDITOR,—Now that Mrs Patricia Cooksley, a nurse who was sacked by Plymouth Hospital NHS Trust in July 1994 because she failed to obtain a doctor's signature on a repeat prescription, has been reinstated, discussion of the issues can take place in a more detached way. The issues surrounding medication are certainly not new, and they have implications for the medical profession.

A study of a community psychiatric nursing service which I carried out for a health authority in the 1980s provides evidence that community psychiatric nurses have long had to face dilemmas over their role in providing drug treatment.

Community psychiatric nurses differed in how far they were prepared to intervene in the oral medication of patients. Half of the 22 nurses in the area studied did modify doses; a further quarter were prepared to increase the dose but by only a single tablet, and they reported to the consultant within a few hours, rather than the more usual 24 hours. There were less overt ways of influencing medication: on occasion, particularly in the case of depot injections, nurses undermined consultants' treatment programmes by emphasising to patients their rights of refusal. Other nurses saw medication as only one of several approaches to mental illness. They viewed medication as the province of doctors and not a remedy which they as nurses would want to use, meddle with, or perpetuate.

Consultants played an important part in determining how some community psychiatric nurses behaved in regard to medication. Nurses whose action was not supported by a changed prescription given after the event would (presumably) not modify dosages again. So, it was not always the nursing profession, or nursing managers in the health authority, or individual nurses who were fixing their own behaviour; rather, it was the medical profession determining standards.

Increasingly, community psychiatric nurses were coming into contact with patients who were looked after solely by general practitioners. Sometimes general practitioners seemed to have less expertise concerning the drugs used in psychiatry than the nurses, and the nurses could find themselves in the anomalous position of advising those who, in fact, carried the responsibility for prescribing.

While variable and ill defined ways of working cannot be satisfactory, for the most part it seemed likely that the involvement of community psychiatric nurses in medication was safe. The practices had clearly evolved among people who had worked together for some time, not because

Cost (£) to patient of six week course of chemoprophylaxis against malaria if obtained over the counter, on NHS prescription, or on private prescription

Drug regimen	Over the counter	NHS prescription	Private prescription*
Chloroquine 300 mg/week	0.76	5.25	7.53
Proguanil 200 mg/day	6.93	5.25	11.9
Chloroquine 300 mg/week and proguanil 200 mg/day	7.69	10.5	19.43
Mefloquine 250 mg/week	Not available	5.25	22.35
Maloprim one tablet/week† and chloroquine 300 mg/week	Maloprim not available	10.5	15.19
			(2 charges by GP) 9.19
			(1 charge by GP)

GP=general practitioner.

*Includes general practitioner's charge to patient of £6 (charge recommended by BMA), cost of drug(s), and pharmacist's dispensing charge.

†Maloprim costs £5.25 on NHS prescription and £7.66 on private prescription.

they were irresponsible, but because there was mutual respect between professionals and this way of working was helpful and useful to patients.

As community care gains pace and more patients with ever more complex conditions are cared for at home, the questions surrounding medication will increase for all staff in the community.

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GPs' attitudes to a self diagnosis of myalgic encephalomyelitis

Sufferers continue to be misrepresented

EDITOR,—Shonagh Scott and colleagues' paper on general practitioners' attitudes to self diagnosed myalgic encephalomyelitis illustrates, if nothing else, the continuing misrepresentation of this illness and those who suffer from it.¹ Contrary to the authors' claims, Action for ME has never encouraged self diagnosis, and nor have the other "active support organisations" in Britain. Moreover, we have never advocated that patients should make unreasonable demands on their general practitioners.

Despite what Scott and colleagues imply, it is not just patients who recognise the existence of myalgic encephalomyelitis but also the World Health Organisation (the disease appears in the *International Classification of Diseases* (10th revision)), several handbooks, and many doctors. Indeed, positive attitudes to fatigue syndromes such as myalgic encephalomyelitis have been noted in several studies in the past few years. For instance, Ho-Yen and McNamara surveyed 178 general practitioners in Scotland and found that 71% accepted the existence of the disorder.² In New Zealand the figure was 90%.³

With regard to the dangers of diagnosing myalgic encephalomyelitis, I have not come across any evidence in the literature that correct diagnosis is associated with maladaptive attitudes, resentment, or "unnecessarily prolonged disability" except in a tiny minority. Indeed, the latest research available to us (unpublished) suggests that early diagnosis and appropriate advice help over 80% of patients to improve within six months. There is no evidence that self diagnosis is more common in myalgic encephalomyelitis than in early multiple sclerosis.

Finally, the vignettes used in the article do not describe a typical case of myalgic encephalomyelitis. Indeed, one of the psychologists who advise us thought that the list of symptoms was more indicative of clinical depression. It is not surprising, therefore, that the doctors responded to the different descriptions in slightly different ways.

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Evidence supports presence of encephalitis

EDITOR,—Although the precise pathoetiology of myalgic encephalomyelitis remains the subject of debate, Shonagh Scott and colleagues are incorrect in asserting that "no evidence exists" of encephalitis.

Buchwald *et al* carried out a large cohort study in which neurological symptoms, results of magnetic resonance imaging, and lymphocyte phenotyping suggested that the patients were experiencing "a chronic, immunologically mediated inflammatory process of the central nervous system."² More recently, Schwartz *et al*, who used single photon emission computed tomography, described abnormalities that were consistent with the hypothesis that "a chronic viral encephalitis" may be present.³ Furthermore, in the only postmortem study to have been published the polymerase chain reaction showed enteroviral sequences (compatible with coxsackie virus B3) in samples from the hypothalamus and brain stem,⁴ indicating that viral persistence within selective parts of the central nervous system may also play a part.

Given the uncertainties surrounding both the pathoetiology and the diagnostic criteria for myalgic encephalomyelitis, it is not surprising to learn that self diagnosis produces difficulties in the doctor-patient relationship. The conclusions of and motives behind Scott and colleagues' study must, however, be questioned in view of the fact that the fictitious patients had a list of vague symptoms that failed to satisfy diagnostic criteria for either a chronic fatigue syndrome (as defined by the International Chronic Fatigue Syndrome Study Group)⁵ or a postinfectious fatigue syndrome (as defined by current British criteria).⁶ Neither did the symptoms accord with those that patient support organisations would agree constitute a satisfactory diagnosis of myalgic encephalomyelitis.

I must also correct the mistaken belief that support groups such as the ME Association "encourage" self diagnosis. Our booklet *Guidelines for the Care of Patients* places considerable emphasis on the fact that several physical and psychiatric conditions can produce chronic fatigue as a principal symptom and that these may well need to be excluded before myalgic encephalomyelitis is confirmed. Even so, if patients are still faced with a general practitioner who "doesn't believe in myalgic encephalomyelitis" what option do they have but to make a provisional diagnosis using their own initiative?

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Cot death among Maori

EDITOR,—Charles Essex implies that the cot death rate has failed to fall in the Maori community because "advice not to share the bed has alienated leaders of some ethnic groups, who claim that it goes against traditional infant care practices."¹ Essex seems to be placing the blame directly on to the Maori community and absolving the public health system for its failure to deal with cot death among Maori.

The higher cot death rate in Maori than non-Maori since the national cot death prevention programme was started in 1991 is to do with the profile of risk factors of the Maori community—that is, the high rate of maternal smoking and the prevalence of multiple risk factors in Maori households—and the inappropriateness of much of the prevention programme's strategy for the Maori community.

Bed sharing is one example of the inappropriateness of the campaign's activities. After bed sharing was announced as the fourth modifiable risk factor,² and despite consistent professional advice and advice from lay Maoris that this message would be highly offensive to a large proportion of Maori mothers, at the end of 1991 the national cot death prevention programme began to promulgate in the mass media the message that bed sharing is a risk factor for cot death.

When it later became clear that bed sharing is a risk only when the mother smokes³ the Maori cot death prevention programme forcefully advocated that smoking should be the main behaviour targeted for change in the media campaign, with the issue of bed sharing being left to a trained primary care worker. The campaign's message, however, continued to be strongly against bed sharing.

The New Zealand Cot Death Study Group recently supported the stand taken by Maori with regard to the message about bed sharing, saying that there is only a small marginal gain to be had by extending the message from smoking mothers to non-smoking mothers. This should be balanced against the fact that bed sharing is common and that current favourable attitudes to it may jeopardise the acceptance of other messages aimed at preventing cot death.⁴

Appropriate messages should be developed by the appropriate people. The reason for the failure of the 1991 campaign to reduce rates of cot death in the Maori community is not the "alienation of the leaders of some ethnic communities" but the standard of the public health services delivered to the Maori community.

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Mortality among twins

EDITOR,—Kaare Christensen and colleagues' study of the lifetime mortality of the 24% of Danish twins whom they were able to trace is offered as a challenge to the validity of the fetal origins hypothesis.¹ The authors assume that if growth retardation in fetal life were associated with cardiovascular disease then twins, who have a lower birth weight than singleton infants even after adjustment for gestational age, would have relatively higher mortality. Thus their finding of a similar mortality in twins and singletons is taken as evidence against the fetal origins hypothesis. In the same manner the similar mortality of monozygotic and dizygotic twins is presented as a defence of criticisms that the twin method for estimating the contribution of genetic and environmental factors to disease may