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.

BILLIE SHEPPERDSON
Research associate

Nuffield Community Care Studies Unit,
Faculty of Medicine,
University of Leicester,
Leicester LE1 7LT

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 Indeed, the latest research available to us (unpublished) suggests that early diagnosis and appropriate advice help over 80% of patients to improve within six months.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.

MARTIN ARBER
Chairman

Action for ME,
PO Box 1302,
Wells BA5 2WE

1 Scott S, Deary I, Pelosi AJ. General practitioners' attitudes to patients with a self diagnosis of myalgic encephalomyelitis. *BMJ* 1995;310:508. (25 February.)
2 Ho-Yen DO, McNamara I. General practitioners' experience of the chronic fatigue syndrome. *Br J Gen Pract* 1991;41:324-6.
3 Denz-Penhey H, Murdoch JC. General practitioners' acceptance of the validity of chronic fatigue syndrome as a diagnosis. *NZ J Med* 1993;106:122-4.

Evidence supports presence of encephalitis

EDITOR,—Although the precise pathoaetiology 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, decribed 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 pathoaetiology 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?

CHARLES SHEPHERD
Honorary medical adviser

ME Association,
Stanford le Hope,
Essex SS17 0HA

1 Scott S, Deary I, Pelosi AJ. General practitioners' attitudes to patients with a self diagnosis of myalgic encephalomyelitis. *BMJ* 1995;310:508. (25 February.)
2 Buchwald D, Cheney PR, Peterson DL, Berch H, Wormsley SB, Geiger A, *et al.* A chronic illness characterised by fatigue, neurologic and immunologic disorders, and active human herpes virus type 6 infection. *Ann Intern Med* 1992;116:103-13.
3 Schwartz RB, Komaroff AL, Garada BM, Gleit M, Doolittle TH, Bates DW, *et al.* SPECT imaging of the brain: comparison of findings in patients with chronic fatigue syndrome, AIDS dementia complex, and major unipolar depression. *AJR* 1994;162:943-51.
4 McGarry F, Gow J, Behan PO. Enterovirus in the chronic fatigue syndrome. *Ann Intern Med* 1994;120:972-3.
5 Fukada K, Straus SE, Hickie I, Sharpe MC, Dobbins JG, Komaroff A, *et al.* The chronic fatigue syndrome: a comprehensive approach to its definition and study. *Ann Intern Med* 1994;121:953-9.
6 Sharpe MC, Archard LC, Banatvala JE, Borysiewicz LK. Clare AW, David A, *et al.* A report—chronic fatigue syndrome: guidelines for research. *J R Soc Med* 1991;84:118-21.

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 smokes[3] 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.

DAVID TIPENE-LEACH
Director of Maori cot death prevention programme

Department of Community Health,
Auckland University School of Medicine,
Private Bag 92019,
Auckland,
New Zealand

1 Essex C. Cot deaths: New Zealand: campaign falters over bed sharing. *BMJ* 1995;310:8-9. (7 January.)
2 Mitchell EA, Taylor BJ, Ford RPK, Stewart AW, Becroft DMO, Thompson JMD, *et al.* Four modifiable and other major risk factors for cot death: the New Zealand study. *J Paediatr Child Health* 1992;28(suppl 1):S3-8.
3 Scragg R, Mitchell EA, Taylor BJ, Stewart AW, Ford RPK, Thompson JMD, *et al.* Bed sharing, smoking, and alcohol in the sudden infant death syndrome. *BMJ* 1993;307:1312-8.
4 Scragg R, Stewart AW, Mitchell EA, Ford RPK, Thompson JMD. Bedsharing and smoking in the sudden infant death syndrome; public health policy implications of results from the New Zealand cot death study. *N Z Med J* (in press).

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