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 Indeed, the latest research available to us (unpublished) suggests that early diagnosis and appropriate advice help over 80% of patients to improve within six months.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

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 Fukuda 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 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.

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

be invalid because of the differing prenatal environments of monozygotic and dizygotic twins.

Clearly, however, low birth weight does not have the same importance in twins as in singletons. Studies based on the results of serial ultrasound scanning show that many twins have lower growth rates than singletons in the first trimester.² The fetal origins hypothesis states that coronary artery disease and its associated conditions result from growth retardation during later pregnancy and consequent disproportionate size at birth.³ Animal studies show that the setting of a low growth trajectory during early pregnancy protects the fetus against the effects of undernutrition in later pregnancy.⁴ Hence early down regulation of fetal growth may protect some twins from the effects of undernutrition in late gestation. A balance between the protective effects of early down regulation and the detrimental effects of later undernutrition may be the reason for the unexceptional overall mortality in twins and for the similar mortality of monozygotic and dizygotic twins.

DI W PHILLIPS
Clinical scientist

Metabolic Programming Group,
University of Southampton,
Southampton General Hospital,
Southampton SO16 6YD

- Christensen K, Vaupel JW, Holm NV, Yashin AY. Mortality among twins after age 6: fetal origins hypothesis versus twin method. *BMJ* 1995;310:432-5. (18 February.)
- Leveno KJ, Santos-Ramos R, Duenhoelter JH, Teisch JS, Whalley PJ. Sonar cephalometry in twins: a tale of biparietal diameters for normal twin fetuses and a comparison with singletons. *Am J Obstet Gynecol* 1979;135:727-30.
- Barker DJP, Gluckman PD, Godfrey KM, Harding JE, Owens JA, Robinson JS. Fetal nutrition and cardiovascular disease in adult life. *Lancet* 1993;341:938-41.
- McCance RA, Widdowson EM. Nutrition and growth. *Proc R Soc Lond B Biol Sci* 1974;185:1-17.

Temperature and cardiovascular mortality

EDITOR,—Owen L Lloyd has fallen into some of the common pitfalls when discussing cold and its effects on the body.¹ The environmental cold stress (rate of heat loss) experienced by warm blooded creatures is related not just to absolute temperatures outdoors or indoors but also to movement of air (wind or draughts)—for example, a body loses more heat at 10°C with a wind of 32 km/h than at -10°C in still air.² Similarly, humidity (rain or indoor damp) increases the heat loss, and, as Peter Wilmshurst states,³ the risk of ischaemic heart disease is also related to rainfall.²

Another factor is that the physiology of the body reacts to changing stimuli, rapid change producing more response than slow change. It also adapts to a steady state—for example, 20°C can induce shivering after exposure to 30°C, whereas 0°C feels warm to someone who has left a room at -30°C with wind from a fan. It is therefore to be expected that total cold stress (temperature, wind, and rain) would have more effect on health if the severity changed frequently and rapidly.

The social class gradient in risk of ischaemic heart disease (rising from I to V) is present throughout England and Wales. This can be at least partly explained by housing.⁴ People in the higher social classes tend to have centrally heated houses with minimal internal temperature gradients, whereas people in the lower social classes, with less money, live in houses which are often damp, draughty, and difficult to heat. If they heat only one room they have frequent changes in cold stress as they move around the house. In Glasgow, people in Drumchapel have a higher incidence of ischaemic heart disease than people in affluent neighbouring Bearsden.

The risk of ischaemic heart disease in the regional health authorities of England clearly rises as the climate becomes colder, wetter, windier, and more changeable, mainly to the west and

north.⁵ This even overrides the social class factor, so that people in social class I in north and northwest England have a higher risk than those in social class V in East Anglia.

Scotland is similar to England in that the west has more wind, more rain, and a more changeable climate than the east. On this basis it is easy to understand why the west of Scotland has a higher incidence of ischaemic heart disease and stroke than the east. There is therefore no Scottish paradox related to climate. Similarly, the genetic influence is negligible since people who change their place of residence rapidly acquire the risks of ischaemic heart disease of their new area of residence.⁴

E L LLOYD
Consultant anaesthetist

Department of Anaesthetics,
Western General NHS Trust,
Edinburgh EH4 2XU

- Lloyd OL. Temperature and cardiovascular mortality. *BMJ* 1995;310:467. (18 February.)
- Lloyd EL. *Hypothermia and cold stress*. London: Croom Helm, 1986.
- Wilmshurst P. Temperature and cardiovascular mortality. *BMJ* 1994;309:1029-30. (22 October.)
- Lloyd EL. The role of cold in ischaemic heart disease: a review. *Public Health* 1991;105:205-15.
- Balarajan R, Yuen P, Machin D. Inequalities in health: changes in RHAs in the past decade. *BMJ* 1987;294:1561-4.

Undernotification of whooping cough

EDITOR,—Douglas Jenkinson is to be commended for his painstaking documentation of whooping cough in general practice.¹ He points out that the incidence observed in his practice between 1977 and 1992 was six times the rate of notifications in England and Wales over the same period and highlights an appreciable number of cases among adults. This prompted me to examine notification data on whooping cough in Cardiff.

During 1984-92 there were 1034 notifications of whooping cough in Cardiff, giving an annual notification rate of 42 cases/100 000 population compared with the 26 cases/100 000 for England and Wales. The table shows the age distribution of patients in Cardiff compared with those reported by Jenkinson in Keyworth. Although there was a

Distribution of cases of whooping cough by age in Cardiff and Keyworth.¹ Figures are numbers (percentages)

Age (years)	Cardiff* (n=1013)	Keyworth† (n=500)
0	118 (11.6)	15 (3.0)
1	113 (11.2)	35 (7.0)
2	163 (16.1)	43 (8.6)
3	143 (14.1)	86 (17.2)
4	155 (15.3)	59 (11.8)
5	133 (13.1)	53 (10.6)
6	59 (5.8)	51 (10.2)
7	31 (3.1)	30 (6.0)
8	20 (2.0)	17 (3.4)
9	13 (1.3)	21 (4.2)
10-14	23 (2.3)	33 (6.6)
15-19	8 (0.8)	7 (1.4)
≥20	34 (3.4)	50 (10.0)

*Notification in 1984-92.

†Consecutive cases in general practice in Keyworth, 1977-92.

similar excess of cases in females (47% male and 53% female v 44% male and 56% female in Keyworth), there was a higher proportion of cases in young children and a lower proportion in older children and adults among the 1013 notified cases in Cardiff in which age was known. This suggests that either selective underdiagnosis or undernotification of adult cases is occurring. Since notification rates in Cardiff were broadly comparable to those for England and Wales as a whole this is probably not just a local phenomenon.

As whooping cough becomes less common, such irregularities in the notification system may

assume increasing importance. Coverage for the primary course of immunisation against whooping cough is now 92%, but many older children and young adults have never been immunised. A situation analogous to that with measles might therefore develop, with an upward shift in the age distribution of whooping cough. This has already been documented in the United States, with an increase in reported cases in subjects aged 10 and over from 15.1% during 1977-9 to 19.8% during 1980-9 and 26.9% during 1992-3.² This is an argument in favour of a pertussis vaccine booster as the opportunity for natural boosting of immunity through exposure to the disease diminishes.^{3,4} Furthermore, the use of a clear clinical definition of whooping cough⁵ and a greater awareness among doctors of the possibility of infection in adults would considerably enhance our ability to track the epidemiology of the disease.

MERION R EVANS
Consultant in communicable disease control
South Glamorgan Health Authority,
Cardiff CF4 3QX

- Jenkinson D. Natural course of 500 consecutive cases of whooping cough: a general practice population study. *BMJ* 1995;310:299-302. (4 February.)
- Resurgence of pertussis—United States, 1993. *MMWR* 1993;42:952-60.
- Novelli V, Al-Ansari H, Mok Q, Tasker R. Pertussis vaccination: is there a need for a booster dose? *Lancet* 1994;344:1225-6.
- Miller E, White JM, Fairley CK. Pertussis vaccination. *Lancet* 1994;344:1575-6.
- Centers for Disease Control. Case definitions for public health surveillance. *MMWR* 1990;39(RR-13):1-42.

Management of severe gastroparesis diabeticorum

EDITOR,—The debate over the management of gastroparesis diabeticorum reflects how difficult this condition is to manage.¹ We have had similar difficulties in managing a 46 year old man with longstanding diabetes treated with insulin.

The patient had various long term complications, including peripheral neuropathy, diabetic nephropathy (evident on renal biopsy), and impotence as well as hypertension and angina. He was admitted recurrently with abdominal pain, nausea, and vomiting. On each occasion he received intravenous fluids and insulin and a nasogastric tube was inserted. His symptoms were so severe that he was in hospital almost continuously over six months. Gastroparesis was confirmed by a radionuclide meal, which showed delayed gastric emptying of both solids and liquids. All proved therapeutic agents and withdrawal of his anti-hypertensive and antianginal agents were tried. His glycaemic control was optimised, but the only treatment that afforded relief was nasogastric intubation.

He was discharged with a nasojejunal feeding tube for a trial of enteral feeding, which was successful. We proceeded to permanent percutaneous endoscopic gastrojejunostomy feeding, the tube being inserted under local anaesthesia without complication. The double lumen system allowed drainage of retained gastric contents while facilitating jejunal feeding through a soft biliary tube. The patient was free of symptoms and remained at home for three months. He was readmitted with a recurrence of his symptoms due to blockage of the gastrojejunostomy tube. Attempts to replace the tube were unsuccessful. A gastrojejunostomy and feeding jejunostomy were fashioned surgically under local anaesthesia through a limited laparotomy; this was also unsuccessful. He developed intractable vomiting and worsening renal failure and died of metabolic complications.

We believe that our patient would not have withstood more radical surgical options; in any case, their efficacy has not been established.² The development of percutaneous gastrostomy tubes