The identification of a specific food allergen is difficult and was not achieved in this patient. The persistence of her symptoms during treatment with an elemental diet does indeed suggest that there was continued antigenic stimulation at that time, which continues, as she is now receiving a normal diet and is asymptomatic.

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Aspirin as prophylaxis against migraine

SIR,—I was surprised by Dr K J Zilkha's advice (14 February, p 427) about the use of aspirin as migraine prophylaxis. It has been suggested that the platelet inhibitory effect is the important factor.[1] If this is so 300 mg on alternate days may be a sufficient dose, causing minimal gastric side effects.

Since I read Hanington's article[1] I have taken aspirin daily. Before this I suffered disabling attacks of classical migraine every three to six weeks, but since I began taking aspirin daily during the past eight and a half years I have had only one attack, which occurred when I did not take aspirin for a fortnight. Those who suffer from such frequent attacks have perhaps one worry free week in three as they are either suffering an attack or awaiting the next one with trepidation. The relief of knowing that no further attacks should occur is such that I now become quite anxious if my supply of aspirin runs low.

Are Dr Zilkha's three patients statistically significant? My own experience is supported by a controlled trial,[2] and I would certainly recommend an antiplatelet dose of aspirin to any sufferer.

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1 Hanington E. Migraine: a blood disorder. *Lancet* 1978;ii:501-2.
2 O'Neil BP, Mann JD. Aspirin prophylaxis in migraine. *Lancet* 1978;ii:1179-81.

Ulcerogenicity of piroxicam: an analysis of spontaneously reported data

SIR,—We are concerned that the study by Dr Allen C Rossi and coworkers (17 January, p 147) depended entirely on reported cases of drug related complications, because in the United Kingdom only a minority of such complications are reported to the Committee on the Safety of Medicines.[1] Only a prospective study that relates local ulcer complications to drug prescriptions can assess the incidence of adverse reactions to non-steroidal anti-inflammatory drugs.[2-4]

Over three years we studied prospectively all 235 serious peptic ulcer complications in south Cheshire (population 250 000). Patients were included if they died because of, or required emergency surgery for, a peptic ulcer complication. Thirty two of these 235 patients were using steroids, taking two non-steroidal anti-inflammatory drugs, or taking a non-steroidal anti-inflammatory drug that has subsequently been withdrawn, leaving 203 in our study group. Of the 203 patients, 113 were taking a non-steroidal anti-inflammatory drug, of whom 23 (20%) were using piroxicam. Of these 23, 21 (91%) were over 60 years of age. Nine of the patients using piroxicam died, and all were over the age of 60. These complications were not related to the dose, the duration of use, or the reason for the prescription of piroxicam.

During the same period a consecutive group of 1246 hospital control patients without known peptic ulceration were questioned closely about the use of non-steroidal anti-inflammatory drugs. Of these, 123 (10%) had been using such drugs before admission, and 13 of these were taking piroxicam. Thus although piroxicam was used by only 11% of the control patients who were taking non-steroidal anti-inflammatory drugs, it was associated with 23 out of 113 (20%) ulcer complications related to such drugs ($\chi^2=4{\cdot}362$, $p<0{\cdot}05$).

Our study confirmed that only a minority of drug related adverse reactions are reported to the Committee on the Safety of Medicines and showed that patients taking non-steroidal anti-inflammatory drugs who develop serious peptic ulcer complications are more likely to be using piroxicam. Piroxicam seems to be more ulcerogenic than other such drugs, possibly because of its long half life and the altered pharmacokinetics in the elderly. We would therefore advise against its use as an anti-inflammatory agent in patients over 60 years of age.

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1 Committee on Safety of Medicines. Non-steroidal anti-inflammatory drugs and serious gastrointestinal adverse reactions. 1. *Br Med J* 1986;292:614.
2 Collier DStJ, Pain J. Ulcer perforation in the elderly and non-steroidal anti-inflammatory drugs. *Lancet* 1986;i:971.
3 Blower AL, Armstrong CP. Ulcer perforation in the elderly and non-steroidal anti-inflammatory drugs. *Lancet* 1986;i:971.
4 Ng J, Batey R. Ulcer perforation in the elderly and non-steroidal anti-inflammatory drugs. *Lancet* 1986;i:972.

Identity cards for patients infected with HIV?

SIR,—Recent articles in the *BMJ* concerning identity cards for carriers of the human immunodeficiency virus (HIV) (Dr A C Srivastava and others, 21 February, p 495) and in *Pulse* concerning a general practitioner's right to be informed of the HIV state of his or her patients do not, in my view, give sufficient weight to the importance of maintaining confidentiality and the consequences to the victim if confidentiality is broken.

I would like to add to this debate illustrations of the effects of the acquired immune deficiency syndrome (AIDS), and rumours about AIDS, in the small, geographically isolated community in which I work.

We have treated three cases of full blown AIDS so far. In each case the patient's identity has become common knowledge, forcing the patients to leave the area in search of anonymity. Unfounded rumour and malicious gossip have been rife. One of our patients was a businessman, and his business subsequently collapsed. Another local businessman had to publish an article in the local press to clear his own name because of widespread rumours that he also had AIDS, indeed he was said to be in hospital dying of AIDS. A relative of one of our patients has also been forced to write her story in the local newspaper in an attempt to scotch unfounded rumours and enable her to set up her own business. In addition, a "blacklist" of those said to be infectious for AIDS is being circulated locally. The motive for this seems malicious as it is a foolproof method of harming business and social rivals.

I have had to speak at a meeting of parents at a primary school, where panic was growing because of an unfounded rumour that the parent of a child at the school had AIDS.

I am sure that, having experienced the prevailing attitudes, any future sufferers will certainly refuse to carry an identity card or be labelled in any way. They will be extremely cautious about who is informed of their antibody state, no doubt trusting their general practitioner but perhaps not the receptionist.

I hope that this glimpse of the reactions of an insular community will cause those who think that they have a right to know to have second thoughts. For a disease that carries an extremely low risk of transmission to health workers but in which a breach of confidentiality can be devastating I believe that attempts to label victims are quite wrong and that those who know should be strictly limited, mainly in accordance with patients' wishes.

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SIR,—The question posed by Dr A C Srivastava (21 February, p 495) was not answered by your four experts. Surely if people know that they are carriers of the human immunodeficiency virus (HIV) it is morally wrong for them not to inform doctors who may be affected when they fall ill. Their sexual proclivities are irrelevant, and the paranoia of certain groups should not enter the discussion. Surely it is reasonable that people who know that they are HIV positive should carry a card identifying themselves as such in case an injury renders them unconscious and thus unable to communicate.

Trauma surgeons who deal with unconscious patients, with the best will in the world, often splash blood in their eyes and sustain needlestick injuries during surgery. Dr A J Pinching says that no additional measures are required by health care workers, but this is not the case in operating theatres, where a surgeon has a responsibility not only to the patient but also to staff, his own family, and himself. The carrying of cards identifying carriers (not identity cards) of HIV cannot in itself infringe any personal freedom or human rights.

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Diagnostic classification of the aetiology of mental retardation in children

SIR,—In their suggested scheme for the investigation of children with mental retardation (17 January, p 163) Drs Simon J Newell and Stuart H Green state that "routine testing is done in the hope of classifying cases in which there are no specific clues to aetiology" and cite three studies in which no unsuspected diagnosis was made. We recently undertook a study of 169 children at schools for the educationally subnormal in Southampton to assess what investigations would be valuable.[1] Biochemical screening included amino acid analysis—quantitative in mothers, qualitative in children—and testing of thyroid function in children. Results were negative, and we would agree that such tests need not be performed unless specifically indicated. Chromosome analysis, however, identified five children with previously unsuspected abnormalities of relevance (2-47, XXY, 1-48, XXYY, 1 X autosome translocation, 1 deletion in chromosome 15). None of these children had dysmorphic features, one had a behavioural problem, and two had had perinatal anoxia. Had chromosome analysis been confined to children with congenital abnormalities, or to

those without environmental insults or behavioural problems, as suggested in Drs Newell and Green's algorithm, diagnoses would have been missed.

The three studies quoted were performed more than 10 years ago. Subsequent refinements in techniques of chromosome analysis allow identification of minor chromosome abnormalities that might previously not have been recognised—for example, in our series the X autosome translocation and the chromosome 15 deletion. We recently saw a child with developmental retardation and hypotonia with no "hard" dysmorphic features who was 48, XXYY and a boy with mild retardation and pronounced behavioural disorder who was fragile (X) positive. Neither of these children would have had chromosomes checked under the algorithm. Boys with the fragile (X) syndrome may have fits, but these should not debar them from chromosome analysis.

We would therefore recommended that chromosome analysis should be included in the routine investigation of children with mental retardation, irrespective of findings in the history or clinical examination.

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1 Lamont MA, Dennis NR, Seabright M. Chromosome abnormalities in pupils attending ESN (M) schools. *Arch Dis Child* 1986;61:223-6.

Stress hyperglycaemia and cause of death in non-diabetic patients with myocardial infarction

SIR,—We reported an association between hypoglycaemia and poor outcome after acute myocardial infarction in subjects with concentrations of glycosylated haemoglobin that indicated a low probability of premorbid diabetes mellitus.[1] The major determinants of hyperglycaemia in these patients were raised concentrations of adrenaline, noradrenaline, and cortisol, which were generally independent of the size of the infarct. Drs M W Beckett and D J Shane (29 November 1986, p 1441) noted a previously described relation between raised catecholamine concentrations and subsequent risk of cardiac arrest[2] and requested an analysis of the plasma glucose concentrations in relation to the cause of death of patients with myocardial infarction.

The table shows the plasma glucose concentrations in patients admitted with acute myocardial infarction who had normal (6·9%) or borderline (6·9-7·8%) glycosylated haemoglobin concentrations. Patients were classified according to major cause of death, so that patients in cardiogenic shock were identified as such even if they were suffering from a terminal arrhythmia. Three of the patients in our study were not included in the table because of deaths from unrelated causes. Patients with normal glycosylated haemoglobin concentrations had significantly higher plasma glucose concentrations in both groups of patients who died compared with survivors. Because of the numbers studied the raised plasma glucose concentrations were seen only in patients dying of pulmonary oedema or cardiogenic shock in the borderline abnormal glycosylated haemoglobin group. Though there was a trend for the plasma glucose concentrations to be higher in patients dying of pulmonary oedema or cardiogenic shock than in those dying of cardiac arrest, the differences were not significant.

We reported an association between hyperglycaemia and poor outcome, mainly due to heart failure, even in patients with small infarcts. These data suggest that hyperglycaemia may occur as a result of raised catecholamine and cortisol concentrations in patients with pulmonary oedema or cardiogenic shock. The finding of hyperglycaemia, however, in patients who subsequently died of cardiac arrest suggests that high catecholamine concentrations may contribute to, as well as reflect, a poor prognosis.

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1 Oswald GA, Smith CCT, Betteridge DJ, Yudkin JS. Determinants and importance of stress hyperglycaemia in non-diabetic patients with myocardial infarction. *Br Med J* 1986; 293:917-22.
2 Little RA, Frayn KN, Randall PE, *et al*. Plasma catecholamines in the acute phase of the response to myocardial infarction. *Archives of Emergency Medicine* 1986;3:20-7.

Mean (SD) plasma glucose concentrations in patients admitted with acute myocardial infarction related to glycosylated haemoglobin concentration and outcome

Glycosylated haemoglobin (%)	Survivors		Patients who died from pulmonary oedema or cardiogenic shock		Patients who died from primary cardiac arrest	
	No	Glucose	No	Glucose	No	Glucose
<6·9	146	7·89 (2·44)	27	10·99 (3·99)***	9	9·85 (2·87)**
6·9-7·8	34	9·88 (3·38)	13	12·70 (4·19)*	3	11·57 (3·15)

Patients *v* survivors: *p<0·02, **p<0·01, ***p<0·0001 (Mann-Whitney U test).

Relation between phenotype and banal melanocytic naevi

SIR,—Whereas an unexpected association between a large number of naevi and a dark complexion was shown by Dr J S C English and colleagues (17 January, p 152), we have shown that subjects with a light complexion tend to have more naevi.[1] Our study was hampered, however, by the heterogeneity of the subjects' ages (children and young adults) and by the fact that all visible naevi were counted, irrespective of diameter, although small naevi may be confused with freckles or other pigmentary lesions.

We therefore studied the prevalence of naevi in a group of 508 white students aged 18-30. For practical reasons, naevi were counted only on the chest, back, and legs. Pigmentary traits were analysed according to the subjects' self reported histories of burning and tanning (tendency to burn after one hour of exposure to sun in early summer and tendency to tan after regular exposure to sun during the summer, respectively). The table shows the median numbers of naevi for the various categories. The Kruskal-Wallis test was used to measure the significance in each subgroup.

The number of naevi seemed to be strongly related to complexion, those with light complexions generally having more naevi. This was particularly true of women, possibly because of larger group sizes.

Median number (range) of naevi in volunteers related to self reported tendencies to burn and tan

Complexion trait	No of subjects	No of naevi: 2-5 mm in diameter	No of naevi: >5 mm in diameter
Men			
Tendency to burn:			
None	92	7·5 (0-74)	1 (0-18)
Mild	65	11 (0-68)	1 (0-25)
Severe	20	12 (0-30)	1·5 (0-15)
Significance		p=0·23	p=0·12
Tendency to tan:			
Intense	31	6 (0-74)	0 (0-9)
Moderate	99	9 (0-68)	1 (0-18)
None	47	13 (0-49)	1 (0-25)
Significance		p=0·08	p=0·08
Women			
Tendency to burn:			
None	140	5 (0-66)	0 (0-19)
Mild	134	6 (0-76)	0 (0-12)
Severe	57	8 (0-49)	1 (0-23)
Significance		p<0·01	p=0·01
Tendency to tan:			
Intense	34	4 (0-29)	0 (0-5)
Moderate	162	5 (0-44)	0 (0-11)
None	135	8 (0-76)	1 (0-23)
Significance		p<0·01	p=0·01

It is rather disturbing that our findings clearly contrast with those of Dr English and colleagues. They rightly emphasise that large numbers of naevi are the strongest risk factor for melanoma. Furthermore, the risk of melanoma is greater in subjects with light complexions, and therefore those with light complexions would be expected to have more naevi. As counts of naevi may have important implications for the epidemiology and aetiology of cutaneous melanoma further investigations are warranted.

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1 Rampen FHJ, van der Meeren HLM, Boezeman JBM. Frequency of moles as a key to melanoma incidence? *J Am Acad Dermatol* 1986;15:1200-3.

Syringe driver in terminal care

SIR,—With regard to Dr Simon Dover's article on syringe drivers (28 February, p 55), many of our patients receiving cancer chemotherapy for solid tumours now have subclavian lines or implanted venous access devices. These may remain safely in place for many years, being maintained by the patients themselves. When the time comes to withhold further active treatment for the tumours we try to ensure that these lines are kept in place as they are extremely useful in terminal management. We believe that the systemic venous route allows better control of blood concentrations than subcuticular or intramuscular administration, and we suggest that, wherever possible, these lines should be kept in place and used.

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SIR,—We use syringe drivers, as described by Dr Simon Dover (28 February, p 553), on the general wards of our district general hospital as well as in the community and would like to make two points about their use.

Firstly, the nurses have become so used to them