# Letters

## Progress in reducing inpatient mortality from acute myocardial infarction is slow

EDITOR—Brown et al report a substantial increase in the use of beneficial interventions for patients admitted to hospital with acute myocardial infarction in Nottingham from 1982 to 1992.[1] However, they found no change in inpatient mortality adjusted for age and sex. This contrasts with findings in Ontario, Canada, where hospital discharge abstracts show that inpatient mortality fell from 1981 to 1991[2] and has since fallen further (data on request). Brown et al suggest that the disparate results may reflect coding errors in the Ontario administrative data.

Our group has assessed the classification of deaths from acute myocardial infarction in inpatients in the Ontario discharge abstracts. Matching Ontario administrative data to data from the GUSTO-I and LATE trials, we found the specificity of the administrative data to be 100% (1403/1403 alive), with overall sensitivity of 94% (104/111 deaths detected).[3]

The classification of acute myocardial infarction itself is more problematic. The sensitivity of the primary diagnosis of acute myocardial infarction is high, but specificity varies.[4 5] There are two common reasons for false positive coding. Firstly, patients may be admitted to a coronary care unit with possible acute myocardial infarction but this diagnosis is ruled out and they are sent home quickly. Alternatively, patients recently admitted to hospital with acute myocardial infarction are readmitted for coronary angiography or with complications other than overt reinfarction, and the code for acute myocardial infarction is wrongly used again. Thus the analysis of outcomes in Ontario excluded both patients with a primary diagnosis of acute myocardial infarction who were discharged in under four days and patients who had been admitted to hospital for acute myocardial infarction in the preceding three months.[2]

It is therefore doubtful that imprecise coding explains the improving outcomes of acute myocardial infarction in Ontario. Indeed, only systematic coding errors could explain the observed trends in inpatient mortality, and we have found neither evidence nor a rationale for this.

Ontario's administrative data do mirror the findings of Brown et al in two respects[2]: admissions for acute myocardial infarction, particularly among elderly patients, increased over the period studied, and inpatient mortality ranged from 16% to 21%, which is much higher than in the large randomised trials of thrombolytic treatment. Thus while the trends in Ontario are more positive than those in Nottingham, we agree with Brown et al that the cardiovascular megatrials have been enrolling skewed patient populations[3] and that progress in the battle to reduce inpatient mortality from acute myocardial infarction remains slow.

**C David Naylor** *Chief executive officer*
**Jack V Tu** *Scientist*
Institute for Clinical Evaluative Sciences, University of Toronto, 2075 Bayview Avenue, North York, Ontario M4N 3M5, Canada

1 Brown N, Young T, Gray D, Skene AM, Hampton JR. Inpatient deaths from acute myocardial infarction, 1982-92: analysis of data in the Nottingham heart attack register. *BMJ* 1997;315:159-64. (19 July.)
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## Risk of testicular cancer in boys with cryptorchidism

### Study was based on small number of cancers

EDITOR—Swerdlow et al identified 11 testicular cancers in a cohort of 1182 boys treated for undescended testes in the 1950s and early 1960s and make several contentious observations.[1] Their main, and most startling, observation is of a relation, possibly causal, between testicular biopsy at orchidopexy and later malignancy. Their interpretation, however, is based on a small number of cancers, which detracts from this unexpected conclusion. Similar studies have had a much larger denominator (for example, 794 testicular tumours in the most recent report of the UK Testicular Study Group[2]). Secondly, of all the factors that were looked at, testicular biopsy is the least random. Whatever the authors say about what was in the operation notes, something about the testicular appearance must have been the reason for biopsy. Otherwise why do it in 120 testes and not in the other 1285?

I also take issue with the statistical comparison of the effect of biopsy (irrespective of non-descent). The obvious comparison is between biopsy and non-biopsy in the 1405 testes at risk. This yields a relative risk of 5.5 (95% confidence interval 2.8 to 10.7)—significant perhaps, but far less impressive than the quoted 66.7 (23.9 to 143.3).

There is also the equally contentious suggestion (albeit not made explicit) that intra-abdominal testes (n = 199) do not have an increased risk of malignancy. Again this is presumably a result of small numbers. The authors do not state what was done with these testes and what the result was. Denis-Browne or Torek orchidopexies (certainly as one stage procedures) are not technically possible with the vast majority of these testes because the vessels are too short. If done as two stage procedures, they are associated with a high (up to 40%[3]) incidence of atrophy. Many surgeons take the view that orchidectomy is a valid option if the condition is unilateral.[4] In this cohort, I assume that either excision or atrophy was a probable outcome. Clearly, excision removes the organ at risk, and atrophy may be associated with an increase in risk. Nevertheless, both are confounding factors. This is not mentioned in the paper, and therefore we have to assume that all 199 testes were brought down into the scrotum intact and without recourse to further intervention—which seems unlikely.

**Mark Davenport** *Consultant paediatric surgeon*
Department of Paediatric Surgery, King's College Hospital, London SE5 9RS

1 Swerdlow AJ, Higgins CD, Pike MC. Risk of testicular cancer in cohort of boys with cryptorchidism. *BMJ* 1997;314:1507-11. (24 May.)
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4 Gough MH. Cryptorchidism. *Br J Surg* 1989;76:109-12.

### Authors' reply

EDITOR—Our results relating to biopsy cannot be compared with results in the case-control literature, which has not investigated this variable. For those variables that have been examined in case-control studies, the comparative power of these studies is not well represented by the number of testicular tumours, since the great majority of these tumours were not in cryptorchid testes. It is also important when comparing studies to take into consideration the quality

of the data and biases in the different approaches as well as power. Clearly, however, although our cohort is larger than those published previously, the number of testicular cancers is not large and leads to wide confidence intervals in several analyses.

We agree with Davenport (and we stated in our paper) that the reasons why testes were selected for biopsy may be the factors predictive of a raised risk of malignancy. Our comment about the absence of reasons stated in the operation notes does not imply that there were no reasons, simply that the reasons were not recorded in the notes as far as we could find.

As Davenport states, the difference in risk between biopsied and non-biopsied testes in the study is important: that is why we provided the significance of this difference ($P < 0.001$). We did not suggest, explicitly or otherwise, that intra-abdominal testes are not at increased risk of malignancy. We simply analysed risk by site of testis and gave relative risks for each category. The result for abdominal testes had a wide confidence interval, based on a small number of expected tumours, and it would certainly not be sensible to conclude that there is no raised risk of malignancy in such testes just because no malignancies occurred in them in our study. Indeed, as we have noted elsewhere, the risk of testicular cancer is probably higher in abdominal than inguinal maldescent.[1] The results in the present study, with a small number of abdominal testes, do not contradict this.

Finally, Davenport notes that some testes may have been excised and would not then have been at risk of malignancy. As we noted in the paper, there were indeed orchidectomies in the cohort, and the numbers of these were stated. As we also noted, these testes were not included in the risk calculation after they were excised.

**A J Swerdlow** *Professor of epidemiology*
**C D Higgins** *Lecturer in medical statistics*
Epidemiological Monitoring Unit, London School of Hygiene and Tropical Medicine, London WC1E 7HT

**M C Pike** *Professor of preventive medicine*
Department of Preventative Medicine, University of Southern California School of Medicine, Los Angeles, CA 90033-0800, USA

1 Swerdlow AJ. Epidemiology of testicular cancer. In: Raghavan D, Scher HI, Leibel SA, Lange PH, eds. *Principles and practice of genitourinary oncology.* Philadelphia: Lippincott-Raven, 1997:643-52.

## Trial of thyroxine treatment for biochemically euthyroid patients has been approved

Editor—Skinner et al's suggestion that patients with symptoms of hypothyroidism and normal results of thyroid function tests might benefit from treatment with thyroxine[1] received considerable publicity in our local evening newspaper. As a result, several patients were referred to our clinic.

Since they complained of a considerable reduction in their quality of life, which had not been helped by other measures, we decided that it was justifiable to try treating two of them with 100 µg thyroxine daily (after we had explained the lack of scientific rationale and obtained their written consent). Much to our surprise, they both reported a considerable improvement in their condition, while the results of thyroid function tests remained within the reference range; one of them returned to work after an absence of four years. Although this may well have been a placebo response, it should be noted that such patients are often given repeated courses of antidepressants (at worst an expensive and dangerous placebo) without apparent effect.

While our present state of knowledge suggests that there is no scientific justification for this treatment, it is intellectually arrogant to assume that we know everything about the physiology of thyroid secretion and its controlling hormones or the pharmacological effects of exogenous thyroxine.[2] In view of the lack of effective treatment for this group of patients, we believe that further investigation of the effect of thyroxine is justified, as Skinner et al proposed. We have now received approval from our local ethics committee for a double blind, placebo controlled trial of thyroxine in patients with symptoms of hypothyroidism and normal results of thyroid function tests.

**E H McLaren** *Consultant physician*
**C J G Kelly** *Specialist registrar in endocrinology*
**M A Pollock** *Principal biochemist*
Stobhill NHS Trust, Glasgow G21 3UW

1 Skinner GRB, Thomas R, Taylor M, Sellarajah M, Bolt S, Krett S, et al. Thyroxine should be tried in clinically hypothyroid, but biochemically euthyroid patients. *BMJ* 1997;314:1764. (14 June.)
2 Giving thyroid hormones to clinically hypothyroid but biochemically euthyroid patients [letters]. *BMJ* 1997;315: 813-4. (27 September.)

## Doctors have moral imperative to call for end to embargo on Cuba

*See editorial by Delamothe*

Editor—The news story on Cuban refugees who mutilated themselves in order to enter the United States missed an important point.[1] The Cuban economy has collapsed for two main reasons: there has been reduced economic support from the former Soviet Union during the past decade, but the main problem has been the trade embargo imposed on Cuba by the United States in 1960. This embargo was strengthened by the Helms Burton Act in 1996. Before the act was passed, the United Nations tried repeatedly to have the embargo repealed; in 1995 the vote was 137 against the embargo and three in favour. I travelled extensively in Cuba in 1993, visiting hospitals and talking to doctors. The conditions then were poor but have deteriorated further since the Helms Burton Act.

The editorial from the *New England Journal of Medicine* on which the news article was based adds that "economic sanctions are, at their core, a war against public health. … Thus, as physicians, we have a moral imperative to call for the end of sanctions."[2] A report by the American Association for World Health found that the embargo resulted in "malnutrition, poor water quality and the denial of access to medical equipment and drugs."[3] With a change in government and with a Foreign Office more interested in human rights issues it is time for British doctors to be calling for the embargo against Cuba to be lifted.

**Delia Parnham-Cope** *Specialist registrar in accident and emergency medicine*
Conquest Hospital, St Leonards on Sea, East Sussex TN37 7RD

1 Rutter T. Cuban refugees injured themselves to get into the United States. *BMJ* 1997;314:1297. (3 May.)
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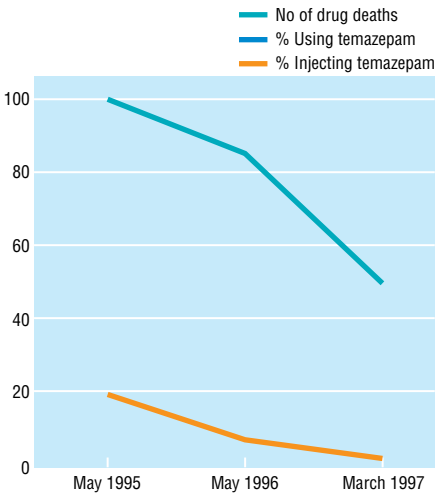
## Reduction in use of temazepam is factor in deaths related to overdose

Editor—The reported increase in drug related deaths in Lothian[1] has not been mirrored in Strathclyde. Despite a large increase in the amounts of methadone prescribed in Glasgow[2] the rate of death due to drug overdose has fallen from eight a month in 1995 to four a month in the first half of 1997 (Strathclyde Police drugs squad, personal communication). The system of prescribing as described by Gruer et al[2] has shown that methadone can be associated with improvements in mortality, in contrast to the experience elsewhere.

The dramatic drop in deaths related to overdose in Strathclyde is not, however, principally due to the methadone programme. The other main change in drug use in the area has been the reduction in use of temazepam since this drug was rescheduled as a controlled drug in January 1996. This is shown in the drug histories of those




Number of deaths due to drug overdose and percentages of drug misusers using and injecting temazepam

attending the Glasgow Drug Crisis Centre over the past two years. Overall use of the drug and, particularly, its injection have fallen to a quarter of the previous levels (figure).

Careful use of methadone is of central importance in the treatment of injecting drug users. Although it often reduces all cause mortality,[3] because of methadone's toxicity it rarely reduces mortality due to overdose.[4] The success of the rescheduling of temazepam shows that such public health measures can also have positive effects in mortality due to overdose.

**T C Gilhooly** *Visiting medical officer*
Glasgow Drug Crisis Centre, 123 West Street, Glasgow G5 8BA

1 Greenwood J, Zeally H, Gorman D, Fineron P, Squires T. Deaths related to methadone have doubled in Lothian. *BMJ* 1997;314:1763. (14 June.)
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## Local research ethics committees

### Oxford committee was concerned that trial might be unethical

EDITOR—Stone and Blogg call for a national research ethics committee and cite in support of their argument their recent experience with an application concerning multicentre research, which was submitted to central Oxford research ethics committee.[1] They are perhaps unaware of the imminent establishment of regional multicentre research ethics committees, which will reduce the bureaucracy entailed in submitting proposals for multicentre research and may well provide some uniformity in decision making. We were surprised that they singled out their application to the central Oxford research ethics committee as an example of a local committee making a poor decision.

Stone and Blogg ask "what is peculiar about the Oxford local research ethics committee." Local research ethics committees have been exhorted to require systematic reviews of existing research before approving new proposals.[2] The central Oxford research ethics committee was perhaps "peculiar" because it (presumably unlike the other 120 committees) was aware of a particular unpublished systematic review. This review raised concerns that without a placebo group the proposed trial design might be unable to answer the scientific question and might therefore conceivably be unethical. Stone and Blogg were invited by letter to allay these concerns, but they failed to do so and the committee heard no more of their research proposal. The file on their application still remains open. It is therefore not correct to state that the research ethics committee would not allow them to take part in a large multicentre study without major revision of the protocol.

Local research ethics committees are on one hand asked to require systematic reviews before considering applications[2] and now, it would seem, on the other hand to ignore them.[1] Stone and Blogg could well have participated in this study if only they had addressed the concerns raised by the local research ethics committee and were able to provide satisfactory answers to a serious ethical question.

**D Talbot** *Chairman, central Oxford research ethics committee*
**D J M Reynolds** *Consultant clinical pharmacologist*
Oxford Radcliffe Hospital, Radcliffe Infirmary NHS Trust, Oxford OX2 6HE

1 Stone PG, Blogg CE. Local research ethics committees. *BMJ* 1997;315:60-1. (5 July.)
2 Savulescu J, Chalmers I, Blunt J. Are research ethics committees behaving unethically? Some suggestions for improving performance and accountability. *BMJ* 1996; 313:1390-3.

### Authors' reply

EDITOR—It was not our intention to criticise the central Oxford research ethics committee specifically but merely to highlight some general problems in the conduct of multicentre trials. We welcome the recently announced intention to streamline the ethical review of multicentre research in Britain through the use of multicentre research ethics committees.[1 2] However, prior approval by these committees will be an additional requirement for approval by the local research ethics committee and will not replace it. An extra layer of bureaucracy may result, but we trust that this will not be so and wait to see how the new arrangements develop in practice.

Research proposals must indeed be considered in the context of best current evidence, such as that assembled, appraised, and synthesised in a published peer reviewed systematic review.[3] An unpublished review that is not in the public domain cannot, however, be accorded such authority.

In any conflict of opinion in a multicentre trial individual participants are inevitably caught between the study's principal investigators and their own local research ethics committee. If the study protocol cannot be strictly adhered to, or if a local requirement for extensive revision causes substantial delays, participation is not possible. In our particular area of study the use of placebo groups has been much discussed and criticised.[4 5] In our view the denial of timely treatment of known efficacy to patients in a placebo group is a greater ethical concern than that posed by the lack of a placebo group causing a loss of scientific elegance. It would seem that the other 120 study centres were of the same opinion.

**Peter G Stone** *Senior registrar in anaesthetics*
**Colin E Blogg** *Consultant anaesthetist*
Oxford Radcliffe Hospital, Nuffield Department of Anaesthetics, Radcliffe Infirmary NHS Trust, Oxford OX2 6HE

1 Department of Health. Multi-centre research ethics committees. *CMO's Update* 1997;15:8.
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### Research discovers the right thing to do; audit ensures that it is done right

EDITOR—I would like to resolve the apparent contradictions identified by Scott and Pinnock[1] in the BMA's publication *Ethical Issues in Clinical Audit*.[2] The authors stated that there was a contradiction between the two sentences "audit is intended to influence the activities of an individual or team, i.e. local; but research attempts to influence medical practice as a whole" and "there is no need to consult research ethics committees on matters which are appropriately classified as audit." They are concerned that this confusion might mean local research ethics committees becoming overloaded with work.

The first issue concerns the distinction between research and clinical audit. Scott and Pinnock have concluded that, by publishing results of clinical audit nationally, authors are attempting to influence medical practice as a whole and the work must therefore be regarded as research. The level of dissemination of a piece of work does not determine whether it is audit or research. Although results of clinical audit have traditionally been restricted to local use, they now have increasing prominence in national and international journals. A succinct but generalised distinction is that "research is concerned with discovering the right thing to do; audit with ensuring that it is done right."[3] As systematic pieces of work, published clinical audit projects can inform others on best methods and be used as comparators for other clinical audit work. This does not equate with attempting to influence medical practice as a whole.

The second issue concerns the workload of local research ethics committees. As a general rule, any ethical issues of clinical audit, whether aiming to inform, educate, or influence an audience, are the responsibility of clinical audit committees. Only in exceptional cases (for instance, when a clinical audit committee cannot reach consensus on an ethical issue, or when it believes that the work is research, not audit) should it need to refer the matter to the local research ethics committee. As with all BMA publications, the book is kept under constant review. We will consider any revisions in the light of recent inquiries.

**Richard Rawlins** *Chairman, clinical audit committee*
BMA, Tavistock Square, London WC1H 9JP

1 Scott PV, Pinnock CA. Local research ethics committees. *BMJ* 1997;315:60. (5 July.)
2 BMA. *Ethical issues in clinical audit.* London: BMA, 1995.
3 Smith R. Audit and research. *BMJ* 1992;305:905-6.

### Monitoring body is needed for audit

EDITOR—Scott and Pinnock are right to point out that the BMA's advice on audit is confusing.[1] Audit cannot be defined by reference to who reads the report (local = audit; national = research). Under the terms of the BMA's guidelines, audits need not be submitted to local research ethics committees.[2] Those of us who advise on such matters, however, feel distinctly uneasy at having no equivalent body for clinical audit, acting with impartial expertise in the interests of patients.

Although the question of equipoise should never occur, the intrusion into the patient's personal feelings (by questionnaire, for example) may be every bit as great during an audit as during research. Distinguishing between audit and research, although usually straightforward, is a red herring in such cases. Surely we should be asking whether patients' participation could conceivably be harmful. If so, a monitoring body of some kind is required. Local research ethics committees would indeed be "bombarded with demands for prospective ethical approval," but this indicates a need to overhaul the monitoring system rather than to tweak definitions.

**Andrew Barton** *Coordinator*
Research and Development Support Unit, Plymouth Postgraduate Medical School, Derriford Hospital, Plymouth PL6 8DH

1 Scott PV, Pinnock LA. Local research ethics committees. *BMJ* 1997;315:60. (5 July.)
2 BMA. *Ethical issues in audit 1995*. London: Clinical Audit Committee, BMA, 1995.

## Total ban on landmines is unnecessary

*See editorial by Nathanson*

EDITOR—The announcement of a unilateral ban on the use and stockpiling of antipersonnel landmines by the government of the United Kingdom is the culmination of a vigorous media campaign by a coalition of agencies led by the Red Cross. Some media reports, however, have seriously distorted the facts about landmines.

The debate on the international regulation of landmines has been emotional and conducted by people with little firsthand experience. Much of the commentary is ill informed, and wider issues of landmine control have not been adequately discussed. A complete ban on antipersonnel landmines is impossible to enforce and not necessarily desirable. The number of mines said to be still active in various countries is in the tens of millions, but there is little evidence to support these estimates. Some have claimed that landmines made in Britain injure civilians in many countries. This is in contradiction to our experience: over the past five years we have worked with landmine clearance teams in many heavily mined countries and neither of us has ever seen a mine retrieved that was made in Britain.

On 12 May 1995 the United Kingdom adopted the joint action of the council of the European Union on antipersonnel mines. This action bans the export of all types of undetectable and non-self destructing landmines and restricts the export of self destructing weapons to countries that have signed the United Nations Weapons Convention.[1] It has been extended to ban the export of "all types of antipersonnel mines to all destinations."[2] Previously, official policy had been to work for an immediate international ban on the use of mines that are not fitted with self destructing or self neutralising mechanisms and to work towards a total ban on the use of antipersonnel mines.

A better option would be to enact a treaty which permits the use of self destructing or self neutralising weapons but completely bans the use of non-detectable mines. The explosive charge could be limited to a maximum of 30 g; this would reduce the number of patients requiring amputations above the knee and thus lessen the problems of rehabilitation. This type of approach rather than an outright ban would increase the chance of the United Nations enacting a treaty or tightening protocol II of its weapons convention.

The medical profession has been in the forefront of the campaign for stricter regulation of landmines. As scientists we know that complicated situations cannot be reduced to black and white issues. Writing emotional sketches and exaggerating the known facts may sell newspapers, but it is not going to solve the problem.

**Eddie Chaloner** *General surgical registrar*
Royal London Hospital, London E1 1BB

**Steve Mannion** *Registrar in orthopaedic surgery*
Lewisham Hospital, London SE13 6LH

1 Council decision 12 May 1995. *Official Journal of the European Communities*. No L115. 1995 May 22;38:1-3.
2 House of Commons official report (Hansard). 1996 April 22;276:cols 28-9.

## Treating alcohol dependence

### One glass of wine is usually 1.5 units

EDITOR—In their article on alcohol dependence Ashworth and Gerada state that a unit of alcohol is equal to 10 ml[1] (which, incidentally, is approximately 8 g, not 10 g as they state). They also say that many doctors are unaware of the unit values for common alcoholic drinks. Indeed, they show this themselves by saying that a glass of wine contains one unit. Most wine is 12% alcohol and a glass is 125 ml (six glasses to a bottle). An average glass of wine therefore contains 15 ml of alcohol, or one and a half units (as indicated on many supermarket labels). It makes a big difference when calculating people's intake.

**David M Keeling** *Consultant haematologist*
Oxford Haemophilia Centre, Churchill Hospital, Oxford OX3 7LJ

1 Ashworth M, Gerada C. ABC of mental health. Addiction and dependence—II: alcohol. *BMJ* 1997;315:358-60. (9 August.)

### Supplementation with parenteral B vitamins should be routinely considered

EDITOR—We were pleased that the article on alcohol dependence mentioned use of parenteral B and C vitamins for a selected group of patients during detoxification.[1] Currently the use of B complex vitamins in alcohol withdrawal varies in Britain.[2]

The prevalence of brain lesions indicative of deficiency of B vitamins in long term alcohol misusers at necropsy has been reported to be as high as 35%.[3] Failure to treat severe deficiency of B vitamins is associated with a high morbidity and a 17-20% mortality.[3] Of the patients who survive, four fifths develop Korsakoff's psychosis and many will require long term institutionalisation.[3] It is therefore crucial to provide adequate supplementation with B vitamins as soon as possible. Oral supplementation is insufficient to restore depleted vitamin concentrations in long term heavy drinkers because it is not adequately absorbed.[4]

Patients requiring parenteral treatment are those at high risk of severe deficiency of B vitamins.[2] This arguably includes all those qualifying for inpatient detoxification. Patients who require inpatient detoxification but for whom no inpatient facilities are available, and those unwilling or unable to be admitted, must be considered for parenteral treatment.[2] As the Committee on Safety of Medicines recommends, facilities for treating anaphylaxis should be available when vitamins B and C are given parenterally.

The intramuscular route of giving vitamins B and C is associated with a reduced rate of adverse events, at one reaction per five million pairs of ampoules used, compared with 1 in 250 000 for the intravenous route.[2 5] In detoxification when a patient is at risk but not exhibiting signs or symptoms of severe deficiency of B vitamins the intramuscular route is appropriate.

Given the high prevalence and severity of deficiencies of B vitamins in alcohol withdrawal, the high morbidity and mortality in Wernicke's encephalopathy, and the lack of efficacy of oral supplementation, parenteral B vitamins should be routinely considered for all patients at high risk of severe deficiency of B vitamins.

**C C H Cook** *Professor of the psychiatry of alcohol misuse*
Kent Institute of Medicine and Health Sciences, University of Kent at Canterbury, Canterbury, Kent CT2 7NR

**A D Thomson** *Consultant gastroenterologist*
Greenwich District Hospital, London SE10 9HE

1 Ashworth M, Gerada C. ABC of mental health. Addiction and dependence—II: alcohol. *BMJ* 1997;315:358-60. (9 August.)
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### Chlormethiazole is widely used in Europe

EDITOR—In their article on alcohol dependence Ashworth and Gerada remark that "chlormethiazole is no longer recommended as attenuation therapy, particularly in general practice, because of the high risk of dependence and the lethal cocktail that results if it is taken with alcohol," but they do not support this assertion with any data.[1] In fact, dependence, like respiratory depression and other side effects, has been ascribed to chlormethiazole with little or no scientific basis.[2]

We do not know of any study showing that chlormethiazole has a dangerous interaction with alcohol compared with other sedatives used in alcohol detoxification. More attention has been paid to the possibility of dependence on chlormethiazole: several articles have reported cases of dependence, but there are few works reviewing this subject. Stille studied chlormethiazole's potential for leading to dependence and found that animal studies did not show any major physical or psychological dependence.[3] This author, analysing clinical literature covering 17 years, also found that the evidence for primary dependence was weak when subjects with a current or previous history of misuse of or dependence on other drugs were excluded. Since then, there have not been any new data supporting the hypothesis that the risk of dependence is high. We have done a Medline search and have not found any relevant study.

In a recent meta-analysis reviewing the pharmacological management of alcohol withdrawal the benzodiazepines were found to be suitable pharmacological agents.[4] Chlormethiazole was acknowledged to be superior to placebo for reducing signs and symptoms of withdrawal but was not included in the analysis because the size of the studies was not adequate for conclusions to be drawn on the prevention of seizures and delirium. We agree that benzodiazepines could be the treatment of choice in alcohol withdrawal, but they are also liable to misuse.[4] Chlormethiazole is widely used in Europe, mainly in alcohol withdrawal and in geriatric medicine, and we know of no evidence to cause us to disapprove its use.

**Andrés Herrán** *Psychiatrist*
**José Luis Vázquez-Barquero** *Professor*
Clinical and Social Psychiatry Research Unit, University Hospital "Marqués de Valdecilla," Santander, Spain

1 Ashworth M, Gerada C. ABC of mental health. Addiction and dependence—II: alcohol. *BMJ* 1997;315:358-60. (9 August.)
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### Acamprosate is another drug to help maintain abstinence

EDITOR—In their review of alcohol problems Ashworth and Gerada discussed ways of reducing relapse after detoxification in people who are dependent on alcohol.[1] They mentioned disulfiram but not acamprosate, despite its being the only other drug licensed in Britain for maintaining abstinence in alcohol dependence. Acamprosate is a relatively new drug, and clinical experience is limited, though several double blind randomised placebo controlled trials have shown its benefit. For example, Whitworth et al reported that 41 (18%) patients treated with acamprosate and 16 (7%) treated with placebo had been continuously abstinent over 12 months of treatment ($P = 0.007$).[2] If used, acamprosate should be started as soon as possible after abstinence begins, though it is not a treatment for alcohol withdrawal itself.

Whereas disulfiram enhances abstinence by acting as a deterrent to drinking, acamprosate does not cause an adverse reaction with alcohol. Its mechanism of action is unclear, but it may act by reducing craving for alcohol by modulating γ-aminobutyric acid and glutamate pathways.[3] Like disulfiram, it cannot be regarded as a treatment on its own; rather, it is an adjunct to psychosocial treatment programmes. Used in this way it seems to benefit some patients. Further work is needed to see if this subgroup can be predicted.

**Peter Haddad*** *Senior registrar*
**Christopher Daly*** *Consultant psychiatrist*
Department of Psychological Medicine, Hope Hospital, Salford M6 8HD

*The authors have never held any consultancy or other paid position with the manufacturers of acamprosate.

1 Ashworth M, Gerada C. ABC of mental health. Addiction and dependence—II: alcohol. *BMJ* 1997;315:358-60. (9 August.)
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3 Littleton J. Acamprosate in alcohol dependence: how does it work? *Addiction* 1995;90:1179-88.

### Authors' reply

EDITOR—Keeling raises the issue of underestimation of alcohol intake by the doctor. When this is compounded by failure even to ask about alcohol intake and underreporting by the patient it is easy to see why problem drinking so often goes undetected. Keeling is correct to say that 1 unit of alcohol equals 8 g and not 10 g, which we stated as an approximation. It is useful to bear in mind that patients may mean different things when they refer to a glass of wine and that a bottle of wine of ordinary strength contains 8 units even though there are only about six glasses of 125 ml in a bottle. In the more deprived inner city area in which we practise cans of beer are the preferred drink, being a cheap source of alcohol units, and we would reiterate that just one can of extra strong lager contains 4 units of alcohol.

The problems of deficiency of vitamin B in alcohol dependence need to be emphasised, and Cook and Thomson highlight that of absorption of oral vitamin B. We believe that treatment with vitamin B should be included more commonly in the management of patients undergoing detoxification in the community and should feature in all prescriptions alongside the prescription of chlordiazepoxide or other benzodiazepine. Whether detoxification in the community should now include administration of intramuscular vitamin B, and whether the doctors concerned need to be prepared to deal with rare but possible anaphylactic reactions, should probably be determined by controlled trials.

Although agreeing that benzodiazepines are the treatment of choice for detoxification, Herrán and Vázquez-Barquero state that chlormethiazole is widely used in Europe. McInnes has summarised the evidence against using chlormethiazole for detoxification in the community, and particularly its danger in accidental or deliberate self poisoning.[1]

Finally, Haddad and Daly mention the use of acamprosate. This new drug has a role in maintaining abstinence as an adjunct to psychosocial treatment after detoxification.[2] It may be used for up to a year and acts to reduce craving for alcohol. Because it produces no adverse reaction with alcohol it has no deterrent effect; its main role is in maintaining abstinence.

**Mark Ashworth** *General practitioner*
**Claire Gerada** *General practitioner*
Hurley Clinic, London SE11 4HJ

1 McInnes GT. Chlormethiazole and alcohol: a lethal cocktail. *BMJ* 1987;294:592.
2 Acamprosate for alcohol dependence? *Drug and Therapeutics Bulletin* 1997;35:70-2.

## Lactic acidosis induced by phenformin is still a public health problem in Italy

EDITOR—Phenformin is a well established cause of lactic acidosis, particularly in diabetic patients with renal failure. For this reason, in 1977, the drug was removed from the market in the United States. Since then, only sporadic reports of lactic acidosis induced by phenformin have been published in the North American literature, describing patients who have been prescribed the drug abroad.[1] The European Union does not yet have uniform rules for marketing drugs, and though phenformin has been removed from the market in most countries in the union, it is still marketed in Italy. In a Medline survey we found several recent reports of lactic acidosis induced by phenformin in Italian medical journals.[2-5]

In the past five years in our own centre for kidney diseases we have treated two patients with lactic acidosis induced by

phenformin. Both patients had chronic renal failure (serum creatinine concentration 248 and 265 mmol/l) and were taking a combination of phenformin and glibenclamide. The acidosis was severe (bicarbonate concentration 3.8 and 5.8 mmol/l), with a high anion gap (56 and 51 mmol/l). Serum lactate concentration in one of the patients was 20 mmol/l. Serum glucose concentration was low normal in one case (3.7 mmol/l) and moderately high in the other (11.1 mmol/l). Both patients were admitted in shock. One patient was treated with bicarbonate dialysis, and the other with massive infusions of saline and bicarbonate. Both patients recovered after stopping the drug and were well when discharged, with insulin being prescribed instead of oral drugs. Both patients had been referred after several days in medical wards, where phenformin treatment had been maintained. This implies that lactic acidosis induced by phenformin is poorly recognised.

We suggest that phenformin should be banned from the market in all European countries. In the meantime, doctors working in areas where it is still available must avoid its use, particularly in patients with renal failure. Lactic acidosis induced by phenformin should be suspected everywhere if suggestive symptoms occur in diabetic patients who have travelled in Italy.

Metformin has been introduced in both the United States and Europe because of its value in the treatment of overweight patients with non-insulin dependent diabetes. This drug has a lower potential for inducing lactic acidosis than phenformin and is considered safe in the general population; however, it remains a serious threat for patients with chronic renal failure.

**G Enia** *Vice director*
**M Garozzo** *Research fellow*
**C Zoccali** *Director*
Centro di Fisiologia Clinica del Consiglio Nazionale delle Ricerche, Reggio Calabria, Italy

1 Chung Lu H, Parikh PP, Lorber DL. Phenformin-associated lactic acidosis due to imported Phenformin. *Diabetes Care* 1996;19:1449-50.
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## Death of Diana, Princess of Wales

*See p 1457*

### People experiencing emotional difficulties react in different ways

EDITOR—Public emotion after the death of Princess Diana[1] has been associated with an increase in consultations with general practitioners for depression,[2 3] although it is not clear if this represents a true increase. Most people might show some emotion, but how do people experiencing emotional difficulties react? We have observed in psychiatric practice a range of responses, showing that an emotional response was incorporated into the cognitions of many of our patients, influencing their efforts to cope.

One common response was related to the individual's experience of loss. For example, an HIV positive gay man with a depressive illness in remission had given a friend's funeral eulogy days before the event. He felt he had coped well by "putting [his] grief behind [him]" despite the resonance with his situation. The public grief resulted in tearfulness and thoughts of his death, leading to a better attitude towards his future. He developed no symptoms of depression. In another instance, a widow unable to progress through her grief found that actively mourning Diana allowed her to express hidden emotions about her partner and her father, who had also died suddenly.

In another case a man who had been HIV positive for 13 years reported identifying with Diana's feelings of loneliness at being misunderstood and victimised. This enabled him to deal with his anger at people's response to his diagnosis in the early days of public awareness. Finally, a professional man coming to terms with his homosexuality was driven to strong feelings of anger about the public expression of emotion, which he perceived as threatening because he feared a loss of the carefully established control of his feelings and behaviour.

The intense public feeling seems to have influenced the cognitions and affect of people who were already vulnerable. The death of a public figure perceived as psychologically troubled but who seemed to have made a constructive adjustment could, by a process of identification, lead to feelings of loss and grief.[1] These could be magnified by observing others express emotions, a phenomenon recognised in situations such as suicidal behaviour.[4] What remains unclear is what the longer term effect of this public grief will be: life events research shows that there may be an increase in psychiatric morbidity.[5] A formal diagnosis of depression, however, requires symptoms to be present for 2 weeks (international classification of diseases, 10th revision). As in the above examples, this public and constructive expression of feelings may be beneficial, leading to a reduction in psychological distress.

**Edwina R L Williams** *Specialist psychiatric registrar*
Riverside Mental Health Trust, Psychological Medicine Unit, South Kensington and Chelsea Mental Health Centre, London SW10 9NG

**Jean Meadows** *Department and information coordinator*
**Jose Catalàn** *Reader in psychiatry*
Imperial College School of Medicine, Division of Neuroscience and Psychological Medicine, London SW10 8RP

1 Smith R. Death of Diana, Princes of Wales: a special life forged from adversity. *BMJ* 1997;315:562. (6 September.)
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### Her death and funeral rate as traumatic stressors

EDITOR—In a report of links between the death of Princess Diana and depression, Dr André Tylee, the director of the Royal College of General Practitioners' mental health unit, was reported as stating that people countrywide were experiencing grief and psychological distress.[1] However, the death of Princess Diana is not characteristic of traumatic events, or stressors, seen in the research literature on post-traumatic stress disorders arising from war combat, assault, and natural and human disasters. Nevertheless, symptoms associated with post-traumatic stress disorder can be found in people not directly exposed to the traumatic event[2] or exposed to stressors that do not meet the DSM-III-R criteria of severity.[3] In light of such findings, we measured the degree of psychological distress that the death and funeral of Princess Diana caused.

We used the impact of events scale,[4] which was designed to assess the impact of any specific traumatic event and has been shown to have sound psychometric properties. In relation to a specific event, respondents rate the frequency of intrusive thinking and avoidance tendencies during the previous 7 days. Scores of 0-8 are interpreted as subclinical, 9-25 as mild, 26-43 as moderate, and over 43 as severe, and a score $\geq 26$ is regarded as a "clinically significant reaction."[5]

Three weeks after Princess Diana's death the scale was administered to an opportunity sample of 205 respondents drawn from a city in the midlands. Of those indicating their age (47%), the mean was 40.97 (SD = 17.45, range 17-82) years. The death of Princess Diana was used as the specific traumatic event for 102 respondents and the funeral for 103.

The mean (SD) score on the impact of events scale was 16.94 (12.75) for the death of Princess Diana and 19.17 (13.32) for the funeral. Scores ranged from 0 to 44 for the death of Princess Diana (28% scored above the diagnostic cutoff score of 25) and from 0 to 57 for the funeral (32% above the cutoff).

These results show that the profound psychological impact of the death and funeral of Princess Diana can be equated with traditional stressors identified in the trauma research literature, evidenced by a substantial percentage (28-32%) of our sample that showed symptoms indicating post-traumatic stress.

**Mark Shevlin** *Lecturer*
**Viv Brunsden** *Researcher*
**Stephanie Walker** *Lecturer*
**Mark Davies** *Lecturer*
**Tina Ramkalawan** *Lecturer*
Quantitative Research Group, Department of Social Sciences (Psychology), Nottingham Trent University, Nottingham NG1 4BU

1 Morris B. GPs called into action to help nation recover from shock of Diana's death. *BMA News Review* 1997 September 24:18.
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## Review of interventions to prevent heart disease

### Study neglected to examine benefits of exercise

EDITOR—In their systematic review of interventions to reduce risk factors for coronary heart disease Shah Ebrahim and George Davey Smith examined changes in blood pressure, smoking, and blood cholesterol concentration.[1] Meta-analysis supports physical inactivity as a fourth risk factor.[2 3] Although the relative risk of physical inactivity (1.9) is similar to that of hypertension, smoking, and hypercholesterolaemia,[4] the population attributable risk is higher owing to its greater prevalence.[5] This suggests that interventions to increase physical activity may be efficacious in reducing the incidence of coronary heart disease.

Exercise was an intervention in nine of the 14 studies reviewed, yet no analysis of change in exercise was reported. Although disparate measurement methods often make the comparison of physical activity difficult between studies, conclusions about the efficacy of the exercise interventions would inform policymakers and be topical in light of the Health Education Authority's current national campaign to promote physical activity.

**David Batty** *Researcher*
Exercise and Health Research Unit, University of Bristol, Bristol BS8 2LU

1 Ebrahim S, Davey Smith G. Systematic review of randomised controlled trials of multiple risk factor interventions for preventing coronary heart disease. *BMJ* 1997;314:1666-74. (7 June.)
2 Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. *Annu Rev Public Health* 1987;8:253-87.
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4 Pooling Project Research Group. Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major chronic events: final report of the pooling project. *J Chron Dis* 1978;31:201-306.
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### Authors' reply

EDITOR—We agree with David Batty that a lack of exercise is an important risk factor in cardiovascular disease. The mechanisms by which exercise may influence the risk of cardiovascular disease are not clear but may include effects on other known risk factors. In a meta-analysis of 22 randomised controlled trials of exercise substantial reductions in blood pressure were reported, but these may have been confounded by concomitant weight loss.[1] Falls in body weight probably confound meta-analyses of the role of exercise in reducing blood cholesterol concentration too.[2] No effects of exercise on blood pressure or blood cholesterol concentration were found in the exercise trials reporting follow up of at least six months; this suggests that exercise interventions lack long term efficacy in reducing risk factors.[3]

Changes in exercise behaviour in multiple risk factor intervention trials

| Study | Sample size | Exercise measurement | Effect on exercise behaviour† (95% CI) |
|---|---|---|---|
| Cost effectiveness of lipid lowering study (*BMJ* 1995;310:1105-9) | 681 | Arbitrary exercise score | –0.01 (–0.21 to 0.20) |
| Oxcheck study (*BMJ* 1994;308:308-12) | 11 090 | Vigorous exercise less than once a month | Men: 5.6% (1.5% to 9.8%)* |
| | | | Women: 1.4% (–2.5% to 5.2%) |
| Primary prevention of hypertension trial (*JAMA* 1989;262:1801-7) | 201 | Self reported behaviour | 75% increase (control not reported) |
| | | Resting pulse rate | 0.04 beats/minute |
| | | Blood pressure response to exercise | 11.6 exercise maximum double product‡ |
| Tromsø family trial (*Prev Med* 1991;20:197-212) | | | |
| Men | 1 373 | Physically active in leisure activities | –2% (–4% to 0%) |
| Wives | 809 | | 5% (3% to 7%)* |

*P<0.05.
†Negative changes indicate decline in exercise or physical capacity in the intervention groups.
‡Derived from blood pressure and pulse rate.

The meta-analysis cited by Batty[4] is frequently used to support the view that exercise reduces the risk of cardiovascular diseases. Predominantly non-experimental observational studies were included that do not give any indication of the ability of exercise interventions to alter behaviour.

Our recent review of multiple risk factor interventions did not examine pooled changes in exercise because only four of the nine studies that used an exercise component provided information about changes in exercise, and this was inconsistently reported (table). Self reports of health behaviours in necessarily unblinded trials always raise the possibility of bias towards inflation of positive behaviour. Meta-analysis of the results shown in the table is meaningless since the studies are concerned with quite different aspects of exercise behaviour, measured in different ways. With the exception of one study the reported effects seem to be small; in the Tromsø study activity levels declined over six years in both the intervention and control groups.

The findings of our review do not support the use of personal or family counselling and education in the general population. Further research is needed to identify more effective interventions in exercise, diet, and smoking for use in primary prevention. Among patients who have clear evidence of cardiovascular disease, personal counselling and educational interventions seem to be beneficial, although those focusing only on exercise may not be as effective in reducing mortality from coronary heart disease as those advising on diet and smoking as well as on exercise.[3]

**Shah Ebrahim** *Professor of clinical epidemiology*
Department of Primary Care and Population Sciences, Royal Free Hospital School of Medicine, London NW3 2PF

**George Davey Smith** *Professor of clinical epidemiology*
Department of Social Medicine, University of Bristol, Bristol BS8 2PR

1 Arroll B, Beaglehole R. Does physical activity lower blood pressure: a critical review of the clinical trials. *J Clin Epidemiol* 1992;45:439-47.
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## Academia: the view from below

### Inner city scheme provides springboard for entry into academic general practice

EDITOR—Chaudhry et al outlined the uncertainties of academic careers and called for a national career structure for medical academics.[1] In general practice the need for properly conducted community based research performed by clinicians is even more pressing. Few general practitioners, however, can pursue research interests alongside clinical and administrative commitments and on call rotas. The infrastructure in which to acquire research skills, such as skills in planning research strategies, literature searches, procuring grant money, and conducting the studies, themselves barely exists. General practitioners are sometimes perceived as being at the bottom of the academic scale, as indicated by a comment in the *Independent* newspaper that perhaps it is not necessary to select students of A level standard for an ultimate career in general practice.[2] In short, there is little perception of an academic culture associated with general practice outside departments of general practice.

Nevertheless, small inroads are being made, and a research culture in general practice is being nurtured. In response to a recruitment crisis in general practice posts in the inner city of London[3 4] the Department of Health launched the London initiative zone educational incentives.[5] A component of this is the London academic training scheme. Recent graduates from vocational training are attached to a department of academic general practice in one of the London teaching hospitals. The emphasis in this training year is to provide a foundation in research methods, including the development of a research project in which the registrar is principal researcher. The registrar has seven protected sessions for research and an experienced supervisor, and computing, network, and library facilities are provided. There is an additional clinical and teaching commitment, which reflects practical academic general practice.

One great strength of this scheme, as stated by the last year's cohort of registrars, is a regular group meeting of academic registrars throughout London. This provides peer support, acts as a sounding board for research ideas, and allows skill sharing. The London academic training scheme provides a well structured, resourced, and supervised year for learning about the challenge, reward, and practicalities of a career in academic general practice coupled with continued clinical practice. Perhaps those developing academic career structures and awarding grants to researchers have something to learn from general practitioners after all.

**Sonia Saxena** *South Thames clinical research fellow*
Division of General Practice and Primary Care, St George's Hospital Medical School, London SW17 0RE

1 Chaudhry B, Winyard P, Cale C. Academia: the view from below. *BMJ* 1997;315:560-1. (6 September.)
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### Academic medicine does not fit in with motherhood

EDITOR—Chaudhry et al's editorial is the latest of many articles bemoaning poor recruitment into academic medicine.[1] To my knowledge, none of them have touched on the position of women doctors.

Of 3819 doctors who qualified in 1983, 169 were working in academic medicine in 1994.[2] Only 37 of these were women. This is only partly due to there being fewer women in absolute terms. Men were twice as likely to have entered academic medicine than were women. In no other employed category was there such a large difference in percentage terms between the sexes.

Why do women opt out of academic medicine? Are they less able? The quality of medical students at intake and qualification at this medical school makes me doubt this. What is almost totally lacking, at both training and career grades, is the opportunity to work part time. The University of Wales College of Medicine has only two part time clinical senior lecturers, and both are in my department. Child health has long been more accessible to women doctors than most specialties.

In both my pregnancies I lost out on maternity rights and pay as a consequence of losing continuity of employment when moving between NHS and academic posts during training and subsequently, despite in one case remaining within the same health authority. That was galling. Academic medicine is not an easy career to fit in with motherhood because of such institutional barriers, which must mean that many able women are excluded or discouraged. There are women professors, but how many of them are mothers? I suspect few.

I am grateful to those whose imagination and foresight gave me and a colleague an opportunity denied to many others. If there is a recruitment crisis in academic medicine surely part of the answer must be to make it a much more accessible career to women graduates.

**Elspeth Webb** *Part time senior lecturer*
Department of Child Health, University of Wales College of Medicine, Cardiff CF4 4XW

1 Chaudhry B, Winyard P, Cale C. Academia: the view from below. *BMJ* 1997;315:560-1. (6 September.)
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## Public health information on world wide web is hard to find

EDITOR—Impicciatore et al, and Wyatt in his commentary on their article, draw attention to the problem of the quality of public health information on the world wide web.[1] They do not, however, address the perhaps more serious issues of locating the information in the first place and the accessibility of this information to the general public.

The two most widely used web browsers, Netscape Navigator and Microsoft Internet Explorer, have built in links to web pages devoted to searching the Internet. Both pages contain links to over 10 search engines. Different search engines implement complex searches in different ways, and the choice of terms can alter dramatically the number of results returned. The Alta Vista search engine yields no results when the search pattern "fever management" and "child" and "parent information" is used,[2] whereas the same search performed with Yahoo yields 3563 results[3] and with Excite yields 2 632 740 results.[4] We doubt anyway that these search terms are the ones that a lay person would use when looking for information about "treating a high temperature" or "treating a fever."

If this search for information is prompted by the illness of a child, rather than academic interest, how much time is an anxious parent going to spend on the search? Even if parents restricted themselves to looking at the first 10 results from two search engines, this would still mean accessing 20 sites and trying to evaluate the quality of the information presented. The overwhelming likelihood is that the web page chosen as the definitive source of information will be of North American origin. What reaction would parents receive if they walked into a pharmacy in Britain and asked for acetaminophen?

As it stands, the world wide web is a poor source of public health information, particularly for the non-North American public, but publishing information on the web is a cheap and relatively simple way of disseminating information to a large audience and keeping that information up to date. The numbers of parents with access to the Internet from home is low (only 7% of adults in Britain have access to the Internet[5]). Efforts must be made to increase the availability of access to the Internet, as well as to increase the quality of the information, if the enormous potential of the Internet as an educational medium is to be realised.[3]

**David A Agbamu** *Consultant histopathologist*
Department of Histopathology, Arrowe Park Hospital, Upton, Wirral L49 5PE
dagbamu@pathologists.org.uk

**Ewen Sim** *Specialist registrar in histopathology*
Department of Histopathology, Hope Hospital, Salford M6 8HD

1 Impicciatore P, Pandolfini C, Casella N, Bonati M. Reliability of health information for the public on the world wide web: systematic survey of advice on managing fever in children at home [with commentary by J C Wyatt]. *BMJ* 1997;314:1875-81. (28 June.)
2 Alta Vista. URL: http://www.altavista.digital.com
3 Yahoo. URL: http://www.yahoo.com
4 Excite. URL: http://www.excite.com
5 Internet Marketing Hotlist: http://www.internet-sales.com/hot/ukover.shtml

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