# Letters

*Website:* **bmj.com**
*Email:* **letters@bmj.com**

## Not reading and signing letters you have dictated is dangerous

EDITOR—For some time I have been unhappy at the number of letters that I receive unsigned, usually from fellow consultants but more recently also from general practitioners. After the warm best wishes at the end of the letter they usually state "Dictated but sent unsigned to avoid delay"; as we both know, this is almost always totally untrue.

I know many consultants who virtually never sign their letters and, worryingly, never read them after dictating them. To add insult to injury, I recently saw a copy of a letter from a consultant to a general practitioner, unsigned and from the mistakes obviously unread, in which he had the cheek to encourage the general practitioner to send patients for assessment at the private hospital where he worked.

Last year I returned from having spent one month in the United States; there was an enormous amount of post awaiting my attention, much of it medical. The final trigger to my writing this letter was that of this large number of letters (mostly from fellow consultants but also from general practitioners) over half were unsigned and had that dishonest explanation in lieu of a signature.

Several years ago I recall reading a letter in a newspaper from a medical colleague expressing concern about this matter, and he neatly and precisely gave his view—which I share—that the practice is both discourteous and dangerous.

The discourtesy is of course regrettable, but the dangerousness is of far greater importance—not least now, when the public image of doctors is so severely battered. I can certainly confirm the dangerousness of the practice but will give just two examples. One letter from a consultant physician ended with the warm greeting "with very best wishes, yours very sincerely," and referred to a patient and her family requiring my assessment concerning her "antihypertensive treatment" whereas in fact it should have said "antidepressive treatment." Another consultant's letter referred to a patient receiving chlorpromazine when in fact she was being treated with clomipramine; had he read the letter he would have seen this error.

This matter must be aired, and to have any impact I am sure that it requires a journal of the calibre of the *BMJ* for any useful impact to be made.

**Stanley Shere** *consultant psychiatrist*
Woodley House, Nackington Road, Canterbury, Kent CT4 7AX

PS. I have just received an unsigned letter from a consultant informing me that he is treating the patient with clonazepam in a dose of 500 mg daily; I imagine that this dose would be lethal.

***Advice to authors***

*We prefer to receive all responses electronically, sent either directly to our website or to the editorial office as email or on a disk. Processing your letter will be delayed unless it arrives in an electronic form.*

*We are now posting all direct submissions to our website within 24 hours of receipt and our intention is to post all other electronic submissions there as well. All responses will be eligible for publication in the paper journal.*

*Responses should be under 400 words and relate to articles published in the preceding month. They should include ≤5 references, in the Vancouver style, including one to the BMJ article to which they relate. We welcome illustrations.*

*Please supply each author's current appointment and full address, and a phone or fax number or email address for the corresponding author. We ask authors to declare any competing interest. Please send a stamped addressed envelope if you would like to know whether your letter has been accepted or rejected.*

*Letters will be edited and may be shortened.*

**bmj.com**
**letters@bmj.com**

## Hospital league tables

### There are lies, damn lies, and hospital statistics

EDITOR—Kmietowicz's news article on hospital league tables states that the biggest predictor of death rates was the number of doctors in the hospital.[1] This conclusion was drawn by Professor Sir Brian Jarman and (in the *Sunday Times*) underpinned by data from Greenwich District Hospital.

Unfortunately for the conclusion, the data are wrong. The Department of Health's figures for Greenwich seem to be for consultants only and do not include junior doctors. This introduces an error of an order of magnitude between two and three. All hospitals' figures are likely to be distorted further by the fact that staff in unrecognised posts (often posts with strange titles such as "trust doctors") are not counted.

When such a fundamental and massive data error passes unchecked and results in false deductions, doubt is cast on the whole process. We cannot blame Dr Foster Ltd, which issued a disclaimer on data accuracy in the small print, but in my view it is quite wrong of the Department of Health to allow publication without looking closely at figures that departed so greatly from the mean.

If garbage is put in, one gets garbage out. A pity, really, because the idea is not bad.

**Andrew Bamji** *consultant rheumatologist*
Queen Mary's Hospital, Sidcup, Kent DA14 6LT
Andrew_Bamji@compuserve.com

1 Kmietowicz Z. Hospital tables "should prompt authorities to investigate." *BMJ* 2001;322:127. (20 January.)

### Analysis is flawed

EDITOR—The analysis by Dr Foster Ltd of death rates in hospital trusts is so flawed that the NHS should ignore it.[1] Standardised hospital mortality ratios are inappropriate for this exercise and difficult to interpret. They were originally public health measures intended to apply to whole area populations that are relatively static.

Patients admitted to a hospital do not constitute a predefined population; this population is arbitrary and depends heavily on admissions policy and the availability of support and other community services locally. Furthermore, standardised mortality ratios cannot be used to compare different areal units.[2]

The report does not give managers and clinical leaders any clue about how to improve quality. Should they look for rogue surgeons or killer nurses or shortcomings in clinical care? If the latter, then what's new? We were doing that anyway.[3 4] The study has served only to divert managers in "bad" hospitals into answering hysterical queries from the press; to induce self righteous complacency in "good" hospitals; and to encourage lawyers to chase after every death, expected or otherwise.

Patients don't benefit either. How does knowing a hospital's mortality index help? This index is a crude estimate of the a priori average risk of dying while in hospital. Who does it apply to? It applies only to statistically "average" patients—an esoteric concept for risk modelling enthusiasts, but of no help to individual patients, who need an estimate of their individual chances of a successful outcome.

Nor can the analysis be improved. Clever statistical manipulation of the dataset cannot get us out of the mess resulting from the inversion of the logical process of

rational epidemiological analysis. The study started with data that happened to be there; then the researchers did some sophisticated (and therefore seductively persuasive) analysis, suggested a few answers (if you torture a dataset enough it will confess to whatever you want), and then asked "What possible question is this the answer to?" It certainly does not answer the question "Which hospitals have poor quality care as judged by mortality?"

Ideally we should start with the question, refine it as far as possible, determine what data we need to answer it with an acceptable degree of validity, collect the relevant data, and then analyse them. There are other and better approaches to quality measurement.[5] Blunderbuss analysis of a dataset collected for administrative purposes is unhelpful.

**Jammi N Rao** *deputy director of public health*
Sandwell Health Authority, West Bromwich B70 9LD
jammi.rao@sandwell-ha.wmids.nhs.uk

1 Kmietowicz Z. Hospital tables "should prompt authorities to investigate." *BMJ* 2001;322:127. (20 January.)
2 Court BV, Cheng KK. Pros and cons of standardised mortality ratios. *Lancet* 1995;346:1432.
3 Department of Health. *A first class service. Quality in the new NHS.* London: DoH, 1999.
4 Department of Health. *An organisation with a memory: report of an expert group on learning from adverse events in the NHS.* London: DoH, 2000.
5 Mohammed MA, Cheng KK, Rouse A, Marshall T. Bristol, Shipman and clinical governance: Shewhart's forgotten lessons. *Lancet* 2001;357:463-6.

## Opportunity may be more important than profession in serial homicide

Editor—Kinnell suggests that the incidence of serial homicide among doctors may indicate a pathological interest in the power of life and death.[1] He notes that other professions may have fewer murderers. Our analysis of serial killers in nursing suggests an alternative interpretation.[2]

At least in relation to the murder of patients, nursing provides further examples of healthcare staff who have murdered patients in their care. In a review of 34 serial murderers in the United States, six were nurses.[3] We identified 13 convictions of nurses for the serial murder of patients up to 1997.[2] In Hickey's series, nurses had often killed for several years before they were identified.[3] Higher risk is associated with the delivery of intravenous fluids, with being in a bed out of sight of a nursing station, and with evenings or nights.[2]

When this information is combined with Kinnell's observations on Shipman and Nesset, it begins to seem plausible that all walks of life have people with the potential to murder. The key difference may be opportunity. The features associated with risk noted above suggest that access and a low chance of observation are important. The difference between nurses and doctors may be that doctors also control the means of disposal—in the case of Nesset and Shipman, they also provided the death certificate. The reason for the difference in the number of reported deaths may simply relate to the doctors' greater opportunity to remain undiscovered.

The difference between professions may be less striking than it first seems. We should not focus on one occupation. It is more important to develop safe systems. We have argued for the importance of appropriate critical incident review, which allows the identification of serial murder as well as other far commoner problems.[4] Shipman's case should not promote paranoia but should lead us to consider how best to identify problems. The techniques for further analysis of unexpected deaths are readily available, but we should not confine our attention to any one possible cause or profession.

**Cameron Stark** *consultant in public health medicine*
Highland Health Board, Assunt House, Inverness IV2 3HG
Cameron.Stark@hhb.scot.nhs.uk

**Brodie Paterson** *lecturer*
Department of Nursing, Stirling University, Stirling FK9 4LA

**Brian Kidd** *consultant psychiatrist*
Forth Valley Primary Care NHS Trust, Royal Scottish National Hospital, Larbert FK7 8AH

1 Kinnell K. Serial homicide by doctors: Shipman in perspective. *BMJ* 2000;321:1594-7. (23-30 December.)
2 Stark C, Paterson B, Henderson T, Kidd B, Godwin M. Counting the dead. *Nursing Times* 1997 93;46:34-7.
3 Hickey EW. *Serial murderers and their victims.* Pacific Grove, CA: Brooks/Cole, 1991.
4 Stark C, Sloan D. Murder in the NHS: audit critical incidents in patients at risk. *BMJ* 1994;308:477.

## "Old age" should be not be acceptable on death certificates

Editor—Dyer reports that a new agency to catch underperforming or incompetent doctors has been set up.[1] It was set up in the wake of an official report confirming that Harold Shipman, a British general practitioner serving life for killing 15 patients, was one of the most prolific serial murderers in history.

One of the diagnoses found to be unusually common on death certificates for deaths certified by Shipman was "old age." The rules in the front of books of death certificates allow this diagnosis to be used for people aged over 70. But patients aged 70-75 are not classified as elderly today. Few doctors who work with elderly people are unable to give a specific cause of death, even for patients over 95. I use "old age" only for those few patients over 85 (no more than one a year) who make an active decision to fade away and in whom disease has been excluded by physical examination, blood tests, basic radiology, and psychiatric examination.

The time has come to restrict or even abolish use of this cause of death on certificates.

**Kalman Kafetz** *consultant physician, medicine for elderly people*
Whipps Cross Hospital, London E11 1NR
kalmankafetz@hotmail.com

1 Dyer C. New agency set up to identify incompetent doctors. *BMJ* 2001;322:67. (13 January.)

## Headache after diagnostic lumbar puncture

### Using 20 gauge needle is blunderbuss technique

Editor—The results of Thomas et al's trial of atraumatic versus standard needles for diagnostic lumbar puncture[1] confirm those of others on this topic,[2–5] as well as my own personal experience. As both gauge and needle type matter,[5] I am curious as to why they did not use a finer needle, consistent with most previous efforts to reduce spinal headache.

Use of a 20 gauge needle improved the study's power by increasing the overall rate of spinal headaches versus rates in other studies,[2–5] but it can hardly be recommended as standard technique. The fact that one can kill houseflies with a shotgun does not mean that it is better to do so.

**James S Smeltzer** *consultant, maternal fetal medicine*
Wellstar Physicians' Group, Northwest Women's Care, 787 Campbell Hill Street, Marietta, GA 30060, USA
James.Smeltzer@wellstar.org

1 Thomas SR, Jamieson DRS, Muir KW. Randomised controlled trial of atraumatic versus standard needles for diagnostic lumbar puncture. *BMJ* 2000;321:986-90. (21 October.)
2 Eriksson AL, Hallen B, Lagerkranser M, Persson E, Skoldefors E. Whitacre or Quincke needles—does it really matter? *Acta Anaesthesiol Scand Suppl* 1998;113:17-20.
3 Holst D, Mollmann M, Ebel C, Hausman R, Wendt M. In vitro investigation of cerebrospinal fluid leakage after dural puncture with various spinal needles. *Anesth Analg* 1998;87:1331-5.
4 Corbey MP, Bach AB, Lech K, Frorup AM. Grading of severity of postdural puncture headache after 27-gauge Quincke and Whitacre needles. *Acta Anaesthesiol Scand* 1997;41:779-84.
5 Morewood GH. A rational approach to the cause, prevention and treatment of postdural puncture headache. *Can Med Assoc J* 1993;149:1087-93.

### Smaller is better where needles are concerned

Editor—It is unfortunate that Thomas et al devoted so much effort towards reducing the risk of headache after lumbar puncture from 50% to 28% by using an atraumatic needle[1] when a smaller needle would have had a much more beneficial effect. In a line or two they dismissed anaesthetic and radiological practice, where atraumatic needles as small as 24 gauge are routine and an incidence of headache of $<10\%$ after lumbar puncture is expected.

A large meta-analysis, which included 12 studies from 1966 to 1993, found that the incidence of headache after lumbar punc-

Odds ratios and confidence intervals in meta-analysis looking at design of spinal needles and headache after lumbar puncture[2]

| | Pooled odds ratio | 95% CI |
|---|---|---|
| Headache after lumbar puncture: | | |
| Non-cutting *v* cutting needle | 0.26 | 0.11 to 0.62 |
| Small *v* large needle | 0.18 | 0.09 to 0.36 |
| Failure: | | |
| Non-cutting *v* cutting needle | 0.52 | 0.27 to 1.01* |
| Small *v* large needle | NA | 0.67 to 2.42* |

NA=Not applicable. *Not significant.

ture was significantly reduced by both the use of smaller needles and the use of an atraumatic point (table).[2] The combined odds ratios favoured the smaller needle. As a measure of ease of use, there was no significant difference in the failure rate with type of point or needle size.

Anaesthetic trainees rapidly acquire a facility with small, atraumatic spinal needles. Cerebrospinal fluid may flow more slowly, but what other reason is there not to do diagnostic taps with a small atraumatic spinal needle?

**John R Davies** *consultant anaesthetist*
Department of Anaesthesia, Lancaster Acute Hospitals NHS Trust, Royal Lancaster Infirmary, Lancaster LA1 4RP
john.r.davies@k.bay-tr.nwest.nhs.uk

1 Thomas SR, Jamieson DRS, Muir WM. Randomised controlled trial of atraumatic versus standard needles for lumbar puncture. *BMJ* 2000;321:986-90. (21 October.)
2 Halpern S, Preston R. Postdural puncture headache and spinal needle design. Metaanalyses. *Anesthesiology* 1994;81:1376-83.

### Authors should have used smaller gauge needles

EDITOR—We wish to raise some issues about Thomas et al's trial of atraumatic versus standard needles for diagnostic lumbar puncture.[1]

Firstly, the principal objective of the study was to see if use of 20 gauge atraumatic spinal needles rather than 20 gauge standard needles reduced the incidence of headache after lumbar puncture. The authors stated that there had been only two previous studies of this type that were methodologically adequate; these were not referenced.

Secondly, the authors stated that the incidence of headache after spinal anaesthesia is typically half that after diagnostic lumbar puncture. There is no evidence that that is the case when needles of the same calibre are used and when follow up is adequate. The articles they referenced here do not make that claim.

Thirdly, the authors chose to use 20 gauge needles; this needle size is associated with a rate of headache after dural puncture of up to 36.5%.[2] The headache rate falls with decreasing needle gauge. An article in 1956 described the use of 22 gauge needles,[3] and an editorial in the *BMJ* in 1997 recommended the use of 22 gauge atraumatic needles for diagnostic lumbar puncture.[4] One of the authors' references showed that the flow rate of cerebral spinal fluid through a 22 gauge atraumatic needle was adequate. So why are they still using large gauge needles when this can lead to appreciable morbidity?

The outcome of this study was predictable. Greene first recommended the use of atraumatic needles in 1926, and Whitacre developed the needle in 1951. Many units are now using 22 gauge atraumatic needles for diagnostic lumbar puncture. Our unit recently changed to using 22 gauge Whitacre needles; this change has seen a reduction in headache after lumbar puncture from 27% to 17% and has not been associated with an increase in the average number of attempts (1.6 with the standard needle versus 1.4 with the Whitacre needle).

If the authors wish to put together a systematic randomised trial that shows a reduction in headache after diagnostic lumbar puncture[5] perhaps they should have considered using smaller gauge needles.

**David Turnbull** *lecturer in anaesthesia*
University of Sheffield, Sheffield S10 2RX
David.Turnbull@csuh.nhs.uk

**Fiona McKevitt** *neurology research fellow*
Royal Hallamshire Hospital, Sheffield S10 2JF

1 Thomas SR, Jamieson DRS, Muir KW. Randomised controlled trial of atraumatic versus standard needles for diagnostic lumbar puncture. *BMJ* 2000;321:986-90. (21 October.)
2 Kuntz KM, Kokemon E, Stevens JC, Muller P, Offord KP, Ho MM. Post lumbar puncture headache: experience in 501 consecutive procedures. *Neurology* 1992;42:1884-7.
3 Vandam LD, Dripps RD. Long term follow up of patients who received 10 098 spinal anesthetics. *JAMA* 1956;161:586-91.
4 Broadley SA, Fuller G. Lumbar puncture needn't be a headache. *BMJ* 1997;315:1324-5.
5 Sudlow C. Preventing headache after lumbar puncture: Evidence must come from randomised trials put together systematically. *BMJ* 1998;317:1589.

### Author's reply

EDITOR—These correspondents identify a reduced incidence of headache after dural puncture with smaller gauge needles. Their citations in support of this testify to the origins of such data, which are almost exclusively in anaesthesia. In neurological practice, use of needles smaller than 20-22 gauge is rare since smaller needles prolong the time taken to obtain the moderately large volumes of cerebrospinal fluid and for pressure measurement by manometry.

We certainly did not choose 20 gauge needles to increase the power of our study; we chose them because this represents routine practice. Reduction of headache after dural puncture must be balanced against the technical difficulty of the procedure, and use of very small gauge needles may be subject to the same increase in difficulty that we showed for atraumatic needles. A recent assessment under the auspices of the American Academy of Neurology emphasises the limited evidence for adoption of anaesthetic innovations for diagnostic lumbar puncture.[1]

Critics of neurologists and others performing diagnostic lumbar punctures should bear in mind that diagnostic lumbar puncture is not identical with spinal anaesthesia; extrapolation of anaesthetic data without proper appraisal may not be justified.

**Keith W Muir** *consultant neurologist*
Southern General Hospital, Glasgow G51 4TF
k.muir@clinmed.gla.ac.uk

1 Evans RW, Armon C, Frohman EM, Goodin DS. Assessment: Prevention of post-lumbar puncture headaches. Report of the therapeutics and technology assessment subcommittee of the American Academy of Neurology. *Neurology* 2000;55:909-14.

### Doctor given intrathecal methotrexate had headache when 20 gauge standard needle was used

EDITOR—In their editorial Serpell and Rawal remind us that use of small atraumatic needles can greatly reduce the incidence of spinal headache after lumbar puncture.[1 2] This is well known to anaesthetists; why not to others?

Doctors' "see one, do one, teach one" mentality is largely to blame. As a medical house officer I was taught to use 20 gauge standard (Quincke) needles for lumbar punctures, but as an anaesthetics senior house officer I soon learnt that using 25 gauge atraumatic (Whitacre) needles makes dural puncture and spinal anaesthesia simple, quick, and effective.

Two years ago I had to receive regular intrathecal methotrexate for non-Hodgkin's lymphoma. After my first session of chemotherapy, when a 20 gauge standard needle was used for the intrathecal dose, I was bedbound for a week with backache and the classic signs of a spinal headache. At my next visit I tried in vain to explain to the consultant that I had had a severe spinal headache, and I asked for smaller atraumatic needles to be used. I was told that this time I would be given intrathecal steroid to counteract my "arachnoiditis." I was sceptical but, as a disempowered patient, had to submit.

This time I was bedbound for two weeks. Thereafter I refused intrathecal methotrexate unless an anaesthetist performed the procedure. As I expected, the anaesthetics senior house officer or registrar was happy to do this. Dismayed at the routine use of large traumatic needles on the oncology day unit, the anaesthetist also taught the oncology senior house officer how to use 25 gauge Whitacre needles routinely.

As a patient with cancer, you accept that some unpleasant side effects of treatment are unavoidable. But spinal headaches often arise from a simple lack of interest in and understanding of prevention and treatment.[3 4] Incidentally, patients receiving chemotherapy cannot receive blood patching because of the risk of infection and thrombocytopenia. Many patients having chemotherapy are young and therefore at greatest risk of spinal headache. When patients are having chemotherapy every three weeks there is a small window between recovery from treatment and the onset of neutropenia when it is possible to feel well and live a relatively normal life. This precious time is lost when it is spent lying in a dark room.

The use of small atraumatic needles should become routine for all medical specialties.

**Susannah Baron** *specialist registrar in dermatology*
Leeds General Infirmary, Leeds LS1 3EX
zannerzu@onetel.net.uk

1 Serpell MG, Rawal N. Headaches after diagnostic dural punctures. *BMJ* 2000;321:973-4. (21 October.)
2 Thomas SR, Jamieson DRS, Muir KW. Randomised controlled trial of atraumatic versus standard needles for diagnostic lumbar puncture. *BMJ* 2000;321:986-90. (21 October.)
3 Serpell MG, Haldane GJ, Jamieson SR, Carson D. Prevention of headache after lumbar puncture: questionnaire survey of neurologists and neurosurgeons in United Kingdom. *BMJ* 1998;316:1709-10.
4 Sharma A. Preventing headache after lumbar puncture: most doctors are unaware of features of headache after lumbar puncture. *BMJ* 1998;317:1588.

## Strengthening governance for global health research

### Improved undergraduate and postgraduate training may raise awareness

EDITOR—Globalisation, as mentioned in the article by Lee and Mills,[1] is gaining increasing relevance to medical practice in the developed and developing world. But it is an issue barely covered in undergraduate medical curriculums. British medical students, through the Medical Students' International Network (www.medsin.org), are trying to redress this balance by increasing the awareness of humanitarian issues and inequalities in health locally and globally. Through community based projects—for example, working with local asylum seekers—students are introduced to the concept that global health begins on their doorstep. Through collaboration with international counterpart organisations, medical students can learn the true nature of the diseases affecting large proportions of humanity, which, as Lee and Mills describe, are given little global attention. The medical students of today are the global health practitioners of the future. If these practitioners are to be effective in targeting the disease burden of the world's poorest they must first be made aware of its presence during their undergraduate education.

If the partnership models of research into the developing world, such as those suggested by Costello and Zumla,[2] are to be widely implemented, the participation of doctors in the West must be encouraged through greater flexibility in their undergraduate and postgraduate training.

**Sarah Finer** *fourth year medical student*
Royal Free and University College Medical School, London WC1E 6BT
s.finer@ucl.ac.uk

1 Lee K, Mills A. Strengthening governance for global health research. *BMJ* 2000;321:775-6 (30 September.)
2 Costello A, Zumla A. Moving to research partnerships in developing countries. *BMJ* 2000;321:827-9. (30 September.)

### Ways of getting vitamin A to children in developing countries have to be improved

EDITOR—Lee and Mills in their article have raised crucial questions about drug related medical research.[1] Much research supports an agenda determined by industrialised nations. Diseases of tropical countries are often neglected. Even where appropriate diseases are investigated and results are epidemiologically unassailable, the nature of the research and the application of findings can undermine efforts of resource poor countries to deal with the primary problem.

A study that elucidates biochemistry does not always provide a solution. Researchers suggest that children under six months be given vitamin A supplements.[2] Giving capsules to children to resolve dietary deficiencies tells their families the problem is beyond their control. It creates a net capital flow out of poor countries when the issue is those countries' lack of economic power.

Roche funds Sight and Life, a humanitarian initiative to combat vitamin A deficiency with $23m (£16.1m) donated between 1986 and 1999. To further this work, the Sight and Life Research Institute was established in 1998 with the Johns Hopkins School of Public Health.

In 1999 Roche, with 40% of the vitamin market, was found to be the leader of "Vitamins Inc," a conspiracy to set vitamin prices. The head of the antitrust division of the United States Justice Department described this as the most pervasive and harmful antitrust conspiracy ever.[3] Roche, BASF, Rhone-Poulenc, and Takeda Chemical Industries were involved, paying nearly $1 billion in criminal fines, the biggest settlement in American history.[3] A Roche executive was sentenced to five months jail. Further penalties are likely in Europe and Canada. The group made billions of dollars, more than it will ever repay.[3]

These companies and three others have agreed to settle a related class action for $242m.[4] Altogether 224 companies (which provided 75% of the vitamins purchased) have withdrawn from this suit to reach larger individual settlements. One antitrust lawyer working on the case stated there was no reason to think that it would not happen again.[3] In 1976 Roche was fined DM1 098 000 (£350 000) for similar activities.[5]

Vitamin A is important to childhood development, but it is time to rethink the best ways of getting it to children in poorer countries. Supplementation takes pressure off the search for a sustainable solution. The best way for children to get vitamin A is through their food. It is not naive to imagine that this should be the foundation and end point of all research into vitamin A deficiency in children.

**Jim Black** *national health and Medical Research Council scholar*
jim.black@med.monash.edu.au
**Bebe Loff** *senior lecturer*
Department of Epidemiology and Preventive Medicine, Monash Medical School, Alfred Hospital, Prahran, VIC 3181, Australia

1 Lee K, Mills A. Strengthening governance for global health research. *BMJ* 2000;321:775-6 (30 September.)
2 Humphrey JH, Rice AL. Vitamin A supplementation for young infants. *Lancet* 2000;356:422-4.
3 Barboza D. Tearing down the façade of "Vitamins Inc." *New York Times* 1999 Oct 10.
4 Anonymous. Settlement in vitamin case is approved. *New York Times* 2000 Mar 13.
5 Adams S. *Roche versus Adams*. London: Jonathan Cape, 1984.

## Ranitidine and gastrointestinal bleeding in intensive care

### Should prophylaxis against stress ulcer be abandoned for patients in intensive care?

EDITOR—The meta-analysis of Messori et al clearly confirms the lack of usefulness of systematic prophylaxis against stress ulcer with $H_2$ receptor antagonists or sucralfate,[1] but an important issue is left unanswered by this study and by the meta-analysis of Cook et al.[2] Indeed, some critically ill patients receive prophylaxis against stress ulcers for specific reasons, including brain injury (trauma, surgery, haemorrhage), steroid treatment, and coagulation abnormalities.

Does the available literature support such prophylaxis for these patients? In other words, should intensivists prescribe stress ulcer prophylaxis for selected subgroups of patients? If the available literature does not resolve this issue, should the further trials suggested by Messori et al stratify the groups according to the patient's condition?

**Jean-Charles Preiser** *clinical director*
Clinique Reine Fabiola, 73 avenue du Centenaire, B6061 Montignies-sur-Sambre, Belgium
preiserj@ulb.ac.be

1 Messori A, Trippoli S, Vaiani M, Gorini M, Corrado A. Bleeding and pneumonia in intensive care patients given ranitidine and sucralfate for prevention of stress ulcer: meta-analysis of randomised controlled trials. *BMJ* 2000;321:1103-6. (4 November.)
2 Cook D, Reeve BK, Guyatt GH, Heyland DK, Griffith LE, Buckingham L, et al. Stress ulcer prophylaxis in critically ill patients: resolving discordant meta-analyses. *JAMA* 1996;275:308-14.

### Occult blood loss is clinically important

EDITOR—Intensive care has always been a difficult area for research.

Firstly, patients are rarely able to give informed consent, which places increased pressure on researchers and ethics committees to ensure patients are not exposed to risk. This makes placebo controlled trials difficult even when the standard treatment has no good evidence for effect.

Secondly, the patient population is variable and often small in number, which makes comparison and recruitment of sufficient numbers for statistical analysis difficult. This is borne out by the meta-analysis by Messori et al, in which meta-analysis B assessing the effectiveness of sucralfate consisted of one paper.[1]

We also have problems with the outcome used in meta-analysis A, comparing ranitidine with placebo. In four out of five papers the outcome was acute, rapid blood loss as seen by melaena, red blood through a nasogastric tube, or haemodynamic changes. These are the signs of an acutely bleeding vessel in an ulcer, as is often seen in patients presenting to accident and emergency with haematemesis. The usual pathology in intensive treatment units is different, with the presence of multiple small ulcers, stress ulcers, causing continual, low grade blood loss, breakdown of mucosal defences, and increased need for transfusion. Few of these ulcers go on to erode gastric vessels and cause dramatic blood loss. However, 75-100% of patients with critical illness develop these within three days of being admitted to intensive treatment units, even in the absence of low perfusion states.[2] We dispute the implication from the paper by Messori et al that this occult blood loss is not clinically important. A paper by Burgess et al suggested a benefit with ranitidine.[3]

The argument for prophylaxis against stress ulcer has now moved on from sucralfate with the renewed emphasis on early enteral feeding, including the instillation of enteral feed at a low rate even in

patients with high nasogastric aspirates and ileus.[4]

Finally, there is the issue of prevention of aspiration by using pharmacological agents. Endotracheal intubation or tracheostomy are not absolute guardians of the airway, and leakage of material past the balloon of the airway device is a real risk, in both intensive care and anaesthesia. Ranitidine has been shown to reduce gastric acidity during periods of high risk, and its use should still be considered in the intensive treatment unit during manipulation of the airway. Overall, it is our practice to use stress ulcer prophylaxis in all patients having intensive treatment; the agent will depend on the individual patient. We find it difficult to criticise any of our colleagues' choices in this contentious area.

**Tim Dexter** *consultant anaesthetist*
**Stephen Drage** *specialist registrar in anaesthetics*
drage@clara.net
Wycombe Hospital, Wycombe HP11 2TT

1 Messori A, Trippoli S, Vaiani M, Gorini M, Corrado A. Bleeding and pneumonia in intensive care patients given ranitidine and sucralfate for prevention of stress ulcer: meta-analysis of randomised controlled trials. *BMJ* 2000;321:1103-6. (4 November.)
2 Shuman RB, Schuster DP, Zuckerman GR. Prophylactic therapy for stress ulcer bleeding: a reappraisal. *Ann Intern Med* 1987;106:562-7.
3 Burgess P, Larson GM, Davidson P, Brown M, Metz CA. Effect of ranitidine on intragastric pH and stress related upper gastrointestinal bleeding in patients with severe head injury. *Dig Dis Sci* 1995;40:645-50.
4 Pingleton SK, Hadzima SK. Enteral alimentation and gastrointestinal bleeding in mechanically ventilated patients. *Crit Care Med* 1983;11:13-6.

## Glycated haemoglobin, diabetes, and mortality in men

### Maybe disturbance in physiological mechanisms regulating blood glucose is risk factor for cardiovascular death

EDITOR—Khaw et al contribute to the substantial literature showing an association between measures of glycaemia and subsequent morbidity and mortality.[1] They suggest that "preventive efforts need to consider not just those with established diabetes but whether it is possible to reduce the population distribution of $HbA_{1c}$ [glycated haemoglobin]." This implies that the determinant is the degree of hyperglycaemia.

In the non-diabetic population, glycated haemoglobin principally reflects the fasting blood glucose concentration, which has been shown by several groups to predict morbidity and mortality. One of these groups, the DECODE (diabetes epidemiology: collaborative analysis of diagnostic criteria in Europe) Study Group, also measured the blood glucose concentration two hours after a load and found that fasting concentrations were not additionally predictive within two hour blood glucose categories.[2]

In a cross sectional study with carotid intima media thickness as a marker of atherosclerosis, blood glucose concentrations after a load (in particular the two hour value) and incremental values (that is, above fasting) were more strongly related to intima media thickness than were fasting glucose or glycated haemoglobin concentrations.[3] Furthermore, in a population based study in Italy instability of fasting blood glucose concentrations over the years of observation was an independent predictor of cardiovascular mortality.[4]

Clearly there are several possible interpretations of these findings. One is that it is not glycaemia itself that is the risk factor but the disturbance(s) in the physiological mechanisms that regulate the blood glucose concentration. This explanation would agree with the disappointing results of hypoglycaemic treatment in type 2 diabetes, commented on by Barrett-Connor and Wingard.[5]

**R J Jarrett** *emeritus professor of clinical epidemiology, University of London*
45 Bishopsthorpe Road, London SE26 4PA

1 Khaw KT, Wareham N, Luben R, Bingham S, Oakes S, Welch A, et al. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of European prospective investigation of cancer and nutrition (EPIC-Norfolk). *BMJ* 2001;322:15-8. (6 January.)
2 DECODE Study Group. Glucose tolerance and mortality: comparison of WHO and American Diabetes Association diagnostic criteria. *Lancet* 1999;354:617-21.
3 Temelkova-Kurktschiev TS, Koehler C, Henkel E, Leonhardt W, Fuecker K, Hanefeld M. Postchallenge plasma glucose and glycemic spikes are more strongly associated with atherosclerosis than fasting glucose or HbAlc level. *Diabetes Care* 2000;23:1830-4.
4 Muggeo M, Verlato G, Boncra E, Zoppini G, Corbellini M, de Marco R. Long-term instability of fasting plasma glucose, a novel predictor of cardiovascular mortality in elderly patients with non-insulin-dependent diabetes mellitus: the Verona diabetes study. *Circulation* 1997;96:1750-4.
5 Barrett-Connor E, Wingard DL. "Normal" blood glucose and coronary risk. *BMJ* 2001;322:5-6. (6 January.)

### Analytical information is required for generalisation of data

EDITOR—Analytical information is required before data can be generalised. Khaw et al's data suggesting that the relation between cardiovascular disease and glycaemia is a continuum extending throughout the non-diabetic population are fascinating.[1] Unfortunately, as the paper stands, the results are not generalisable since neither the methodology for assessing glycated haemoglobin nor calibration data have been included.

Professional organisations throughout the United Kingdom agree that percentage glycated haemoglobin concentration should be harmonised in relation to a common standard[2] and that, in the absence of a primary calibrant, this standard should be that used in the diabetes control and complications trial.[3] Without this information, the results of the present study cannot be compared against others; it is inappropriate to discuss "a threshold commonly accepted for diagnosis of diabetes."

With an ion exchange method (HA-8140, Menarini Diagnostics) widely used in Europe the upper limit of the reference range observed in non-diabetic subjects has been reported as 5.1%[4]—equivalent to 5.9% after standardisation as in the diabetes control and complications trial. If either of these cut off values was applicable to the present study a large (although vastly different) number of subjects in the study could not be considered to have normal glycaemic control. This could be explicable in terms of the age range of the cohort (45-79 years) and the association of increased age and decreased glucose tolerance.[5]

The suggestion that a large percentage of the cohort might have abnormal glycaemic control would not be surprising: Harris et al, for example, showed that 27.1% and 42.9% of the male population of the United States aged 55-64 and 65-74 respectively had either impaired glucose tolerance or diabetes.[5] The present data could therefore be explained on the basis that many patients with impaired glucose tolerance were included in the upper two thirds of the non-diabetic population and that these patients have an increased rate of cardiovascular disease.

The authors should be encouraged to provide at least a non-diabetic reference range for their assay and preferably some data relating their results to a method aligned to the diabetes control and complications trial. This study can then be compared with other work in this area.

Finally, the authors state that "$HbA_{1c}$ [glycated haemoglobin] may provide a practical screening tool for diabetes or impaired glucose tolerance." Although this statement could be true, the authors do not provide any evidence to support it. The World Health Organization has categorically stated that glycated haemoglobin concentration should not be used to establish the diagnosis of diabetes, and this has been reiterated by Diabetes UK.

**Edmund Lamb** *consultant clinical biochemist*
East Kent Hospitals NHS Trust, Kent and Canterbury Hospitals, Canterbury CT1 3NG
edmund.lamb@kch-tr.sthames.nhs.uk

1 Khaw KT, Wareham N, Luben R, Bingham S, Oakes S, Welch A, et al. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of European prospective investigation of cancer and nutrition (EPIC-Norfolk). *BMJ* 2001;322:15-8. (6 January.)
2 Marshall SM, Barth JH. Standardization of HbA1c measurements—a consensus statement. *Diabetic Med* 2000;17:5-6.
3 Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 1993;329:977-86.
4 John WG, Braconnier F, Miedema K, Aulesa C, Piras G. Evaluation of the Menarini-Arkray HA 8140 hemoglobin A1c analyzer. *Clin Chem* 1997;43:968-75.
5 Harris MI, Hadden WC, Knowler WC, Bennett PH. Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in US population aged 20-74 yr. *Diabetes* 1987;36:523-34.

### Authors' reply

EDITOR—Jarrett suggests that it is not glycaemia itself that is a risk factor but disturbances in the physiological mechanisms that regulate the blood glucose concentration. The point of our analysis was to examine the predictive value of glycated haemoglobin measures for the risk of death and the shape of the risk curve. In this cohort, glycated haemoglobin predicted risk of death continuously across the whole population distribution.

Most of the excess events associated with raised glycated haemoglobin concentrations occurred at values below those that would be used to define diabetes. Indeed, in the

diabetes control and complications trial there was no glycaemic threshold for the development of long term complications; as the glycated haemoglobin concentration was reduced below 8% there were continuing relative reductions in the risk of complications such as microalbuminuria and retinopathy.[1] In the United Kingdom prospective diabetes study, strict control of blood glucose reduced microvascular complications significantly by 25%, but the study had inadequate power to detect a smaller difference (10%) in mortality related to diabetes.[2]

In response to Lamb, we would point out that full details of the assays, though omitted from our paper in the printed journal, are given in the longer version of the paper on the *BMJ* website (www.bmj.com/cgi/content/full/322/7277/15). As stated in that longer paper, the glycated haemoglobin measurements were made in a single laboratory using high performance liquid chromatography on a Biorad Diomat. This is a diabetes control and complications trial standardised method.

The World Health Organization and Diabetes UK may well have categorically stated that glycated haemoglobin should not be used to establish the diagnosis of diabetes. We would hope, though, that such consensus statements are based on evidence rather than opinion and may be reconsidered in the light of new evidence from studies such as ours about the nature of the relation between glycated haemoglobin concentration and risk of future events.

Of course we accept that there may be other reasons not to use measurements of glycated haemoglobin, such as the problems of standardisation between methods and the limited availability of the test in less developed parts of the world. However, both these technical issues could be rectified. There are no fundamental obstacles to using glycated haemoglobin concentration as a predictor of macrovascular complications and risk of death associated with hyperglycaemia.

**Kay-Tee Khaw** *professor of clinical gerontology*
kk101@medschl.cam.ac.uk

**Nick Wareham** *Medical Research Council clinician scientist*
Institute of Public Health, University of Cambridge, Addenbrooke's Hospital, Cambridge CB2 2QQ

1 Diabetes Control and Complications Trial Research Group. The absence of a glycemic threshold for the development of long-term complications: the perspective of the diabetes control and complications trial. *Diabetes* 1996;45:1289-98.
2 UK Prospective Diabetes Study Group. Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 1998;352:837-53.

### Medicine is now using diagnostic criteria rather than reference ranges

Editor—Khaw et al's study showed that glycated haemoglobin concentration is associated with mortality in men without diabetes.[1] It is another illustration of the general rule for a continuous variable: subjects with characteristics outside the normal range have an increased incidence of acute and chronic conditions and higher mortality, and subjects with normal characteristics do not have equal mortality—in most cases the lowest mortality is not close to mean values.[2]

This rule is true for many variables, from height and weight to glycated haemoglobin concentration. It is true for population samples and for very healthy subjects.[3] For most diseases the shape of the curve describing incidence against the variable is individual to that disease. For example, the curve of cancer mortality versus blood cholesterol concentration is different from that of cardiovascular mortality versus blood cholesterol concentration.[4] Every curve can be explained if we try hard enough.

Medicine is drifting from using reference ranges to using diagnostic criteria. This means that the diagnostic threshold is based not on the distribution of values in healthy subjects but on the trade-off between getting false negative and false positive results.[5] Current diagnostic criteria for diabetes trade off the probability of complications. Already there is a tendency for people with blood glucose concentrations in the normal range (but with impaired tolerance) to receive interventions; now comes the proposal to involve the whole population.

The situation is similar for the diagnosis of obesity: interventions are now given to people with a body weight indicating not obesity but overweight, and the population is under pressure to reduce weight. Khaw et al are wrong in stating that "it is uncertain whether the relation between blood glucose concentration and … diseases has a threshold or is a continuum." A threshold makes for a simplified decision rule; a blurred border between healthy subjects and people who are sick or at risk makes simple explanation hard, but it is reality.

Medicine is historically limited to more or less definite groups of people (those who are "diseased"). The alternative is a medicalisation of traits, habits, and risks. This expansion to the whole population arises because only a small proportion of subjects at risk is in the group above the diagnostic threshold; it is a simple consequence of the low effectiveness of the diagnosis. Indiscriminate interventions are usually inefficient.

**Vasiliy Vlassov** *professor*
Saratov State Medical University, PO Box 1528, Saratov, 410601 Russia
vvvla@sgu.ru

1 Khaw KT, Wareham N, Luben R, Bingham S, Oakes S, Welch A, et al. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of European prospective investigation of cancer and nutrition (EPIC-Norfolk). *BMJ* 2001;322:15-8. (6 January.)
2 Vlassov VV. *Reaction of the organism to external stimuli: general patterns of reaction's development and related methodological problems of research.* Irkutsk: Irkutsk University, 1994:343. (In Russian.)
3 Vlassov VV. Age changes of the haemoglobin and erythrocyte concentrations in pilots. *Aviakosmich Ecologich Med* 1992; 4:19-23. (In Russian.)
4 Schuit AJ, Van Dijk CEMJ, Dekker JM, Schouten EG, Kok FJ. Inverse association between serum total cholesterol and cancer mortality in Dutch civil servants. *Am J Epidemiol* 1993;137:966-76.
5 Remein QR, Wilkerson HL. The efficiency of screening tests for diabetes. *J Chronic Dis* 1961;13:6-21.

## Catheterisation in elderly women is no "easy" option

Editor—In their review of the management of urinary incontinence in women, Thakar and Stanton say that patients with chronic urinary incontinence, particularly elderly patients, may be easier to manage with a permanent indwelling catheter.[1] As one of the "geriatric giants" described by Isaacs in 1992, urinary incontinence is a serious problem in older patients, with a prevalence of one in five to 10 in women older than 65 years, rising to two in three in residents of nursing homes.[2 3] These patients deserve the same attention and assessment as their younger counterparts, even if they have medical conditions that preclude appropriate surgical treatment, or cognitive impairment that prevents them from complying with pelvic floor exercises or bladder retraining.

Simple measures can often noticeably improve symptoms. These might include switching to decaffeinated tea or coffee, excluding urinary tract infections or causes of polyuria such as diabetes mellitus or hypercalcaemia, reviewing drug treatment, including the use of diuretics and drugs that predispose to urinary retention, and practical measures to ensure that those with physical disabilities have easy access to toileting facilities. Cognitively impaired patients may benefit from timed, prompted voiding. Liaison with the continence nurse adviser may help with the provision of aids that make management of continence acceptable to carers.

Long term urinary catheterisation causes inevitable bacteriuria, which is difficult to eradicate and increases the risk of pyelonephritis, bacteraemia, and sepsis. It is an invasive procedure with an appreciable morbidity and mortality,[4] a fact that should be taken into consideration before applying it to frail elderly patients. In addition, in a condition with such a high prevalence, widespread use of long term urinary catheters for incontinence has important cost implications, with the cost of medical consequences of catheterisation outweighing the savings in continence devices.[5] Long term urinary catheterisation should be considered only in women with urinary retention for whom intermittent self catheterisation is not appropriate, and as a last resort in patients in patients with excoriated skin or pressure sores in whom other measures have failed.

**Virginia Aylett** *specialist registrar*
gin_aylett@yahoo.com

**Olwyn Lynch** *specialist registrar*
Department of Medicine for the Elderly, St James's University Hospital, Leeds LS7 9TF

1 Thakar R, Stanton S. Management of urinary incontinence in women. *BMJ* 2000;321:1326-31. (25 November.)
2 Isaacs B. Incontinence. In: *The challenge of geriatric medicine.* Oxford: Oxford University Press, 1992:101-22.
3 Department of Health. *Good practice in continence services.* London: DoH, 2000.
4 Platt R, Polk BF, Murdock B, Rosner B. Mortality associated with nosocomial urinary-tract infection. *N Engl J Med* 1982;307:637-42.
5 Ouslander JG, Kane RL. The costs of urinary incontinence in nursing homes. *Med Care* 1984;22:69-79.

## Weekend binge drinking may be linked to Monday peaks in cardiovascular deaths

EDITOR—As Martyn notes in an editorial, the Monday peak in cardiovascular mortality and morbidity has been documented in various settings.[1] We would like to develop further the notion that alcohol consumption is at least partly responsible. In countries with known weekend binge drinking the Monday peak is pronounced and is accompanied by slight increases in mortality on Saturdays and Sundays. This has been shown in countries of the former Soviet Union[2] and in Scotland.[3]

The epidemiological evidence on high alcohol consumption and cardiovascular mortality supports this hypothesis. Studies that look at the pattern of drinking, either directly or indirectly, have consistently found an increased risk of cardiovascular death (particularly sudden death) with binge drinking.[4]

Physiological studies have also shown that the effects of regular moderate drinking versus binge drinking differ greatly.[5] In binge drinkers, cardioprotective changes in high density lipoproteins are not seen and adverse changes in low density lipoproteins are acquired. Binge drinking seems capable of predisposing the heart to arrhythmia, both by reducing the threshold for ventricular fibrillation and by causing scarring of the myocardium.

The myocardium may be especially sensitive during withdrawal, as will occur after weekend binges. In addition, irregular drinking is associated with an increased risk of thrombosis, which is most likely to occur after heavy drinking stops. These physiological mechanisms may explain the observed increase in cardiovascular events during the weekend and on Mondays.

We hypothesise that alcohol, particularly when drunk in binges, acts as a catalyst on acute ischaemic heart diseases, possibly by being synergetic to other triggering factors.

**Laurent Chenet** *lecturer*
European Centre on Health of Societies in Transition, London School of Hygiene and Tropical Medicine, London WC1E 7HT
laurent.chenet@lshtm.ac.uk

**Annie Britton** *lecturer*
Health Promotion Research Unit, London School of Hygiene and Tropical Medicine

1 Martyn C. Hebdomadal rhythms of the heart. *BMJ* 2000;321:1542-3. (23-30 December.)
2 Chenet L, McKee M, Leon DL, Shkolnikov VM, Vassin S. Alcohol and cardiovascular mortality in Moscow: new evidence of a causal association. *J Epidemiol Community Health* 1998;52:772-4.
3 Evans C, Chalmers J, Capewell S, Redpath A, Finlayson A, Boyd J, et al. "I don't like Mondays"—day of the week of coronary heart disease deaths in Scotland: study of routinely collected data. *BMJ* 2000;320:218-9.
4 Britton A, McKee M. The relationship between alcohol and cardiovascular disease in eastern Europe: explaining the paradox. *J Epidemiol Community Health* 2000;54:328-32.
5 McKee M, Britton A. The positive relationship between alcohol and heart disease in Eastern Europe: potential physiological mechanisms. *J R Soc Med* 1998;91:402-7.

## Fillers can transform people's lives

EDITOR—Not only can fillers be educational, as Singh points out,[1] but they have the power to transform people's lives.

In his filler, entitled "Thank you for ending 40 years of misery," Lakhani described his memorable patient, an 85 year old woman whose life had been made "sheer hell" by diarrhoea up to six times a day since she had had a vagotomy and gastroenterostomy in 1957, which had proved resistant to treatment with a fat free diet, pancreatic supplements, antibiotics, and codeine.[2] Suspecting this might be diarrhoea after vagotomy, he treated her with the bile acid sequestrant cholestyramine, which promptly restored her bowel function to normal.

After describing this case in my weekly medical column in the *Sunday Telegraph* nine readers reported a similar dramatic response after the initiation of cholestyramine treatment. Two readers said that they had had 26 years of "wind and fluid" and 31 years of "severe diarrhoea" after vagotomy and two three years of "severe painful diarrhoea" and diarrhoea for "several years" after cholecystectomy. Two readers with irritable bowel syndrome reported having had "many years" of severe diarrhoea and having had diarrhoea "six times a day." Three readers with diverticulitis said that they had had diarrhoea "every few days and without warning," colicky pains and diarrhoea "as soon as I eat anything," and "very bad diarrhoea."

Cholestyramine is a well recognised (if probably underused) treatment for chronic diarrhoea due to bile acid malabsorption as may occur after abdominal surgery or in association with inflammatory bowel disease. Its value in chronic idiopathic diarrhoea—often attributed, as in this series, to irritable bowel syndrome or diverticulitis—is more contentious, with conflicting evidence as to the contributory role or bile acid malabsorption (or increased endogenous bile acid secretion) and responsiveness to bile acid sequestrants.[3 4]

It might be more useful, in the absence of a clear understanding of the pathophysiological mechanisms involved, to take an empirical view. Cholestyramine is prescribed as an antidiarrhoeal agent in several conditions, while 40% of those taking the drug in a primary prevention trial for coronary heart disease reported constipation as a side effect.[5]

Before patients with chronic diarrhoea are diagnosed as having diverticulitis or irritable bowel syndrome, they should first be given a therapeutic trial of cholestyramine. Those experiencing a marked symptomatic improvement could then be described as having cholestyramine responsive diarrhoea. This would have the merit, certainly for patients, of making doctors more aware of this remarkably effective, if poorly understood, remedy for a most debilitating condition.

**James Le Fanu** *retainer general practioner*
Mawbey Brough Health Centre, London SW8 2UD

1 Singh S. Are fillers educational? *BMJ* 2000;321:1277. (18 November.)
2 Lakhani M. Thank you for ending 40 years of misery. *BMJ* 1999;319:86.
3 Smith MJ, Cherian P, Raju GS, Dawson BF, Mahon S, Bardhan KD. Bile acid malabsorption in persistent diarrhoea. *J R Coll Physicians Lond* 2000;34:448-512.
4 Schiller LR, Bilhartz LE, Santa Ana CA, Fordtran JS. Comparison of endogenous and radio labelled bile acid excretion in patients with ideopathic chronic diarrhoea. *Gastroenterology* 1990;98:1036-143.
5 Lipid Research Clinics Coronary Primary Prevention Trial results. I. Reduction in incidence of coronary heart disease. *JAMA* 1984;251:351-73.

## Invalid health information is potentially lethal

EDITOR—A picture of the cover of Benjamin Spock's record breaking bestseller *Baby and Child Care* was used to illustrate a point Ferguson made in the section of his article subtitled, "From Dr Spock to drkoop.com."[1] We are told that a Harvard pioneer in cybermedicine—Warner Slack—has compared the rapid growth of online health resources to the seismic impact of the publication of Spock's book, which, he suggested, had rapidly made it clear that well informed parents could take much better care of their kids.

By coincidence, I had used the same picture of the cover of Spock's book in the previous week at the Cochrane Colloquium in Cape Town, but to make a different point. In the edition of *Baby and Child Care* that I bought as a recent medical graduate in the mid-1960s, I had marked a passage which read: "There are two disadvantages to a baby's sleeping on his back. If he vomits, he's more likely to choke on the vomitus. Also he tends to keep his head turned towards the same side … this may flatten the side of his head … I think it is preferable to accustom a baby to sleeping on his stomach from the start." No doubt like millions of his other readers, I passed on and acted on this apparently rational and authoritative advice.

We now know that the advice promulgated so successfully in Spock's book led to thousands, if not tens of thousands, of avoidable cot deaths. This should be a sobering warning to those who exploit the internet to promulgate health advice without ensuring that reliable empirical research evidence has shown that their prescriptions and proscriptions are more likely to help than to harm other people.

**Iain Chalmers** *director*
UK Cochrane Centre, Oxford OX2 7LG
ichalmers@cochrane.co.uk

1 Ferguson T. Online patient-helpers and physicians working together: a new partnership for high quality health care. *BMJ* 2000;321:1129-32. (4 November.)