Letters

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Politics surrounding last winter's flu crisis

NHS's fundamental problems must be solved

EDITOR—The NHS Confederation and the BMA have argued that the recent problems in dealing with emergency demand conceal more fundamental problems: the NHS has too little capacity run at too high a rate of use.[1]

Running hospitals at the current rates of occupancy is not efficient. Bagust et al show that hospital occupancy of more than 85% will guarantee periodic bed crises and the cancellation of hospital admissions.[2] NHS occupancy information excludes patients who stay less than one day and therefore underestimates the true picture. In critical care a growing body of evidence suggests that there is insufficient spare capacity in the system.

Many NHS staff have a ridiculous workload. They are required to cope with the chaotic results of high levels of admissions and occupancy and, in particular, the problem of patients on outlying wards. There is no time for staff to muster new resources, and nurses have had the parts of their work that allowed recuperation devolved to other staff. There is growing evidence that these problems affect outcomes and lead to staff seeking employment elsewhere, further exacerbating the pressure on those staff who remain.

These problems have arisen because for 20 years the NHS has sought to do more work for less money. The measures of efficiency used by the government have in general paid little attention to the quality of the result, although there has been more of a move in this direction recently.[3] The tight finances of the NHS have meant that improvements in efficiency can lead to a dilution of quality services, overstretching staff, being slow to adopt new medicines and technology, and failing to invest in major change that could improve the quality of what we do. Unfortunately, the latest planning guidance issued by the NHS Executive requires a further 3% increase in efficiency, which could make the problems worse.[4]

The absence of good baseline data means that it is difficult to prove that this will cause problems, and there is a danger of appearing to complain without evidence. We need to look at the way we measure performance and relate this more closely to broader measures than simple efficiency.

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The NHS Confederation is a membership organisation that represents 95% of NHS trusts and health authorities.

1 Woodman R. Doctors and politicians clash over size of flu problem. *BMJ* 2000;320:138. (15 January.)
2 Bagust A, Place M, Posnett JW. Dynamics of bed use in accommodating emergency admissions: stochastic simulation model. *BMJ* 1999;319:155-8.
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Spin doctoring the problem

EDITOR—In an otherwise excellent editorial on the contradiction in government policy about acute beds in the NHS, Pollock and Dunnigan describe January's flu epidemic as making life tough for British health ministers.[1]

Although the politicians wished us to believe otherwise, the number of cases of flu reported in England and Wales in January barely reached half of the 400 cases per 100 000 population defined as epidemic by public health doctors, and in reality seemed to be little more than the normal rise in cases associated with the winter.[2] If this year's mild winter has brought the NHS to its knees a real epidemic would surely stretch it well beyond breaking point. It is a pity to see both the authors and the *BMJ* perpetuating disinformation put about by the government's spin doctors to minimise the plight of acute hospitals.

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1 Pollock AM, Dunnigan MG. Beds in the NHS. *BMJ* 2000; 320:461-2. (19 February.)
2 Woodman R. Doctors and politicians clash over size of flu problem. *BMJ* 2000;320:138. (15 January.)

Elderly people deserve more than a free television licence

EDITOR—Last winter hospitals throughout the United Kingdom struggled hard to cope with elderly patients with respiratory infections.[1] A high winter mortality in the United Kingdom is documented in the literature. The mean excess winter death index (defined as the percentage excess deaths in the four winter months, December to March, compared with the average in the preceding and following four months) for eight winters (1976-84) was exceptionally high in England and Wales at 21 and Scotland at 20, compared with Canada (7), Finland (8), Germany (8), and the United States (9)[2].

During the 1996-7 winter the number of excess deaths in England and Wales was nearly 50 000, with 48% of the deaths due to respiratory infection and 36% to circulatory diseases. In the 1989-90 winter the number of deaths due to influenza alone was estimated to be over 25 000. The excess winter deaths were almost exclusively among the elderly population.[3]

The high excess winter death index in the United Kingdom is related to three potentially modifiable factors.[2 4]

- The World Health Organization recommends a minimum indoor temperature of 18°C, but 2-3°C warmer for rooms occupied by sedentary people.[5] Evidence suggests a lower standard of home heating in the United Kingdom,[2] since many elderly people live in poor housing conditions and cannot afford the heating bill. Finland has invested vastly on housing improvement, including heating
- People in the United Kingdom often make brief excursions into the cold outdoors environment.[2] Efficient and free home delivery services for elderly people may help to reduce this risk

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● More than half of Europe adopted influenza vaccination policies based on age and covering people aged ⩾65. In the United Kingdom, however, vaccination is offered to people with chronic diseases and those living in nursing and residential homes. This leaves 5.6 million people aged ⩾65 uncovered.[4]

The winter puts more pressure on hospital staff and waiting lists every year. Keeping elderly people warm and vaccinated at home during the winter helps to reduce the burden and the consequent mortality. The cost of providing a free television licence for people over 75 might be more beneficially invested in these preventive measures.

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Cerebral dysfunction after water pollution incident in Camelford

Results were biased by self selection of cases

Editor—Altmann et al's results on a group of litigants were published almost 10 years after the water pollution incident in Camelford.[1] Although they acknowledge competing interests, they overlook the main problem—the bias inherent in self selection of cases.

The cases may have already had unexplained symptoms and cognitive problems, the incident serving to focus attention on a possible cause. The results show significant impairment in neuropsychological and neurophysiological tests among the cases, which the authors argue must be the result of prolonged toxicity to acute exposure to aluminium in drinking water. Neuropsychological tests are assumed to be objective, automated, computerised, and quantitative, but they do require the conscious effort of subjects. Those complaining of poor memory and concentration are given a test that requires both, so performance cannot be taken at face value. Subjects are not carrying out a deliberate deception, but their performance like everyone else's is influenced by the context. Similarly, the choice of relatives as controls is unfortunate. Out of loyalty, they will tend to give more than their best, thus widening the gulf in performance.

Sensory evoked potentials are less liable to such effects. The authors report an increased latency in "flash minus pattern" evoked potential (EP), an index of cognitive impairment in patients with Alzheimer's disease and aluminium related dialysis dementia. Their data are inadequate for evaluating this finding since neither the raw latencies for the controls nor the waveforms for the cases are given. The results hang on the difference between patterned visual evoked potentials (VEPs) and flash EPs. The VEP is also known as the P100 since it occurs reliably 100 ms after the onset of the stimulus. The response to flash is characteristically longer, by around 20 ms in normal subjects, more in those with brain disorders.[2 3] The table compares results in four groups.[1–4] It shows that the latency of the flash EP is not prolonged in the Camelford cases but that the increased difference between the two measures is accounted for by the apparent quickening of the patterned VEP. The most parsimonious explanation for this unphysiological result is a calibration error and wandering baseline.

The reopening of the Camelford case is regrettable as the people concerned may worry anew about their health. Wessely and I recommended that incidents such as these should trigger a rapid response by public health professionals to gather complete data before litigation, media attention, and local fears distort health perception.[5] It seems that such lessons have still to be learnt.

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Study has several methodological errors

Editor—Altmann et al conclude that some people who lived in Camelford when water supplies were contaminated with aluminium sulphate have suffered "considerable damage to cerebral function."[1] This conclusion is wrong for several reasons.

The design of the study is flawed in selecting as cases people who believed that they had suffered damage and who would therefore be more prone to show errors in psychological tests which they knew were designed to show abnormalities. The authors then used as controls siblings living in a different part of the United Kingdom and presumably unaffected by any similar poisonings. Why did they not use siblings who had been exposed but did not complain of any cognitive difficulties? Fifteen sibling pairs is a small sample size and means that significant differences between the groups are more likely to be found, but this possible bias is not taken into account by the authors.

The authors emphasise the finding of a significant difference between the cases and controls in the absolute differences between pattern and flash evoked visual responses. However, they do not give any absolute values for the latencies for either method of stimulation. Thus whether the increase in difference between the two stimuli was because of a reduction in latency with one form of stimulation or an increase in the other is uncertain. Interpreting a prolongation in latency is difficult and was not attempted. If aluminium causes direct neuronal death, why would this lead to slowing of conduction along the axons of the optic nerves? Would not a reduction in the amplitude of the response be more likely? They raise the contentious link between Alzheimer's disease and aluminium but do not mention that abnormalities in visual evoked response are not observed in Alzheimer's disease.

There is scant comment on the concentration of aluminium in current tap water samples from the area described as high and no indication of aluminium concentrations in the controls or their tap water.

Latencies (in ms) for visual evoked potentials (VEPs) in published studies. Values are means (SE) in studies by Altmann et al and means (SD) in the others

Reference	Patterned EP latency	Flash EP latency	Difference
Altmann et al[1] (Camelford):			
Cases (n=28)	93.5 (0.9)	119.8 (2.6)	27.3 (1.64)
Controls (n=42)	NK	NK	18.6 (1.47)
Altmann et al[4] (dialysis):			
Cases (n=10)	101.8 (3.2)	133.4 (2.4)	31.6 (4.3)
Controls (n=22)	NK	NK	19.4 (2.4)
Philpot et al[2]:			
Elderly normal subjects (n=13)	106 (11)	128 (11)	21 (15)
Elderly subjects with mild memory impairment (n=12)	105 (5)	129 (10)	24 (11)
Elderly subjects with dementia (n=12)	110 (6)	137 (10)	26 (8)
Sloan et al[3]:			
Elderly normal subjects (n=40)	100.5 (7.5)	140.5 (13.8)	40.0*
Elderly subjects with major depression (n=32)	104.3 (11.7)	153.0 (15.8)	48.7*
Elderly subjects with Alzheimer's dementia (n=30)	101.7 (14.7)	152.5 (19.1)	50.8*

NK=not known.
*SD not given.

The authors also dismiss the effects of underlying anxiety as a cause of the observed phenomena on the basis of a low SCL90 score, but almost 35% of their study population did not attend for testing.

This paper is little more than a series of observations in a group of people. Evidence of considerable cerebral damage is not evident from the abnormalities on testing, there is no correlation with the level of day to day impairment, and bias abounds in the methods. It would be wrong to conclude that this study substantiates the claims that these people's symptoms are due to direct toxic effects of aluminium poisoning.

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Study may prolong the agony

EDITOR—Altmann et al conclude that poisoning with aluminium sulphate in Camelford 10 years ago "probably led to long term cerebral impairment."[1] Media attention immediately after this paper was published led to speculation about damage to children and unborn fetuses at the time and possibly a further round of litigation. The results of this paper were, however, obtained under the instruction of plaintiffs' solicitors in the earlier round of litigation, which ended in 1994 with small out of court settlements (average £2000), and they are not new evidence.

Some people exposed to polluted water in Cornwall in July 1988 may have developed brain damage,[2] but the absence of physiological measures of absorption of aluminium and other toxins at the time of the accident made it impossible for a definite conclusion to be reached. With regard to persisting complaints attributed to the accident, the paper by Altmann et al is unhelpful and its conclusion unwarranted. Methodological flaws include use of a highly self selected group of litigants and sibling controls, which is a source of bias, as described by David and Esmonde (above) and by Fahy in bmj.com (bmj.com/cgi/eletters/319/7213/807/DC1#EL3).

Altmann et al persist in using the unrevised normative data for the national adult reading test, which is used to estimate premorbid ability. Revised normal values for this test were published in 1991 and are used throughout the United Kingdom; by using the outdated version Altmann et al overestimate IQ in the patient group, increasing the likelihood of finding a decrement in tests of current ability.

They must also be criticised for not discussing in depth the two key papers on such incidents.[3,4] Neither do they defend the use of evoked potentials, given the criticisms levelled specifically at their work in the second report of the Clayton committee.[5]

Finally, they do not acknowledge or explain the absence of effect in the only study on the entire exposed population (of children) who were compared with an age matched non-exposed group of Cornish children in routinely administered psychometric tests of educational attainment given before and after the accident. No evidence for impairment in the exposed group was found—implying that severe brain damage was unlikely to have occurred.[6]

I agree with David (previous page) that it is regrettable that psychological wounds may be reopened in North Cornwall at such a late stage and seemingly without good reason. I hope, however, that plans have been made for early systematic assessment of exposure and any physical and psychological sequelae in the event of future accidents.

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Inappropriate study, inappropriate conclusions

EDITOR—We believe that the design and experimental technique limit the conclusions of Altmann et al about cerebral dysfunction in the Camelford incident of 1989.[1] In addition, their study does not report on the subsequent developments to respond to or prevent a similar incident.

In 1988 the domestic water supply of 20 000 people in the Lowermoor area was contaminated with aluminium sulphate. Much of the information on the adverse health effects arising from the exposure was obtained from self reported questionnaires that may not have been completed by a representative sample of the local population.[2] In the study of Altmann et al the 55 cases were also a self selected group. Differences in psychomotor tests and visually evoked potentials (VEP) were found when cases were compared with unmatched controls and the cases' siblings, but VEP were measured in only half of the cases. In addition to the issues raised by David, Esmonde, and McMillan in the previous letters, differences in VEP have also been observed in major depression[3] and the study group was not formally evaluated psychologically.

A timely response to assess the impact of any chemical incident on a population is essential. Since 1996 health authorities have been required to have contracts with chemical incident service provider units. The Chemical Incident Response Service is one of five providers in the United Kingdom and has contracts with all health authorities in six of the eight English regions.

Incident identification and management has pointed to the need for early response with appropriate collection of biological and environmental samples, usually within hours of exposure. These issues are discussed by public health physicians attending training courses and supported by relevant guidance material, including a recent handbook.[4] With continuing surveillance to monitor response effectiveness, methods are now in place to prevent the delays in investigation identified in the Camelford incident. All water utilities have also responded to the lessons learnt from Camelford. A report was written after the Camelford incident by an independent non-executive director of the South West Water Authority which made recommendations to prevent a similar incident occurring in the future. These recommendations were largely concerned with the control of access to water treatment plant and control of chemical deliveries.[5]

The government sent copies of the report to the chief executives of all water authorities, who were then obliged to inform the government of the management and operating procedures they had put in place to implement the recommendations. In addition, water utilities are now required to inform health authorities immediately about any customer health concerns or operational incidents.

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❦The study authors were asked to reply but did not do so in time for publication here.

Inadvertent dural puncture

Avoiding unintentional puncture is a primary goal of obstetric anaesthetists

EDITOR—While reading Weir's account of her experience of dural puncture I was trying to determine what could have been done to improve the situation.[1] Severe postdural puncture headache is fortunately uncommon and represents one end of the range of an event that occurs in $<2\%$ of

parturients having a regional technique. Avoiding this complication completely would mean denying women the most effective, reliable means of providing analgesia for labour and delivery.[2]

The administration of three failed epidurals and two spinal blocks suggests that this patient's lumbar spine presented technical difficulty to the anaesthetist(s) concerned. Early onset of postdural puncture headache is unusual as most such headaches do not begin for 24 hours; headache during caesarean section is commonly related to fatigue, stress, or dehydration.

Once postdural puncture headache is diagnosed, early administration of a blood patch may be less effective than waiting 24 hours.[3] Giving opioids to supplement analgesia, followed by conversion to general anaesthesia, was a perfectly reasonable response by the anaesthetist to Weir's distress at delivery. Two blood patches being administered within four days of delivery also suggests that the anaesthetist believed that Weir was experiencing a severe postdural puncture headache, and this treatment was both timely and appropriate. Indeed, the second blood patch provided enough resolution of the headache to allow Weir to stand and "let life and light back in."

Backache after labour and delivery occurs in half of parturients irrespective of whether they received regional anaesthesia. Tenderness at the site of the epidural usually resolves within two weeks. There seemed to be no delay in diagnosing a long term, low grade cerebrospinal fluid leak; this can only be determined if symptoms persist. Surgical repair of the torn dura may occasionally be indicated, although Weir does not mention this.

Avoiding unintentional dural puncture is one of the obstetric anaesthetist's primary goals whenever he or she performs an epidural technique. The institution where I work, and those I have worked in in the United Kingdom, takes postdural puncture headaches very seriously. I was saddened to read that Weir believed that her headache was dealt with with flippancy, arrogance, and lack of interest—although, in fact, the details of her care suggest otherwise. Unfortunately, anaesthetists are like any other doctor—only human, and without all the answers.

If symptoms persist despite all appropriate care ongoing support for the patient, further investigation, and follow up are needed until resolution occurs.

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Further study is needed of possible long term sequelae

EDITOR—Weir's experience of anaesthetic pain management for postdural puncture headache has clearly left an unfortunate impression.[1] Regional anaesthesia for labour and delivery has gained widespread use because of its effectiveness and safety. Though many obstetric anaesthetic units now regularly audit outcome and patients' satisfaction with regional techniques, this tends to happen during the early days, leaving us with little information about long term sequelae after inadvertent dural puncture.

The incidence of dural puncture during labour epidural analgesia ranges from 0.04% to 6%.[2] Patients with severe refractory postdural puncture headaches are usually treated with an epidural blood patch after simpler methods have been exhausted. The previously reported success rates of epidural blood patching of between 94% and 100% were overgenerous and are not supported by the evidence available. A Canadian review of the literature suggested that persistent symptomatic pain relief could be expected in 61-75% of patients with an initial epidural blood patch.[3] A North American survey found complications to be common after epidural blood patching, with 86% of centres reporting patch failures and 44% reporting persistent headache after two or more patches.[2]

Costigan and Sprigge reported that in patients in whom accidental dural puncture had occurred during obstetric epidural analgesia, headache was perceived to be the most severe symptom, occurring in 86% of patients.[4] Altogether 47% of patients received an epidural blood patch, which was initially effective in 70%, although the headache recurred in 71% of these after discharge. Overall, headache lasted for a median of eight days and recurred after discharge in 47% of all patients with inadvertent dural puncture. Backache occurred in 70%, and 58% continued to have it after discharge.

MacArthur et al found that, among 4700 women who had delivered their most recent baby under epidural anaesthesia, inadvertent dural puncture had occurred in 74.[5] Altogether 23% of these reported a new headache, migraine, or neck ache or a combination of the symptoms, starting within three months after childbirth and lasting for from nine weeks to over eight years.

Although most women would remember a dural tap, and this might influence their reporting of subsequent symptoms, the above findings provide a clear indication of the need for further study of the possible long term sequelae of accidental dural puncture. The previously expressed optimism about the efficacy of epidural blood-patching may be unwarranted.

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Registering clinical trials

Register of clinical trials in children must be set up

EDITOR—Tonks's paper[1] and other efforts to boost registration of new clinical trials[2] should be supported. Registration of clinical trials would benefit evidence based health care and freedom of information and, more importantly, would benefit patients, particularly children.

Randomised controlled trials aimed at meeting the requirements of regulatory agencies are seldom carried out in paediatric patients, and many children thus receive drugs that do not have labelling for paediatric use.[3] Furthermore, there is an imbalance in the distribution of efforts and resources. Systematic studies (and reviews) have focused mainly on a few childhood illnesses such as asthma, otitis media, and respiratory tract infections—as found by consulting the Cochrane Library.

This longstanding, underprivileged position of children underlines the need for further approaches aimed at promoting the rational use of drugs in paediatric patients. Over the past 20 years the number and diversity of databases available to the biomedical research community have grown considerably. These resources, including a few that deal with children, range from registries of patient information to datasets of national demographics.

Comparable approaches in gathering and organising information on randomised controlled trials have not been developed.[2] Although the need to identify unpublished randomised controlled trials in children was noted long ago,[4] no retrospective and prospective registries have been set up. These registries are a useful resource for doctors planning new studies, promote communication and collaboration between researchers, and facilitate patients' (and parents') access to and recruitment into trials.

The European Network for Drug Investigation in Children[5] is considering the feasibility of a pan-European register of randomised controlled trials in children. As Tonks points out, it is encountering some barriers (economical, legislative, ethical, and, in particular, cultural) to establishing a collaboration among all members. Nevertheless, we strongly agree with Tonks's statement that registering a clinical trial is "a clinical declaration of intent by those doing the work, and those paying for it." Registering trials would satisfy the right of children, parents, and their family doctor to have

access to all the available evidence, including that on trials in which they might take part.

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Italian clinical trial registry is kept by pharmacists on local ethical committees

EDITOR—The debate on registering clinical trials[1 2] and its importance for all interested parties (investigators, government agencies, the pharmaceutical industry, local ethical committees, and patients) is of great relevance in Italy nowadays. Recent legislation has given more power and responsibilities to local ethical committees in terms of approval of protocols and monitoring of studies.[3]

The Italian Society of Hospital Pharmacy (Società Italiana di Farmacia Ospedaliera) proposed to its pharmacists' network involved in local ethical committees that it should keep a registry of all clinical protocols under evaluation by the committees.[4] The Italian law requires a pharmacist to be appointed ex officio in each local ethical committee,[3] and the registry was set up in August 1998.

The registry has four objectives: to share information among participating committees on protocols under evaluation at different sites; to give an overall picture of clinical experimentation in Italy; to monitor clinical trials at central and local levels while they are being carried out and after they have ended; and to give local committees the possibility of giving predefined activity reports on their activity.

Data are now available on one year of activity of 20 local ethical committees in different geographical areas in Italy. Information on 625 evaluations corresponding to 452 protocols was collected. One hundred and nine of the protocols were evaluated by two or more local ethical committees. Most were experimental studies evaluating drugs (n = 376), sponsored (348), and multicentre (393).

Among drug studies, 226 were phase III studies. A larger proportion of phase I-II studies than phase III studies were not sponsored (22/63 (35%) *v* 29/226 (13%)) and single centre (18/63 (29%) *v* 9/226 (4%)). The complete data are available at the society's website (http://www.sifo.it).

Data will be collected via the internet. Access will be password protected and limited to the participating local ethical committees. Data collected through the registry are:

- General characteristics of the study (type, title, objectives, coordinating centre, treatments, phase, study design, population, statistics, sponsor)
- Local ethical committee's decision (approved/not approved/suspended) and reasons leading to the decision
- Study monitoring at local level (first enrolment date, payment, reason for and date of interruption/suspension/ending of the trial in the centre, number of patients enrolled at the end)
- Study monitoring at central level (protocol amendments, adverse drug reactions).

We believe that making clinical trials registries into working instruments and a source of information for local ethical committees' activity will contribute to their completeness and their usefulness at national level.

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Global medical knowledge database is proposed

EDITOR—If every doctor who produced a critical appraisal in response to clinical uncertainty shared that learning globally then access to medical knowledge would be greatly enhanced.

What are the difficulties facing clinicians seeking knowledge?[1] The Cochrane Library, *Best Evidence*, *Clinical Evidence*, or guidelines in hard copy might be searched. If the answer does not seem to exist in a synthesised format the practitioners might do a Medline search. There is then the problem of getting the full text of the article.

The next step is appraising the article(s) for validity and to identify the results. The process that should be used when evaluating the different types of individual articles has been formalised and outlined in a series of articles in *JAMA* and described in an editorial in that journal.[2] This step is time consuming and requires skill and practice. Finally, the results should be presented in a format that is easily recognisable (critically appraised topic), such as that used by the *Journal of Evidence Based Medicine*, with a declarative title, message deriving from the results, and comments relating to real patients.

Realistically it is practical for a clinician to question, search, select, acquire the paper(s) and appraise them, and act only three or four times a year. Importantly, the knowledge acquired remains inaccessible to any other professional. If we could share these appraisals on a web based (and CD Rom) database we could avoid a massive duplication of effort. We could also make access to the knowledge much faster.

The global medical knowledge database will match each clinical query as closely as possible with both answered and unanswered questions. If there is an answer the software will display it automatically, in the form of a critically appraised topic. If the question is unanswered the doctor will be able to see whether someone is trying to answer it (and could offer to help). If the question is not on the database then the doctor will be prompted to post it.

Doctors offering to answer questions would search, appraise, and synthesise the evidence into a summary answer using free software available from the Centre for Evidence Based Medicine. The doctor would then post the answer, via a peer review process, to the database so that the next person would find it in the database and not repeat the work. The non-profit making database will have 24 hour access and be comprehensive, valid, up to date, and easy to search, providing answers to questions within seconds.

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Problem solving treatment for depression

Does the paper really prove that problem solving treatment is helpful?

EDITOR—Please tell me if I am missing something, but I am not convinced that the paper by Mynors-Wallis et al shows that problem solving is an effective treatment for depression.[1] As I read it, patients were allocated to one of four groups. Of the two groups treated with problem solving alone, up to a quarter (10 of 39 treated by the doctor and six of 41 treated by the nurse) withdrew from the trial because the treatment was not working. Those left in the trial—that is, those for whom the treatment was working—were compared with those given antidepressant treatment, and it was found that the treatment was working. I note that

none of the antidepressant group withdrew because the treatment was not working.

It may well be that problem solving is helpful for depressed patients. It may well be that an hour's initial treatment followed by up to six sessions of half an hour is helpful for depressed patients. But I am not convinced that this paper proves it.

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1 Mynors-Wallis LM, Gath DH, Day A, Baker F. Randomised controlled trial of problem solving treatment, antidepressant medication, and combined treatment for major depression in primary care. *BMJ* 2000;320:26-30. (1 January.)

Study should have included placebo group

Editor—Mynors-Wallis et al conclude that problem solving is an effective treatment for depression.[1] They base this statement on the effectiveness of selective serotonin reuptake inhibitors and on the lack of difference that they found between their study groups. We base our critique of their argument on the lack of a placebo group.

The authors compared four groups of depressed patients receiving problem solving treatment given by a general practitioner, problem solving treatment given by a nurse, a selective serotonin reuptake inhibitor, or a combined treatment (antidepressant plus problem solving treatment). Although the authors referred to historical probabilities of recovery in placebo groups in studies that used selective serotonin reuptake inhibitors, they did not use this information in their analysis. They used analysis of variance for continuous variables and found no differences between the groups at baseline, 6, 12, and 52 weeks.

A much more relevant answer to the study's main question entails analysis of the categorical variable recovered subjects versus not recovered subjects at the end of the 12 week trial. The χ^2 test showed no differences between the groups.

Since in this test of independence we can use only marginals to calculate expected values, the test does not permit us to obtain expected values under different models—for example, historical placebo rates. We believe that the results of the analysis used in this paper do not address the question of the effectiveness of these interventions. If the authors' line of reasoning was accepted then there would remain the unsupported statement that problem solving is effective.

We explored recovered versus not recovered subjects for each group, using χ^2 with a 47% baseline recovery rate quoted in the paper. We found $P = 0.59$ for problem solving treatment given by a general practitioner, $P = 0.39$ for such treatment given by a nurse, $P = 0.02$ for antidepressant treatment, and $P = 0.12$ for antidepressant plus problem solving treatment. This suggests that problem solving alone or in combination is not effective in the treatment of depression.

We believe that concluding that problem solving is effective on the basis of no differences between the groups is unwarranted and that this study fails to show that problem solving is effective in treating depression in primary care.

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Cost effectiveness is not clear

Editor—It is disappointing that Mynors-Wallis et al did not include cost data in their paper.[1] If two treatments are equally effective the general principle is that the treatment of choice should be the least expensive, so the following three inferences may be drawn if we accept the paper's conclusions.

Firstly, combining problem solving treatment with antidepressant treatment is not justified as there is no added value.

Secondly, since appropriately trained practice nurses are as effective as general practitioners in problem solving treatment they should be the professionals of choice to deliver this treatment if it is deemed appropriate.

Thirdly, it is unclear whether problem solving treatment is more or less expensive than antidepressant treatment. Given that it typically amounted to 3.5 hours' professional time per patient plus an unspecified amount of training, supervision, and administrative input, however, it is by no means cheap. If it is a more expensive choice although of equal efficacy, antidepressants should remain the treatment of first choice. Problem solving treatment should then be reserved for those patients who cannot or will not take antidepressants and who express a preference for a psychotherapeutic intervention.

As other authors have suggested in their electronic responses to this paper, the question remains as to whether it is feasible to introduce training in problem solving treatment into a typical general practice.

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Author's reply

Editor—West asked to be told if he is missing something. The short answer is yes. The results are analysed on the basis of intention to treat with the last available result carried forward. Thus results from patients who drop out are included in the analysis.

Marchevsky and Adebajo criticise the lack of a placebo group. I agree that it would be helpful to have had a placebo group. In the design of the study, however, it was thought to be unethical to have a placebo group when both drug treatment and problem solving treatment have been shown to be significantly better then placebo. Categorical outcomes are given in the paper (that is, recovered, not recovered). There were no significant differences between the four groups at 12 or 52 weeks.

I would not disagree with the inferences that Waldron draws from the study. In an earlier study of the treatment of emotional disorders in primary care, problem solving was more expensive than drug treatment in terms of treatment costs but resulted in significant savings from indirect costs.[1] Colleagues and I have submitted a bid to evaluate fully the cost effectiveness and cost utility of problem solving and drug treatment in a more naturalistic study of depressive disorders in primary care.

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The debate over complementary medicine continues

Evidence for homoeopathy is lacking

Editor—Apologists for homoeopathy must continue to overlook a mounting tide of evidence that the remedies are not effective. Indeed, there have been at least 13 reviews and meta-analyses conducted since the mid-1980s on various aspects of homoeopathy.[1 2] These reviews have failed to identify a single condition for which the remedies are efficacious and, more recently, have noted that the best studies show no effect. Add to the mix the fact that homoeopathic remedies are at odds with well established principles of physics, chemistry, and pharmacology, and one has ample grounds for sustained scepticism.

What is of most concern about the article on homoeopathy by Vickers and Zollman is the selective quotation in the discussion.[3] The authors noted that "practitioners select a drug that would, if given to a healthy volunteer, cause the presenting symptoms of the patient" but failed to note that blinded studies have shown that such volunteers are unable to distinguish between homoeopathic remedies and water.[4] Vickers and Zollman also stated that "there is currently insufficient evidence concerning the relative benefits of the different approaches to treatment" while failing to note that there is no evidence or good reason to believe that there is benefit to any of those approaches. The authors also said that the "notorious Benveniste affair, which involved accusations of fraud and scientific misconduct after the publication of an in vitro experiment in *Nature*, continues to dampen enthusiasm for basic research in homoeopathy" while failing to note that at

least three attempts at replication of the experiment using similar or identical methodology have failed. There are numerous other examples.

One wonders at what point the evidence showing that the medications are ineffective and, indeed, cannot be effective will be adequate to convince the true believers in homoeopathy. Most likely it will be never, as evidence does not seem to be able to consistently change beliefs.

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"Complementogenic" disease may be increasing

Editor—Your series on complementary medicine was timely.[1] More and more problems are caused by patients' use of complementary and alternative medicines, and all doctors should ask patients specifically about their use of these treatments as it may well be that much NHS time and money is being wasted on "complementogenic" disease. It is only since I qualified as a homoeopath that patients have been open in admitting that they use such treatments, and a recent survey at our large general practice found that over 50% of the women aged 20-60 were consulting complementary practitioners or taking supplements or herbal products. In discussions of these problems at a journal club, other practitioners have been surprised at how many symptoms can be attributed to supplements. Certainly a few treatments such as kava (*Piper methysticum*), which is rich in coumarins which interfere with warfarin, have been mentioned in the *BMJ* in the past year.

In our practice we have seen a case of severe dyspepsia caused by zinc, which had been bought by mail after hair analysis by mail, being taken at six times the recommended daily allowance; a patient with blood pressure that was difficult to control because of ginseng; a patient with severe headaches on waking caused by evening primrose oil; and a patient with myopathy caused by creatine, to mention only a few. These conditions necessitated an endoscopy, a medical referral, and a computed axial tomography scan, as well as numerous blood tests. The aetiology was only ascertained by direct questioning. All cases resolved when the patients stopped taking the substance. We suspect that these cases represent the tip of the iceberg.

Caution should be exercised in condoning the use of any supplement or herbal preparation without checking with a pharmacist or reliable source. Many herbal remedies are dangerous to patients with epilepsy or diabetes and to those taking warfarin; they also have the propensity to cause illness in those who are otherwise healthy and not taking drugs.

It is to be hoped that the scientific committee in the House of Lords that is looking at complementary and alternative medicine will regulate the sale of these products and the practice of those who supply them; it is important that we educate ourselves to recognise their potential problems and acknowledge why our patients seem so keen to consult practitioners other than ourselves. Perhaps if more doctors qualified in the treatments known to be superior to placebo, such as homoeopathy and acupuncture, there would be less need for patients to consult other practitioners.

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Debate of the 1840s is revisited

Editor—Vickers and Zollman's article on homoeopathy was a balanced account of homoeopathic medicine.[1] However, the ensuing dialogue between medicine and "evil quackery" shows some parallels with that in the 1840s. In both cases, the engagement has been vigorous and hostile; alternative medical systems were booming then, just as they are today.

The pressure of public popularity is driving the worldwide growth of alternative medicine.[2] Clearly patients who turn to alternative medicine are unhappy with some aspect of conventional treatment, and they should reveal their motives to their doctors. Unless their disappointments are addressed, more patients will inevitably flock to such therapists. Holistic treatments may offer slender hope to patients, but they seem to prefer hopes to drugs and surgery.

Many people refer to the unproved efficacy of homoeopathy and the "ludicrous" nature of its minimum doses. The usual argument is that because "it cannot work" therefore "it does not work." In the 1840s, Sir John Forbes, physician to Queen Victoria's household, called "the infinitesimal doses" of homoeopathy "an outrage to human reason."[3] Its successes were written off as "self limiting diseases." But as patients know, few diseases improve when left alone. Thus, to claim that homoeopathy works because patients "would get better anyway" does not square with human experience: if doing nothing for self limiting diseases is the reason that homoeopathy works, then why should anyone bother giving any drugs at all?

It is doubtful that patients would pay high fees for treatments of no value. The argument that they are rich and desperate (or stupid) enough not to know whether a treatment works seems unconvincing. Vickers and Zollman state that there is a lack of "evidence that homoeopathy is clearly efficacious for any single condition." How does one define "evidence" when homoeopaths deny the existence of single conditions? Trials of homoeopathy have been disappointing, but the weight of anecdotal evidence must count for something. In the 1850s it was widely predicted that "medical fads" such as homoeopathy would fizzle out in a few years.[4] That they have failed to do so either indicates that they do work or stands as a testament to human credulity.

The growing demand for these treatments is a central and uncomfortable reality which medicine must face up to.[5]

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More radiotherapy resources are needed for children as well as adults

Editor—Burnet et al highlight the prejudicial effect on outcomes of the lack of radiotherapy resources in the United Kingdom.[1] They cite several cancers occurring in adults but fail to mention children's cancers.

Cancer is one of the four big killing diseases in childhood; after leukaemia, tumours of the central nervous system are the most common cancers in childhood. Brain tumours are categorised as moderate risk diseases by the Department of Health. Radiotherapy, either as an adjunct to surgery or as the sole modality, is an important component of the therapeutic strategy for many of the 400 new cases of childhood brain tumour occurring every year in England and Wales.

In medulloblastoma, five year event free survival rates of up to 70% can now be achieved in some countries[2]—a figure hardly approached in the United Kingdom. In single site intracranial germinoma the rate can be 90-100%.[3] There is currently a lag time of up to six weeks to starting sophisticated neuraxial radiotherapy in children. This is unacceptable for both those tumours with a high doubling time, such as medulloblastoma,[2] and those producing distressing symptoms for which radiotherapy is the most effective palliative measure, such as diffuse pontine glioma.[4]

Burnet et al raise the issue of hyperfractionation; this is a further strategy that may be of value in the treatment of certain children's brain tumours but would be difficult to adopt without appropriate resources. At the request of the Department of Health, a working party produced a standards document in response to public concern about the adequacy of treatment for children with brain tumours under the NHS.[5] It is ironic that inadequate radiotherapy facilities continue to hamper endeavours to improve outcomes.

Those centres with specialist multidisciplinary paediatric neuro-oncology teams should be enabled to deliver the optimum treatment to children. On behalf of the children and their families we call not only for the provision of more linear accelerators but also for the clinical oncologists, radiographers, and physicists to operate them.

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Twists in the tale of impossible means

In which a copy of the original manuscript is found safe in Norway …

Editor—Recently, Bland correctly identified impossible means for fatigue and scores with the general health questionnaire in a paper published by Pawlikowska et al in 1994.[1 2] The authors admitted the mistake and reported the correct values, but they could not explain how the values came to be incorrect in their paper.[3] Missing proofs, the theft of a computer, and the passage of time might seem like poor excuses and indicate unreliable researchers.

I can, however, confirm that the values reported by Chalder and Wessely in their authors' reply correspond with the values in the original manuscript. I received a copy in 1993, when the manuscript was being reviewed by the *BMJ*. On the front page one of the authors has written by hand: "submitted to the *BMJ*, not for citation." Since then my copy of the manuscript has been stored on a bookshelf at the University of Oslo. I checked it with some excitement after reading Bland's criticism to find that the values in the authors' reply were the same as the those in my copy of the original manuscript (available on request).

Perhaps the authors were correct when they said that the manuscript had been attacked by gremlins. The gremlins seem to have attacked somewhere in the production line because the referees at the *BMJ* reviewed a manuscript with correct means.

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2 Pawlikowska T, Chalder T, Hirsch SR, Wallace P, Wright DJM, Wessely SC. Population based study of fatigue and psychological distress. *BMJ* 1994;308:763-6.
3 Chalder T, Wessely S. Fatigue and psychological distress. *BMJ* 2000;320:515. (19 February.)

… the researchers rejoice that the gremlins were at the *BMJ* after all …

Editor—Initially this correspondence was both painful and embarrassing to us,[1 2] but it has now become less so. It clearly shows, however, that there is no doubt that the analyses and conclusions of our paper written all those years ago remain valid and are certainly not faulty.

In March I received from Dr Loge a copy of the original manuscript that we sent him as a professional courtesy after we had submitted the manuscript to the *BMJ*. As he states in his letter above, the means are indeed correct. Hence our somewhat ungallant suggestion that the *BMJ* was attacked by gremlins though not gallant is accurate.

What have we learnt?

Firstly, we congratulate Bland for rereading an old paper and spotting what no one else had seen.

Secondly, we thank God for Norwegian colleagues who keep copies of old manuscripts.

Thirdly, we will not dispose of proof copies when the reprints finally arrive.

Fourthly, we feel sure that our sleepless nights over this one will be cured by receipt of a bottle of wine from the *BMJ*.

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… the reviewer shows that the gremlins might have attacked on several fronts …

Editor—Bland found that the analysis was flawed in a paper on fatigue and psychological distress that had been published by the *BMJ* in 1994.[1 2] He quite properly asks why nobody noticed this at the refereeing stage. The authors are unable to account for their errors and have tried, as they say, "totally ungallantly" to transfer the blame to the *BMJ*.[3] They also wonder how the referee could have failed to detect the mistakes.

I refereed the manuscript for that paper. I think I can explain the most serious discrepancy identified by Bland. The authors originally coded the four possible responses in each item of their main questionnaires as 1 to 4. Therefore total scores for the general health questionnaire ranged from 12 to 48 and those for the fatigue questionnaire from 11 to 44. I told them they had to code the responses as 0 to 3. It looks as if they gave the correct scores when describing the total sample but did not recode the responses when examining males and females separately. I made various other suggestions, but I was not going to rewrite the results section of the paper and I certainly draw the line at proof reading.

I remember this manuscript well for several reasons. At first I could make neither head nor tail of some of the analyses. I ended up being angry with myself for spending too much time—on a sunny Sunday afternoon—trying to understand the results instead of just recommending rejection. My irritation melted away when I got a note back thanking me for an "excellent" referee's report. At Christmas I received my first ever invitation to the *BMJ*'s friends of the journal party, and I have always believed it was because of the high standard of that report.

May I take this opportunity gently to point out to the editors at the *BMJ* that they have never again invited me to their Christmas party. Am I doing anything wrong?

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3 Chalder T, Wessely S. Fatigue and psychological distress. *BMJ* 2000;320:515. (19 February.)

… and the editor invites everyone to dinner

This fascinating—but ultimately unimportant—correspondence illustrates the difficulties that historians face. Here we have a small episode that happened only six years ago, with all of the main protagonists (authors, reviewers, and editors) contributing to the debate, and still we cannot be sure what happened. You can thus see the difficulty of trying to work out why the first world war started or why dinosaurs died out.

One problem is that we do not have a copy of the edited paper from 1994. The error was clearly not in the manuscript that was first submitted. It might have been introduced by the reviewer asking for a change and the authors not making it correctly. Or it might have been introduced by us in the editing process. Either way, it is an example of publication subtracting rather than adding value, and we apologise to readers and authors for any part we might have played in it.

How can we make amends? Certainly we will ask Dr Pelosi to our Christmas party, but I think too that we should follow the British dictum that when in doubt brew up, start a public inquiry or a royal commission, or hold a dinner. A dinner seems most appropriate. We will call it the dinner of impossible means, and we will invite the authors, the reviewer, the critic, and the Norwegian colleague—and I'll be there to pay the bill.

Richard Smith *editor*
BMJ