

Letters

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New Medical Education Standards Board will lower standards

EDITOR—The government's intended reorganisation of postgraduate medical education received little mention in the *BMJ* until Pereira Gray's editorial,¹ which outlined the proposals² but gave little analysis. By contrast, the analysis in an editorial in the *British Journal of General Practice*—whose view we agree with—was extremely critical.³

The new Medical Education Standards Board, half of whose 24 members are intended to be lay members, will be the single body overseeing curriculums, standards, and the registration of all medical trainees. It will report direct to the secretary of state, whose responsibility for service provision and training will be a clear conflict of interest.

Although the Department of Health's document makes repeated references to the royal colleges sending members to sub-committee meetings and being extensively consulted, the colleges are explicitly criticised for not taking NHS needs into account. The example is given (paragraph 23, bullet point 7) of an accident and emergency unit being unable to deliver services if training recognition is withdrawn. As this happens only if falling standards are endangering the community, it is

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difficult to see how the new board could improve the situation without lowering standards.

Another criticism of current arrangements, and a reason given for rationalising education across specialties, is that it is "difficult for doctors to ... change career paths" and that there is "inbuilt lack of flexibility" (paragraph 22). This inflexibility is not because the different specialties and colleges are trying to protect their patches but because the training in different specialties is different. It is difficult to see how a generic postgraduate curriculum could allow, say, a trainee in vascular surgery to become a forensic psychiatrist.

This document seems to show that the government does not understand how medical education works. There may be good reasons for better coordination between specialties and between hospital medicine and general practice, but the proposed board as it is intended to function is not the solution.

The first paragraph of the document's introduction places postgraduate medical education firmly within the clinical governance framework. It is true, as the document states, that there are tensions between service and training. The clinical governance solution to this is to have more doctors, not for doctors who have failed in one specialty to find a short-cut route to another specialty, or for standards to be lowered to ensure staffing of accident and emergency departments.

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2 Department of Health. *Postgraduate medical education and training*. The Medical Education Standards Board, London: DoH, 2001. (www.doh.gov.uk/medicaltrainingintheuk)

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Mortality and volume of cases in paediatric cardiac surgery

Paper confirms poor quality of paediatric heart surgery at Bristol during 1991-5

EDITOR—Spiegelhalter's paper represents further statistical evidence confirming the poor quality of paediatric open heart

surgery at Bristol Royal Infirmary during 1991-5.¹ It finally lays to rest the argument that the low number of operations was the only contributory factor to the high death rates.² Spiegelhalter confirms that low numbers contributed to between 12% and 17% of the excess mortality observed at Bristol Royal Infirmary. When his findings are coupled with an article confirming that Bristol was an outlier in terms of its performance as a paediatric cardiac surgical centre at this time, the justification for attempting to obtain a review of the service seems incontrovertible.³

The conclusions of the Hunter/de Leval inquiry are difficult to understand in this context unless there was some interference in the processes of the inquiry by the management of Bristol Royal Infirmary, as there was in the publication of favourable articles in the *BMJ*.² The findings and judgment of the General Medical Council's inquiry are entirely vindicated by these more recent publications.

This paper is one of the final pieces of the jigsaw that was the Bristol Royal Infirmary cardiac surgery service, and it fits neatly with what is already known. There is one further piece of the puzzle, which is also the final defence of one of the surgeons (JDW). This is that the substandard paediatric cardiac surgery represented only a small fraction of his workload. This claim should now be examined, especially in the light of the findings of the investigation by Professors Nick Black, Tom Treasure, and Ken Taylor; they concluded that the risk adjusted mortality for low risk adult coronary artery surgery of one of the disgraced surgeons (code 1231) was five times that of his colleagues.⁴

The lesson for the future must be that all services must prospectively collect standardised outcome data for comparison with other centres and to enable performance monitoring at trainee and specialist levels, as has been introduced in Geelong.⁵ These goals are readily and rapidly achievable if the will to improve exists.

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Volunteered mortality data may be unreliable

EDITOR—Spiegelhalter's article adds to the already persuasive data from the early 1990s suggesting an inverse relation between volume of cases and mortality in surgery for congenital heart disease.¹ But he goes on to compare mortality derived from the cardiac surgical register, hospital episode statistics, and the Bristol Inquiry. This comparison is fundamentally flawed and is almost certainly unfairly biased against Bristol. Mortalities in Bristol have been so closely scrutinised for the period covered by the inquiry that they are likely to be accurate. To our knowledge, however, there has been no attempt to validate the volunteered mortality data from the cardiac surgical register or hospital episode statistics for the same period.

The United Kingdom central cardiac audit database has collected data from all congenital heart disease centres in the United Kingdom since April 2000, including volunteered mortality data. In contrast to the cardiac surgical register and hospital episode statistics, it also tracks mortality independently, using the patient's NHS number and a direct link to the Office for National Statistics. Volunteered and centrally tracked 30 day mortalities differ considerably. Overall, tracked 30-day mortality in individual hospitals was up to 25% higher than reported discharge mortality. Seven of 11 centres in England under-reported early mortality, sometimes because patients were discharged early but also sometimes because the reporting was erroneous. Six patients who died within seven days of operation were wrongly reported as alive at discharge.

Use of hospital episode statistics did not improve the accuracy of status reporting. In a sample of nearly 3000 procedures carried out between 1 April 2000 and 31 March 2001 data from hospital episode statistics under-reported the total number of procedures by 10% and under-reported 30-day deaths by 9% but also classified 1% of surviving patients erroneously as dead. We understand that links between the Office for National Statistics and hospital episode statistics are being explored, but those links did not exist when the 2000-2001 data became available or at the time of the Bristol Inquiry.

The differences between mortality in the volunteered cardiac surgical register data and the Bristol Inquiry data were probably of at least equal magnitude to those described above. Any new or past comparison of mortalities that fails to take into account the difference in data quality from non-validated sources and from Bristol over the period of the inquiry risks doing serious injustice to Bristol as well as to the profession's ongoing attempts to restore the

public's confidence in congenital heart disease services in the United Kingdom.

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A case of murder and the *BMJ*

Personal paper is anything but balanced interpretation

EDITOR—Meadow has sought to overcome criticism of the misapplied statistics in the trial of Sally Clark by his own version of the evidence.¹ He has missed the point. His belief in her guilt does not justify misplaced evidence. Watkins addressed the issue of presenting statistical evidence fairly.²

He concluded that doctors should not use techniques without acquainting themselves with the principles underlying them. Meadow blames biased media reporting while advancing his own interpretation of the prosecution evidence in a manner that leaves little room for doubt. Yet such a strong bid to minimise the influence flawed statistics may have had betrays certainty in a field where there should be considerable room for doubt. I do not know the details of the case, but the mere fact that many medical witnesses were called by both the prosecution and the defence indicates the evidence was not clear cut. Unless Meadow is the victim of some conspiracy by the press, the edition of the *Observer* published the day after the *BMJ* gives details of the defence evidence, suggesting his paper is anything but a balanced interpretation.³

The power of medical opinion in judgments is quite profound, so it is not possible to know what influence the misleading information had on the outcome of the trial. It is crucial that opinion must at all times be accurate and evidence based if miscarriages of justice are to be avoided. Essential criteria have been set down for expert opinion,⁴ and require the expert to:

- Provide a straightforward and not misleading opinion
- Be objective and not omit factors which do not support their opinion
- Be properly researched.

When belief systems become rigid to a degree where perceived guilt is put forward as justifying bad evidence, the concept of an independent and impartial adviser to the court is lost. This undermines the court process and the principles of natural justice as required under article 6 of the Human Rights Act.

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Was it truly murder or sudden infant death syndrome?

EDITOR—Meadow writes of his role in the case of Sally Clark, who was convicted of murdering her two sons.¹ Mrs Clark claimed that the children died of the sudden infant death syndrome. This is not a true syndrome but rather an attempt to explain sudden unexpected death in a young infant. Undoubtedly it is heterogeneous, with some cases having no proved explanation; these are the cases that give rise to concern, particularly when the event occurs within a sibship.

There is now convincing evidence that two genetic entities can be responsible for sudden infant death syndrome events.^{2,3} These reports were not described at the time of Clark's trial, but when death is not satisfactorily explained by events we should not try to manufacture a cause just because we do not understand it. Meadow and others give no credence to the possibility of a genetic explanation for these boys, whereas in fact the risk of recurrence might have been 25% or even higher.

Contrary to what Meadow says about the negligible importance of the 1 in 73 million risk of recurrence, which he firmly stated, I submit that this carried a great deal of weight with the jury and probably also with the appeal judges. Meadow's improper use of statistics was irresponsible, particularly when the point was belaboured in cross examination by the Crown and thus probably bolstered a relatively weak case based on inconclusive pathology.

It is clear from the transcripts that the case for the prosecution was by no means clear cut. The autopsies that were done were not the best possible: in such cases a paediatric pathologist should do them. In addition, in all such unexplained deaths cell cultures should be obtained and DNA banked for possible investigation of a molecular defect.⁴ In cases of recurrent sudden unexplained deaths in infants a clinical geneticist should be involved in the investigation in addition to experts in child abuse or other paediatric experts.

The Clarks asked for a paediatric pathologist, but Cheshire Constabulary refused, presumably on the grounds of cost. If Mrs Clark had been guilty they are unlikely to have requested a superior form of investigation, particularly as the pathologist in their first child's case had said that the death was due to a respiratory infection; he reversed this statement later. Meadow states that the appeal judges found that both the Clarks gave untrue evidence.

But that is only an opinion, not a proved fact.

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Competing interests: None declared.

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Depression and unintended pregnancy in young women

Paper raises at least three concerns

EDITOR—Reardon and Cogle's paper raises at least three concerns.¹

Firstly, their analyses do not address the stated hypothesis. No results indicate whether "prior psychological state is equally predictive of subsequent depression among women ... regardless of whether they abort or carry to term." Nevertheless, their unstated hypothesis, focused on abortion and depressive symptoms, may be the more central question.

Secondly, the final sample of women is surprisingly small. Only 421 of the initial 4463 women reported a first abortion or first unintended delivery between 1980 and 1992. Is it possible that the question in 1992 asking pregnancy intention actually referred to a much narrower time frame (that is, a delivery between the biannual surveys)? Little information is given about the abortion question; it is possible that the index unintended pregnancy defined in 1992 resulted in neither the first abortion nor the first delivery.

Thirdly, the discussion omits mention of possible residual confounding. The national longitudinal survey of youth (NLSY) uses a four item abbreviated version (NLSY Cronbach α 0.35) of Rotter's original 60 item locus of control scale, which itself is probably an inadequate proxy for prior psychological state. Furthermore, a one year measure of income may be only a modest proxy for a person's lifetime socioeconomic position.^{2,3} The robustness of the authors' findings could be examined with other available measures. For example, the 1980 Rosenberg self esteem scale data (NLSY Cronbach α 0.83) and the full 12 years of annual income and family size data would be stronger, though still less than optimal, tests of the hypotheses.

This criticism is not an attempt to dismiss research on the topic; rather, such important and highly contentious questions require published studies with equal degrees of rigour and transparency.

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Unmarried women do not show psychological harm from abortion

EDITOR—Reardon and Cogle start their paper by making unwarranted claims, which they attribute to an article by Major et al.^{1,2} That article in fact makes conclusions opposite to their own: Major et al state that the women experiencing psychological problems or regret after abortion are those with prior episodes of depression.¹ Reardon and Cogle turn this on its head, trying to make it look as though prior psychological state predicts depression associated with a pregnancy, whether aborted or carried to term. Major et al, of course, claim no such thing.

Reardon and Cogle's study finds that in unmarried women levels of depression do not differ between those who abort their pregnancy and those who carry it to term,² which seems to negate the push to limit access to abortion for teenagers. This might well be the most important finding in the study, as enormous effort is placed on limiting teenagers' access to abortion in the United States, where the Elliott Institute is based (www.cpcworld.org/hope-net/CPC/Elliott-Institute.html); this raises doubt about the authors' claim that they have no conflict of interest.

Certainly, this study shows that depression is not a factor in the issue of teenagers obtaining abortions. The authors seek to explain this away, but only with unsubstantiated speculations. If they have so little faith in their result why are they trying to present the study as a factual one?

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Women's marital status may not have been accurate in study

EDITOR—Several methodological flaws in Reardon and Cogle's analysis undermine the conclusions stated.¹ There are two in particular.

Firstly, in one study cited in the article higher scores on the Rotter scale correlated with higher depression scores. However, the scale measures locus of control; it is not a measure of depression and thus is not a valid indicator of prior psychological state or prior depression.

Secondly, women included in the sample were categorised in the analysis according to marital status in 1992, yet data regarding first abortion or first unintended

delivery are taken from 1980-92, with abortions and deliveries on average occurring between 1984 and 1986. Marital status in 1992 was not necessarily the marital status of women included in the sample during their first abortion or first unintended delivery. Thus basing the analysis and conclusions on the categories of married versus unmarried women is invalid and is not meaningful.

Given these observations, more rigorous analysis of the data is needed before any conclusions can be drawn about the link between depression and unintended pregnancy and marital status.

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Readers should bear in mind potential conflict of interest

EDITOR—Reardon and Cogle claim no conflict of interest in their paper.¹ However, the principal author (Reardon) is a professional anti-abortionist and the funding organisation for which he works has as its primary aim propagandising against abortion. Therefore the sampling, the methods, the statistics, and the conclusions should be rigorously evaluated.

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Authors' reply

EDITOR—Longer versions of our replies to the above letters have been posted as rapid responses (bmj.com/cgi/eletters/324/7330/151#top); the shorter versions are given here.

With regard to Kahn's comments, review of the variables showed that the cases of abortion were properly identified but many cases of unintended first deliveries were missed. We apologise for this error. To correct this problem we obtained details regarding intention for delivered pregnancies over all years.¹ The table shows the corrected results, which are within the confidence limits of our original results.

A more careful reading of our remarks and Major et al's study will show Goddik that our statements are correct. Also, Goddik misapplies the finding regarding unmarried women to teenagers. Our study does not examine marital status at the time of the pregnancy. We segregated women on marital status at the time that depression was measured in 1992. Contrary to Billings's complaint, this is a valid and meaningful control for social support in that key year, 1992, since marital status is associated with depression rates.

Women scoring in range for high risk of depression (CES depression score >15) who had their first abortion or unintended childbirth between 1980 and 1992

	Women with unintended births but no subsequent abortions		Women who had an abortion		Adjusted odds ratio (95% CI)*
	Total	No (%) at high risk	Total	No (%) at high risk	
Unmarried†	253	91 (36)	129	37 (29)	0.88 (0.54 to 1.43)
Married†	530	101 (19)	164	43 (26)	1.92 (1.23 to 2.97)
In first marriage	443	78 (18)	131	35 (27)	2.23 (1.36 to 3.64)
All women	783	192 (25)	293	80 (27)	1.39 (1.02 to 1.90)

*Adjusted for family income, education, race, age at first pregnancy, and 1979 Rotter score. †When CES depression questionnaire was administered in 1992.

No study is without weaknesses. In ours, the Rotter scale is admittedly an imperfect measure of psychiatric state before pregnancy, but it does correlate to depression in our study as well as those of others. Our findings are at least sufficient to cast doubt on the prevailing hypotheses that unintended deliveries are more harmful to emotional health than abortion and that any subsequent differences associated with outcome of pregnancy can be explained entirely by prior mental state.

We have never seen political or moral views on abortion identified as a conflict of interest in any of hundreds of studies published on abortion. If Blanchard is recommending a change in this custom he would more credibly begin by identifying his own association to the National Abortion Federation as a conflict of interest.² It is a matter of public record that one of us specialises in research on post-abortion adjustments and rejects the notion that abortion is a harmless panacea.^{3,4} That opinion, like the present study's findings, is consistent with the literature.⁵

Every study should be carefully scrutinised; we claim no exception to this rule. Fortunately, the national longitudinal survey of youth is publicly available and our analyses can be readily replicated.

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Hound of the Baskervilles effect

What about the good days?

EDITOR—Phillips et al have uncovered a fascinating relation between the day of the month and the mortality rate for Chinese Americans and Japanese Americans.¹ There clearly is an increase in chronic heart disease deaths of Chinese and Japanese on the fourth day of the month. Phillips et al

attribute this to the similarity in the Chinese and Japanese languages of the spoken words “death” and “four.”

Have Phillips et al considered whether days with a pleasurable association might have a beneficial impact? Their analysis shows a decrease in mortality for Chinese and Japanese on days 20, 26, and possibly 12. Is there any resemblance, either spoken or pictorially, between the words for those days and words evoking feelings of relaxation, wellness, or happiness?

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Bad4U?

EDITOR—In a large observational study Phillips et al noted an increase in cardiac mortality on the fourth of the month among Chinese and Japanese Americans that does not occur in white matched controls.¹ This increase is particularly marked for inpatient deaths from chronic heart disease in California (figure 3). Phillips et al link this observation to their cultural and linguistic association of the number 4 with death.

This effect may well be real, but some important issues remain unaddressed. Phillips et al have addressed a hypothesis that to them clearly relates to one specific day. To be really convincing, we need to be sure that the mortality on day four really should be regarded as an outlier, relative to other dates, and this is not the foregone conclusion that might be supposed. Directly from figure 3, the most extreme part of the data, the 95% confidence interval for mortality on the fourth day, overlaps the intervals for most of the other dates. Moreover, the relevant rate ratios, 1.07 for all cardiac deaths, 1.13 for chronic heart diseases, and 1.27 restricted to California, are presented with ordinary 95% confidence intervals, but no P values, in accordance with normal *BMJ* policy. The one situation, however, in which P values are more directly informative than the corresponding confidence intervals is when we are trying to assess whether a striking observation might be merely a coincidence. For cardiac mortality as a whole, the log rate ratio seems to be 0.068 (SE 0.021, z=3.17, P=0.0015). These figures are reconstructed from the heavily rounded

ones given and hence are only a crude approximation. Although a P value of 0.0015 seems fairly extreme, this relies heavily on starting with the hypothesis that it is the fourth of the month that is different.

A more conservative (more convincing to sceptics) P value involves a Bonferroni correction by a factor of 28 (the number of days studied, which are common to all months), giving 0.043, which, although technically significant, is far from extraordinary. It is true that when a similar process is applied to the more extreme rate ratios of 1.13 and 1.27 above, they remain highly significant. But it is commonly found that by restricting attention to subgroups of the data, one can enhance the nominal statistical significance in this way. It is far from clear whether it was a prior hypothesis that this effect would be much more marked in California than elsewhere. The hypothesis set out by Phillips et al relates exclusively to cardiac mortality, which conveniently squares with both the data presented here and the Baskerville link. But if stress associated with this date is so devastating to Chinese and Japanese people, one would expect to see marked effect on accidental and suicide deaths also, which do not seem to have been examined.

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Authors' reply

EDITOR—Glaser thinks that low mortality levels on days 20, 26, and 12 might be linked to positive Chinese and Japanese attitudes. This suggestion is implausible. Firstly, the low mortality values on these days are not statistically significant, because the error bars for these days overlap with the regression line representing the expected mortality levels. (Because of a proofreading error, the top of the error bar for day 26 was cropped short and should have extended beyond the regression line to 67.06. Our original graph had the correct error bars, but we missed the distortion in the page proofs.) Secondly, although Chinese have a general preference for even numbers, the emotions evoked by “20,” “26,” and “12” are no more positive than for other even numbers.

Nearly all Newcombe's concerns spring from his conjecture that we noticed the fourth day peak but did not predict it, and therefore need to assess the significance of this peak with the Bonferroni correction. Newcombe acknowledges that, even with this correction, the fourth day peak remains significant. We have used and analysed the Bonferroni correction in our previous articles, but this correction is inappropriate for the current investigation because we did

predict the fourth day peak.^{1,2} The circumstances were as follows.

The lead author (DPP) and two of his Chinese students (KK and GL) were driving past a Chinese supermarket named "Ranch 99." DPP asked the others why "9" appeared in this name, and was told that in Mandarin "9" is pronounced identically to the word for "lasting" and is therefore an auspicious part of a business's name. DPP then asked if there were any inauspicious numbers, and was told that, in Mandarin, Cantonese, and Japanese, the number "4" is pronounced almost identically to the word for "death." At this point, DPP predicted that Chinese and Japanese mortality would peak on the fourth of the month. This prediction sprang to mind because DPP has long used short term mortality fluctuations to seek evidence of psychosomatic processes.³⁻⁵

Incidentally, "4" is the only number between 1 and 28 with pronounced, negative connotations in Mandarin, Cantonese, and Japanese. Because the fourth day peak was predicted, one can legitimately compare mortality on the fourth (first period) with average mortality on days 1-3, 5-28 (second period). The error bar for the second period is very small and does not overlap with the error bar for the first period. This resolves Newcombe's concern about overlapping error bars.

Newcombe wonders why we focused on cardiac mortality (and ignored suicides and accidents). We adopted this focus because we wished to test a surprising hypothesis: psychological stress might trigger heart attacks. We were not interested in testing the unsurprising hypothesis that psychological stress might trigger suicide.

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New act regulating human organ transplantation could facilitate organ donation

EDITOR—As the rapid responses to Hoffenberg's reminiscences show, the United Kingdom has not been spared misunderstandings and controversy over death and brain function.¹ Skegg has chronicled the confusion that followed the report by the conference of Medical Royal Colleges in

1976, when the conference was not asked (nor apparently was it able) to agree whether patients with brain stem death were dead and so avoided saying they were until 1979; yet even the editor of the *BMJ* failed to notice the point in 1976 and declared that the report "sets out clear guidelines for the diagnosis of death."²

The only reason we need to label as dead those patients without brain stem function is to make beating-heart organ harvesting from them legal and because attempts to extend the label to patients with what we now call persistent vegetative state were not generally accepted.³ The main premise on which the colleges have argued the propriety of accepting brain stem death as death of the person has always been the claimed inevitability that when brain stem function ceases "the heart will stop beating shortly thereafter."³ There is no physiological reason why this should happen, and review of the published evidence shows that it is not true.⁴

An important danger of persisting with this false premise is that more perceptive observers and critics of medical practice have already noticed the discrepancy and have concluded (inappropriately) that misdiagnoses are being made.⁵ I suggest that it is time to revise the law, perhaps by a new Human Organ Transplant Act, to allow families to give informed consent to beating-heart organ donation under anaesthesia for patients certified to have irreversible loss of brain stem function. The unanswerable semantic question of whether the donor is dead would then disappear.

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Friends and family are good (and cheap) life coaches

EDITOR—Although I am sure that Kersley's article on how to change your life is a product of her deep desire to help people, I am not convinced of its value.¹ Television, magazines, and bookshops are awash with advice about motivation and personal development, but several questions need to be considered before anyone accepts such advice uncritically.

Firstly, can we be sure that life coaches and other happy-making gurus do any good? Secondly, and more importantly, can we be sure that their techniques and advice do no harm? Although this may be a counterintuitive concern, I do not think we know enough about these strategies to rule out

serious unwanted effects. Thirdly, why are there seven steps on the journey to nirvana?^{1,2} Why not six or eight or a million? Fourthly, are life coaches anything more than paid friends? A similar question has already been asked of psychologists and counsellors.³

Despite my misgivings about life coaches and experts in motivation, I have decided to start my own consultancy and will soon be sending articles to the *BMJ* that will educate readers about the "real" way of achieving fulfilment. Those readers who want one to one coaching should contact me at max@you can have it all 'cos life's a piece of cake so go for it.co.uk. Alternatively, readers can retain responsibility for their own life and save themselves some money by discussing what they want out of life with trusted friends and family. And then they should listen to the best life coach of them all—themselves.

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GPs should reduce antibiotic use with alternative treatments

EDITOR—Increasing evidence accumulating from the Cochrane Database of Systematic Reviews suggests that the benefits of antibiotics in upper respiratory tract illnesses in childhood are modest. Nasrin et al have shown that the use of antibiotics in such cases increases the prevalence of antimicrobial resistance in the children.¹ Their study provides yet more urgency to reduce the use of antibiotics in general practice for acute respiratory infections.

But how can this be achieved? Doctors do not necessarily share a sceptical approach to the use of antibiotics. The barriers to the implementation of best evidence are being explored and described.² For example, general practitioners are more influenced by certain clinical signs and symptoms to use antibiotics for acute respiratory infections than the evidence suggests is effective.³

Are the public campaigns run in Belgium and the United Kingdom to reduce use of antibiotics the best approach? Doctors may be placed in an ethical dilemma to choose between what they think is best for their individual patient and what is deemed best for the community, now or in the future. Another problem is the replacement of "something that can be done for the patient" by a sort of nihilism: "Antibiotics provide such a weak benefit that they are hardly worth the bother. And then there's all the resistance worry."

Other treatments for acute respiratory infections exist that are just as effective, but

they do not have the same ring to them as curative ones: killing bacteria has a more satisfying sounding objective than the palliative alternatives, but does this matter? For spontaneously remitting diseases, anything that reduces the symptoms is just as effective as anything else, bactericidal or not. We therefore suggest wider dissemination and greater promotion of alternative treatments (evidence based, of course). These include short acting agents such as analgesics, non-steroidal anti-inflammatory drugs, and steroids; vaccination against the pneumococcus and influenza; xylitol liquid and chewing gum; and better communication skills.^{4 5}

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Topical negative pressure may help chronic wound healing

EDITOR—Harding et al's review article on healing chronic wounds¹ fails to mention treatment with topical negative pressure, which has an emerging role in the management of chronic wounds.^{2 3}

The simple technique entails applying an open-pore foam dressing (polyurethane ether) to the wound. This dressing is sealed with transparent adhesive drape. A negative pressure or suction force is then applied across the wound via a drainage tube embedded in the foam.⁴ There are various regimens, describing the amount of negative pressure, continuous or intermittent pressure cycle, and frequency of dressing changes.

Delivery of topical negative pressure is critical to the healing of wounds and can be done with a commercially available device (VAC pump, KCI), wall suction, or surgical drainage bottles. Commercially available devices tend to be most reliable, providing controlled pressure delivery, and have built in safety devices.

The exact mechanism of action of treatment with topical negative pressure is still not clear.² Proposed mechanisms of action include:

- Change in microvascular blood flow dynamics, with an improved local blood supply
- Removal of fluid exudate
- Stimulation of the formation of granulation tissue
- Reduction in bacterial colonisation
- Mechanical closure of wounds by reverse tissue expansion
- Maintenance of a moist wound environment with better wound healing

Treatment with topical negative pressure can be used in both hospitals and community settings. Cost implications are important, and currently the commercially available suction pumps are more expensive than conventional dressings. However, such treatment is associated with accelerated healing rates, reduced nursing time, simpler operations, and decreased bed occupancy. All of these mitigate the initial cost of treatment.

Treatment with topical negative pressure is presently undergoing worldwide evaluation and is being used for acute, subacute, and chronic wounds with good results.² A recent Cochrane review suggests weak evidence with regard to chronic wound healing.⁵ This possibly relates to the paucity of large randomised controlled trials of the treatment. In our experience it has an emerging role in chronic wound healing, and a large controlled trial would scientifically address the issue.

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Study of effect of delays on ovarian cancer was weak

EDITOR—Kirwan et al concluded that delays in referral or diagnosis did not adversely affect the survival of patients with ovarian cancer.¹ Their analysis of the relation with survival, however, includes stage of diagnosis as a covariate, so if delays adversely affect survival by producing a more advanced stage of disease at presentation (which seems an obvious possibility) this study would not show it.

In other diseases, such as breast cancer, some analyses show that, after adjustment for stage, a longer symptom history relates to an improved prognosis; this is understandable as for a given stage at presentation a longer symptomatic phase may indicate a less aggressive cancer.^{2 3} Analysis of length of symptom history with regard to survival is

complex, and long follow up is needed to allow adjustment for lead time effects. Analysis should not adjust for variables such as stage, which may be intermediary in the causal pathway.⁴

Kirwan et al's study is also weak because it depends solely on general practitioners' records, with no attempt at validation, and is of a small sample. Surprisingly, the authors can "see no other way to obtain the relevant data" than to rely on general practitioners' records. The issue is little different to that of obtaining reasonably consistent and accurate data about aetiological factors, which is done by using well designed structured questionnaires and trained interviewers who are independent of the staff providing clinical management.

While of course needing more resources, investigations of symptom history and presentation can be carried out on representative series of patients, perhaps in combination with studies of aetiological factors.⁵

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Using twin studies to label disease as genetic or environmental is inappropriate

EDITOR—The cover of the *BMJ* on 2 February poses the question "Rheumatoid arthritis: is it genetic?" and answers "Probably not." Neither the question nor the response is valid.

The Danish twin study on which this conclusion is based identified no concordant monozygotic twin pairs and only two concordant dizygotic pairs from a sample of 37 338.¹ By modelling the reported numbers of confirmed cases of rheumatoid arthritis, we calculate that the study had 80% power to detect a heritability of 65% and 90% power to detect a heritability of 75%. Thus there was insufficient power to detect a genetic influence on rheumatoid arthritis equivalent to the 60% heritability estimated in the two most recent twin studies of the disease.²

The point estimates of concordance are also cause for concern. The prevalence of rheumatoid arthritis at 0.15% seems low, indicating possible deficiencies in screening. The absence of the middle aged birth cohort severely limits the sample's representative-

ness. The failure to identify a single monozygotic concordant pair (we know that they exist: cases identified in recent studies have been characterised in detail³) indicates bias in the zero concordance estimate.

The difficulties inherent in designing twin studies of rheumatoid arthritis inspired the two largest and most recent studies in Finland and the United Kingdom, which yielded remarkably similar results despite having contrasting sampling strategies.² Potential limitations of these studies are incorrectly highlighted in the Danish report. Cases of ankylosing spondylitis were specifically excluded in the Finnish study. The classification of rheumatoid arthritis entailed applying specified criteria. In the United Kingdom study the similarity in concordance among pairs recruited in the media and by general practitioners gave no reason to suspect disproportionate recruitment of concordant monozygotic pairs.

Using the results of twin studies to attach the label "genetic" or "environmental" to disease is inappropriate. This is readily appreciated by considering the classical examples of phenylketonuria (where an inherited disease is expressed only in the context of specific environmental exposure) and tuberculosis (which shows a greater concordance in monozygotic than dizygotic twins).⁴ The contemporary value of estimating heritability from twins is in establishing the likely success of specific strategies to detect the action of individual genes. In rheumatoid arthritis the established importance of genetic variation in HLA and the identification of new genetic regions linked to the disease is ample support for an important genetic contribution.⁵

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Removing barriers for disabled people would be giant leap

EDITOR—Emery and Buch's editorial about drug treatment for rheumatoid arthritis states that after 10 years 50% of people with the disease no longer work, most losing their jobs in the first 12 months after diagnosis.¹ But it is not necessarily rheumatoid arthritis that causes job loss; it is likely also to be due to the barriers in society that exclude disabled people from full employment.

I have had rheumatoid arthritis for over 10 years, and have written this letter using voice-activated software, originally provided through the government's Access to Work programme. This provides funding for equipment and other means of overcoming barriers that would exclude people from work. Health professionals and employers generally aren't familiar with such provision. Thus some people probably give up their jobs without knowing about any of the support and funding that are available, or about protection afforded them by the Disability Discrimination Act 1995.

My continued employment is also influenced by the fact that my job attracts a high salary. I can therefore pay for what I need to overcome barriers to employment and to live a full life outside work. For example, my car has automatic gears and power assisted steering; I can afford taxis; I live in a flat in the city centre with a lift that works; I pay a cleaner; I eat out a lot.

Tumour necrosis factor α blockade costs about £6000-8000 a year per patient.¹ If every year that money was given direct to the patient tax free I expect that, for most, their quality of life would improve far more substantially than it does if they take the drugs.

The government recently required every local authority, in conjunction with health authorities, to develop a Welfare to Work plan for disabled people, to increase their opportunities for employment. In Liverpool this was drafted mainly by disabled employees of the local authority, health service, and voluntary agencies.² Disabled people discussed the barriers they face in society.^{3,4}

According to the editorial's subheading, the drugs discussed might be only "a small expensive step."¹ A giant leap would be to address the disabling barriers in society; this approach is increasingly recognised as important in tackling inequalities faced by disabled people, but health professionals—the medical profession in particular—are slow to recognise this.⁵

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Brain denies being forbidding and insensitive to change

EDITOR—In his editorial on forbidding research, froth, and colour in journals and magazines, Smith places *Brain* and *Cosmopolitan* at opposite ends of the spectrum of articles that are published.¹ We are amused by this, but we hope that, without pausing too long from the important task of developing tactics for encouraging subscribers actually to read the *BMJ* (and for pleasure, not as a chore), he might also find time to look at a copy of *Brain*.

Without wishing to be unduly competitive, either with the *BMJ* or with our stable mate *Cosmopolitan*, we at *Brain* have printed in colour and had an illustrated front cover since 1994; receive and review papers on line; have an e-toc facility (the number of people taking advantage of it has trebled in the past few months, over which period access through our home page has doubled); are set to introduce a pay-for-view system (shades of *Cosmopolitan* here) and provide free electronic access to all articles published more than 12 months previously; and have an increasing number of electronic subscribers (a paper on the neuroanatomy of pleasure while eating chocolate received 14 831 hits).

Nineteenth century copies of our journal are still read and cited for their content, not the advertisements (there are none). Because we have a high reputation among clinicians and scientists and pay attention to language and syntax, *Brain* is purchased, well read, and profitable.

As a charity, we will distribute £200 000 in the current financial year. We offer travel grants to young neuroscientists, entry fellowships for clinical trainees in neurology wishing to get started in research or move back to clinical training, research leave fellowships for those who have completed their clinical training and need re-exposure to research, and top-up salaries to encourage aspiring neurologists to work in basic science laboratories where non-clinical salaries are being offered. These activities are designed to strengthen clinical neuroscience.

Research oriented we may be; forbidding and insensitive to the changing needs of academic medicine we are not.

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Rapid responses

Correspondence submitted electronically is available on our website