Letters

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Risk of connective tissue disease among women with breast implants

Study adds nothing to knowledge of processes of tissue injury induced by silicone

EDITOR—In a study that was five years old at publication Nyrén et al claim to show "no evidence of association between breast implants and connective tissue disease," using classical rheumatic diseases as end points.[1] This report is less comprehensive than that by Gabriel et al, although comparable in scope and shortcomings of definitions.[2] It lacks data on rupture (the prevalence rises with time[3]); rupture enhances the reaction to gel filled devices.[4] Gabriel et al estimated the minimum population necessary for risk assessment to be 62 000 subjects with implants and 124 000 controls.[2]

Table 3 is confusing because the observed numbers of cases are compared with the expected numbers, derived from the standardised hospitalisation ratios. The data can mean only that in Sweden the rate of admission to hospital for rheumatic disease is the same whether a patient has silicone implants or not. Few patients go to hospital for rheumatic assessment in the United States. We have admitted none for rheumatic disease over the past two years from our silicone clinic; not all removals of implants are done in hospital.

Adjustments for pre-existing diagnosis or miscoding do not seem to fit between tables 3 and 5; the total of individual defined disorders exceeds that for all patients by about a third, which is the frequency of overlap syndrome. Overlap syndrome has been ignored in this study despite its importance in the classification of rheumatic disorders.

Data adjustments are necessary to ensure the accuracy of the tables, but they may not show the biological meaning of the data. For example, average follow up does not indicate how skewed the clinical assessment point is with respect to the average. Brawer has shown a time dependent crossover at 5-6 years between change in clinical status and cumulative rupture rate in 300 patients with siliconosis.[3]

The study is short when one considers the immunopathic reaction cycle before autoimmune conversion; the T cell reaction peaks 10-12 years after implantation and then declines, reaching effective quiescence more than 20 years after implantation unless immunomodulatory treatment or removal of the implants intervenes.[5]

The report adds nothing to discerning the processes of tissue injury induced by silicone, is too short to detect autoimmune conversion, ignores the atypical nature of siliconosis seen clinically and in laboratory tests, and is close to the logical fallacy of taking an absence of evidence as evidence of absence.

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Professor Shanklin has acted on behalf of a patient in one case recently; Professor Smalley has not acted in any relevant cases since mid-1995.

1 Nyrén O, Yin L, Josefsson S, McLaughlin JK, Blot WJ, Engqvist M, et al. Risk of connective tissue disease and related disorders among women with breast implants: a nationwide retrospective cohort study in Sweden. *BMJ* 1998;316:417-22. (7 February.)
2 Gabriel SE, O'Fallon WM, Kurland LT, Beard CM, Woods JE, Melton LJ. Risk of connective-tissue diseases and other disorders after breast implantation. *N Engl J Med* 1994;330:1697-702.
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Authors should have made better use of matched control group

EDITOR—In view of their acknowledged funding from Dow-Corning Corporation, Nyrén et al should have been more careful to avoid even subtle bias in the presentation of their results.[1] They analysed two groups of patients with breast implants (cosmetic and breast reconstruction groups) and a carefully matched control group for the patients with cosmetic implants, consisting of women who had breast reduction surgery. The most important comparison should have been between the cosmetic breast implant group and the matched control group;making this comparison is the reason for selecting that control group.

Their finding of a relative risk of 0.8 for being admitted with a connective tissue disorder is derived from the whole breast implant group (cosmetic and reconstruction) compared with the breast reduction group. Thus, having selected a control group, they fail to use it appropriately or to provide data allowing others to do so. From the data they do present, one can compare the cosmetic implant group as a whole with the breast reduction group, although this is imperfect because some patients from the cosmetic implant group are unmatched. This comparison gives an unadjusted relative risk of 1.2 for patients with breast implants later developing connective tissue disorders requiring admission to hospital. Although the 95% confidence interval includes 1.0, this may be a less palatable figure to the funders than the relative risk of 0.8 misleadingly quoted.

I urge the authors to state the relative risk and confidence intervals (from their matched patients) of admission to hospital with connective tissue disorder after receiving cosmetic breast implants compared with breast reduction. They should also present data on the subgroup with silicone gel implants, using the matched controls for that subgroup. The authors should emphasise the size of relative risk of developing connective tissue disorders which they can exclude with 95% confidence. They could allay public concern (if appropriate) by quoting mean or maximum absolute risks and comparing these with other absolute risks from the literature—for example, the risk of developing lung cancer if one smokes.

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1 Nyrén O, Yin L, Josefsson S, McLaughlin JK, Blot WJ, Engqvist M, et al. Risk of connective tissue disease and related disorders among women with breast implants: a nationwide retrospective cohort study in Sweden. *BMJ* 1998;316:417-22. (7 February.)

Authors' reply

EDITOR—We studied over 7000 women, and although this was one of the largest cohorts of women with implants and the follow up was one of the longest, we clearly stated that the outcomes evaluated (admissions to hospital) represented only the more serious illnesses from definite connective tissue disease. No evidence of increased risk of admission for these conditions was associated with implants.

Shanklin and Smalley suggest that silicone would not be expected to induce conditions serious enough to require admission, but many of the original hypotheses about breast implants involved debilitating illnesses. The authors claim that our study was "five years old at publication." This is untrue; we only recently completed the fieldwork. Their confusion about tables 3 and 5 may stem from a failure to understand

the concept of a standardised hospitalisation ratio or their lumping together fibromyalgia with definite connective tissue disease. We considered two broad diagnostic entities—definite connective tissue disease, and related conditions including fibromyalgia. Shanklin and Smalley infer that considerable overlap syndrome occurred; in fact the prevalence of multiple conditions was small and similar among those with breast implants and those who had breast reduction surgery.

Atherton suggests that our analysis was biased by the source of funding. We prefer to have the study judged on its methods rather than on non-scientific innuendo. The cohort study approach that we used is common epidemiological practice and offered the advantage of a standardised ascertainment and follow up of patients. Atherton objects to our use of the entire implant cohort instead of the cosmetic implant subcohort in making the direct comparison with the breast reduction cohort. Although in the design stage the patients who had breast reduction were matched to the patients with cosmetic implants, in the analysis stage—by matching for several variables—we could include all patients with implants. Nevertheless, if the patients with cosmetic implants were compared with those who had breast reduction the resulting relative risk for all definite connective tissue disease would be 1.0 (95% confidence interval 0.6 to 1.8). Because we had information on the silicone content of only a sample of the implants, we did not calculate risk ratios according to implant type.

While subject to the limitations we ourselves highlighted, our study provides systematic information obtained in an area of the world much less affected than others by publicity about adverse effects of implants. It shows that women with implants experience rates of serious connective tissue disease similar to those of other women in Sweden.

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Genetics consortiums can offer views facilitating best practice in Alzheimer's disease

Editor—Alzheimer's disease is often taken as an example of a disorder for which the impact of the new genetics will be met,[1 2] and it was with this in mind that the United Kingdom Alzheimer's Disease Genetics Consortium was formed. Members of the consortium are clinicians involved in both genetics and dementia care, academics, members of lay organisations, and representatives from pharmaceutical research; they meet to consider the consequences of the growing understanding of genetics in Alzheimer's disease. Through the consortium we have participated in the discussions referred to by Gill and Richards,[3] have suggested measures to ensure the ethical conduct of genetic research,[4] and are encouraging the coordinated provision of clinical genetic services for early onset familial Alzheimer's disease. More importantly, however, the collective view of the consortium has helped clinicians facing requests for genetic testing for late onset Alzheimer's disease; current data suggest that apolipoprotein E genotyping offers little in the way of prediction.

Whether apolipoprotein E genotyping should be used to aid diagnosis is more controversial than Bell suggests.[1] While some people have recommended apolipoprotein E genotyping in diagnosis, other consensus groups have come to the contrary view. The United Kingdom Alzheimer's Disease Genetics Consortium is concerned that the criteria to be expected from an adjunctive diagnostic test have not yet been met and that possible adverse consequences of diagnostic testing, although not as great as those of predictive testing, should not be ignored. Genetic tests are not the same as other tests because information may be relevant to other family members. Apolipoprotein E4 homozygosity in a patient with dementia increases the probability of a diagnosis of Alzheimer's disease but simultaneously doubles the lifetime risk for 65 year old first degree relatives of the patient. The consequences for the provision of long term care costs and the implications for any insurance are considerable.[2]

Careful consideration of the impact of genetic testing is important, and this includes diagnostic tests. While waiting for research on strategies to respond to genetic testing, as suggested by Marteau and Croyle,[5] groups such as the United Kingdom Alzheimer's Disease Genetics Consortium can offer a collective view which, though not substituting for empirical evidence, can facilitate best practice. If other such groups are established this will raise questions regarding coordination and dissemination. Perhaps there is a role here for the genetics advisory commission or the Department of Health.

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1 Bell J. The new genetics in clinical practice. *BMJ* 1998;316:618-20. (21 February.)
2 Pokorski RJ. A test for the insurance industry. *Nature* 1998;391:835-6.
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More collaboration is needed about benefits of new genetic screening tests

Editor—Gill and Richards request a full and rigorous assessment of the benefits and costs of introducing new genetic screening tests.[1] They point out the need for collaboration between geneticists, public health specialists, and primary care teams and emphasise the importance of the general public.

A spot analysis of data available for East Sussex, Brighton and Hove Health Authority for the current financial year (1997-8) from an eligible general population of roughly 730 000 shows at least 28 referrals to date for genetic testing, of which 18 were funded, six were cancelled by the originator, and the remainder are still in progress. Readiness to undergo testing involves the capacity of the funding commissioners to ensure that there is a confirmed benefit to the patient so that they can fund such testing. This is in concert with one of the principles identified by Wilson and Jungner for successful screening: "there should be an accepted treatment for patients with the disease."[2]

Currently such tests are normally experimental and have been offered to individuals and couples as part of the overall genetic screening and diagnostic service, usually by centres with established national or international reputations. This experimental nature also means that general and professional information on the benefits and cost benefits is of limited availability and is produced often by the very centre offering the test.

Readiness to undergo testing is also influenced by the patient and the parent, and from their viewpoint there is sometimes conflicting evidence from different perspectives; every screening test does not necessarily bring benefit to the patient.[3] The result is that the commissioners agree the funding of such requests even though the benefits to the patient may sometimes be difficult for them to establish. Primary care practitioners and public health doctors have generally been too distant from the emerging science to enable the usual discussions that would otherwise occur between these professionals on newly evaluated tests.

Arrangements to define likely service needs and the collaboration required, and support for the proposition that expert committees must fully recognise the importance of open debate, will be most welcome.

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1 Gill M, Richards T. Meeting the challenge of genetic advance. *BMJ* 1998;316:570. (21 February.)
2 Wilson JMG, Jungner G. *Principles and practice of screening for disease.* Geneva: World Health Organisation, 1968. (Public health paper No 34.)
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Diagnosis of Creutzfeldt-Jakob disease by measurement of S100 protein in serum

Appropriate study populations must be used

EDITOR—Otto et al describe significant differences in the serum concentrations of S100 protein in patients with and without Creutzfeldt-Jakob disease.[1] The population of patients studied is, however, not appropriate for assessing the usefulness of measuring serum concentrations of S100 protein as a diagnostic test, and Otto et al's calculations of positive and negative predictive values of the test are invalid as they fail to take into consideration the prevalence of Creutzfeldt-Jakob disease as a cause of dementia.

Patients with clinically evident disease do not need a diagnostic test. It is not informative to evaluate the potential of a blood test to distinguish those with clinically definite Creutzfeldt-Jakob disease from those without the disease on clinical grounds. The test is useful only if it helps to predict the likelihood of the disease in a population that does not meet the criteria for probable Creutzfeldt-Jakob disease, which is therefore the population in which it must be assessed.

Otto et al say that the primary use of serum concentrations of S100 protein will be in the differential diagnosis of diseases that cause dementia. At a cut off of 213 pg/ml they calculate a sensitivity of 77.8% and specificity of 81.1% for detecting Creutzfeldt-Jakob disease in a referred population of 108 patients with definite or probable Creutzfeldt-Jakob disease and 74 patients with dementia without Creutzfeldt-Jakob disease (table). When calculating the positive and negative predictive values of a test the prevalence of the target disorder within the population to be examined must be considered.[2] There are no criteria for distinguishing the subgroup of patients with dementia but without Creutzfeldt-Jakob disease (who in Otto et al's study were referred to the surveillance unit for Creutzfeldt-Jakob disease) from other patients with dementia. We must therefore consider them to represent all patients with dementia. If the relative prevalence of Creutzfeldt-Jakob disease in dementia is taken to be 1 per 1000 people aged 60-70—a considerable overestimation—the number of false positive results in the population with dementia increases enormously (table). The positive predictive value of the test falls from 85.7% to 0.4%. The negative predictive value rises from 71.4% to 99.97%. This test therefore has no use in this population.

Finally, we agree with Pocchiari that measurement of serum cencentrations of S100 protein may have a role in the diagnosis of Creutzfeldt-Jakob disease.[3] Further studies are required, however.

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1 Otto M, Wiltfang J, Schutz E, Zerr I, Otto A, Pfahlberg A, et al. Diagnosis of Creutzfeldt-Jakob disease by measurement of S100 protein in serum: prospective case-control study. *BMJ* 1998;316:577-82. (21 February.)
2 Sackett DL, Haynes RB, Guyatt GH, Tugwell P. *Clinical epidemiology, a basic science for clinical medicine.* 2nd ed. Boston, MA: Little, Brown, 1991:86-8.
3 Pocchiari M. Early identification of variant Creutzfeldt-Jakob disease. *BMJ* 1998;316:563-4.

Tonsil biopsy helps diagnose new variant Creutzfeldt-Jakob disease

EDITOR—Early diagnosis of new variant Creutzfeldt-Jakob disease remains a challenge. We agree with Pocchiari that much further work is needed,[1] but we believe that tissue diagnosis after tonsil biopsy is helpful in diagnosing new variant Creutzfeldt-Jakob disease. While important work has been performed on surrogate markers in sporadic Creutzfeldt-Jakob disease—for example, cerebrospinal fluid 14-3-3 protein, S100 protein, and neuron specific enolase and serum S100 protein[2]—sporadic Creutzfeldt-Jakob disease usually does not present diagnostic difficulties. In addition, it is unclear whether these markers will help to differentiate Creutzfeldt-Jakob disease from other neurodegenerative conditions that cause difficulty with the differential diagnosis before death—for example, rapidly progressive Alzheimer's disease with myoclonus. These markers, which are likely to be indirect assays of neuronal injury or glial activation, may be unhelpful in early diagnosis of new variant Creutzfeldt-Jakob disease, as it generally progresses slowly. Indeed, early results with analysis of cerebrospinal fluid 14-3-3 protein in cases of new variant Creutzfeldt-Jakob disease are disappointing.[3]

Currently, the main problem is diagnosing disease in a young person with rapidly progressive neurodegenerative features but normal results of neuroimaging and other investigations. The concern is not to miss a treatable disorder, and brain biopsy will be required in selected patients. This can be avoided, however, if a tonsil biopsy specimen is positive for protease resistant prion protein, which is specific for prion disease and seems to be the main, if not the only, component of the transmissible agent or prion. It has long been recognised that the lymphoreticular system is affected early in scrapie and presymptomatic diagnosis of scrapie by tonsil biopsy has been reported.[4] Since protease resistant prion protein was found in tonsil biopsy specimens in new variant Creutzfeldt-Jakob disease[5] we have been using tonsil biopsy as a diagnostic procedure.

Tonsil biopsy may be restricted to specialised centres, but we disagree with Pocchiari's assertion that the procedure has poor benefit for the patient. There is an obvious need to reach a diagnosis and avoid further investigations, which might well include brain biopsy. Potential risks of cross contamination can be managed by adhering to safety guidelines and using a disposable biopsy kit. Only a small specimen of one tonsil is needed, and the small surface area of the incision margins ensures a complication rate far less than that after standard bilateral tonsillectomy. We recommend that tonsil biopsy be considered in all cases in whom new variant Creutzfeldt-Jakob disease is suspected.

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1 Pocchiari M. Early identification of variant Creutzfeldt-Jakob disease. *BMJ* 1998;316:563-4. (21 February.)
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Sensitivity, specificity, and predictive values of serum concentrations of S100 protein in Creutzfeldt-Jakob disease (CJD) among patients with dementia. Predictive values are dramatically influenced by prevalence of target disorder within the population tested

	Otto et al's study*		Expected false positives and true negatives for S100 test if prevalence of CJD in dementia were 0.1%†
S100 protein (pg/ml)	CJD present (n=108)	CJD absent (n=74)	(n=100 800–108)
>213	84	14	20 412
<213	24	60	87 480
Sensitivity	84/108=77.8%		
Specificity		60/74=81.1%	
Total	108	74	107 892

*Prevalence of CJD (108/182)=59.3%. Positive predictive value=true positives/(true+false positives)=84/(84+14)=85.7%. Negative predictive value=true negatives/(true+false negatives)=60/(60+24)=71.4%.
†Prevalence of CJD in dementia of 0.1%. Positive predictive value=true positives/(true+false positives)=84/(84+20 412)=0.4%. Negative predictive value=true negatives/(true+false negatives)=87 480/(87 480+24)=99.97%..

Authors' reply

EDITOR—We disagree with McConville and Craig that our calculations of predictive values for the diagnostic test based on serum S100 protein concentrations are invalid because we investigated an inappropriate study population.

McConville and Craig show that predictive values depend critically on the prevalence of the disease to be detected in the study population.[1 2] Unlike sensitivity and specificity, which are generally thought of as constant benchmarks of diagnostic test

performance,[2 3] predictive values can be interpreted correctly only if the sampling framework that generates the study population has been understood. We did not attempt to evaluate the diagnostic performance of the S100 protein test in an unrestricted sample of patients with dementia. We compared S100 protein values in patients with Creutzfeldt-Jakob disease (according to standard criteria)[4 5] with those in patients who initially had the same differential diagnosis but later turned out to have another disease. Our study population thus consisted of a sample of patients in whom the initial diagnosis of Creutzfeldt-Jakob disease needed to be verified or proved wrong. We used the ongoing surveillance study of Creutzfeldt-Jakob disease in Germany, to which nearly all suspected cases of the disease are reported, as the basis for our investigation. This provides the best sampling framework as it is closest to the typical situation in which the new test would be used in clinical practice.

Moreover, as it is suspected that S100 protein indicates activation of astroglia, which occurs continuously in Creutzfeldt-Jakob disease, and as S100 protein has a short biological half life, we speculated that serial measurements would improve diagnostic accuracy. It may be possible to distinguish diseases in which a short rise of S100 concentrations occurs as a result of destruction or activation of astroglia or of the short opening of the blood-brain barrier. As activation of astroglia also occurs in Alzheimer's disease but progresses much more slowly, S100 concentrations may be a useful marker even at a much lower level than in Creutzfeldt-Jakob disease. We agree with McConville and Craig that further studies of the potentially important role of serum S100 protein concentrations in diagnosing and monitoring of Creutzfeldt-Jakob disease are required, but not with respect to the predictive performance of the test in all patients with dementia.

We cannot comment on Collinge et al's suggestion to perform tonsil biopsy in patients with suspected new variant Creutzfeldt-Jakob disease, as we have not investigated new variant Creutzfeldt-Jakob disease.

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1 Altman DG, Bland JM. Diagnostic tests 2: predictive values. *BMJ* 1994;309:102.
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Having a practice pharmacist can reduce prescribing costs

EDITOR—The articles by Majeed and Head and by Greenhalgh try to identify why setting prescribing budgets for general practitioners is difficult.[1 2] Both sets of authors agree that the quality and effectiveness of prescribing can be improved; such improvement would bring much greater gains than just containing the cost of prescribing in primary care—something that is exceedingly difficult to manage.

The first step in this difficult task is to control costs and keep them controlled.[3] Since employing a practice pharmacist three years ago my practice has seen a continual fall in prescribing costs in relation to health authority and national averages. Latest prescribing analysis and cost (PACT) data show that the practice's prescribing costs are 29% and 24% below these averages respectively. This represents a saving of over £200 000 annually when our costs are compared with those of an average practice of the same size (9500 patients). This achievement is possible because the practice pharmacist ensures that we pay attention to factors that lead towards rational prescribing. Unfortunately, there is little interest in the fact that we have this post: we have been unable to attract funding to establish why it is successful. Without investment in people rather than in statistics based on assumptions, the expensive activity of prescribing in primary care and concordance will remain unsolved.

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1 Majeed A, Head S. Setting prescribing budgets in general practice: capitation based prescribing budgets will not work. *BMJ* 1998;316:748-50. (7 March.)
2 Greenhalgh T. Setting prescribing budgets in general practice: effective prescribing at practice level should be identified and rewarded. *BMJ* 1998;316:750-3. (7 March.)
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Use of statins

Sheffield table is useful …

EDITOR—Two letters last December on the use of statins, by Betteridge et al (p 1619) and Reynolds et al (p 1620), criticised the Sheffield table.[1] The rate of coronary heart disease events targeted (3% per year) is not "arbitrarily high," as Reynolds et al say, but was proposed after consideration of the number needed to treat, cost effectiveness, proportion of adults needing treatment, and total cost of treatment at different thresholds of risk of coronary heart disease.[2] The European task force's guidelines[3] which suggested the 2% per year threshold for coronary heart disease preferred by the letters' authors predated the statin trials.

We do not accept that high risk people over age 65 should be denied treatment,[1] and treating those below age 65 with a relative risk of coronary heart disease of ≥ 4[1] seems unwise. A 35 year old woman with a total cholesterol concentration of 7.0 mmol/l, systolic blood pressure of 160 mm Hg, and no other risk factors has a relative risk ≥ 4, yet her absolute risk of coronary heart disease is only 0.16% a year. The associated number needed to treat for five years to prevent one coronary heart disease event is 375. She will not reach the WOSCOPS (west of Scotland coronary prevention study) level of risk (1.5% per year) even when she is 70.

The Sheffield table does not exclude those with familial hyperlipidaemia from treatment. The footnotes state clearly that some people "off" the table, including those with familial hyperlipidaemia, may be at high risk. The accuracy of the table is similar to that of the coronary heart disease risk chart in the European task force's guidelines endorsed by the authors.[3 4] Omission of high density lipoprotein cholesterol does underestimate risk in non-insulin dependent diabetes, but only slightly. This is not so for the Sheffield table based on the ratio of total to high density lipoprotein cholesterol, which has a sensitivity of 100% and specificity of 94% versus the full Framingham risk function.[4] Its accuracy also shows that dichotomising blood pressure is not important. The Sheffield table for the ratio of total to high density lipoprotein cholesterol is available on request but not yet advocated for general use because most laboratories do not measure high density lipoprotein cholesterol or report this ratio routinely, and because non-specialists may be unfamiliar with this ratio.

The suitability of guidelines for ordinary practice requires that a balance is struck between simplicity and acceptable accuracy. When considering the choice between the Sheffield table, the full Framingham risk score,[1] and the European task force's guidelines[3] the views of general practitioners are perhaps more important than those of 103 specialists.[1]

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1 Use of statins [letters]. *BMJ* 1997;315:1615-20. (13 December.)
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. . . but New Zealand tables are better

EDITOR—We agree with Muldoon and Criqui that better ways of assessing the risk of developing coronary heart disease are needed.[1] In the letters about the use of statins Betteridge et al discussed (p 1619)[2] the shortcomings of the Sheffield tables.[3] We believe that the tables published by the National Heart Foundation of New Zealand for use in the assessment and management of dyslipidaemia[4] have several advantages over the Sheffield tables.

The New Zealand tables estimate the absolute risk of developing a cardiovascular event (myocardial infarction, new angina, stroke, or transient ischaemic event) for a wide range of ages and include total cholesterol:high density lipoprotein cholesterol ratios. They show, for example, the substantial difference in the absolute risk of a cardiovascular event between smokers and non-smokers. They can be used by general practitioners to discuss with patients changes in risk factors that may reduce overall risk and to show alternative strategies to lipid lowering drugs. We have revised the Dorset guidelines for the management of hyperlipidaemia and have adapted the New Zealand tables for use in assessment of risk for primary prevention of cardiovascular events (figure). These revised tables show annual risk, and we advise that lipid lowering treatment should be considered if the risk remains at ≥3% after other interventions, as recommended by the Standing Medical Advisory Committee.[5] The 3% risk recommended by the committee to consider lipid lowering treatment has not been universally agreed,[2] and these tables permit alternative levels of risk to be considered.

We believe that these tables adapted from the New Zealand tables are the best available tool at present for assessing the need for lipid lowering treatment for primary prevention of cardiovascular disease. Because prescriptions for statins continue to increase, we believe that general practitioners will welcome these guidelines now.

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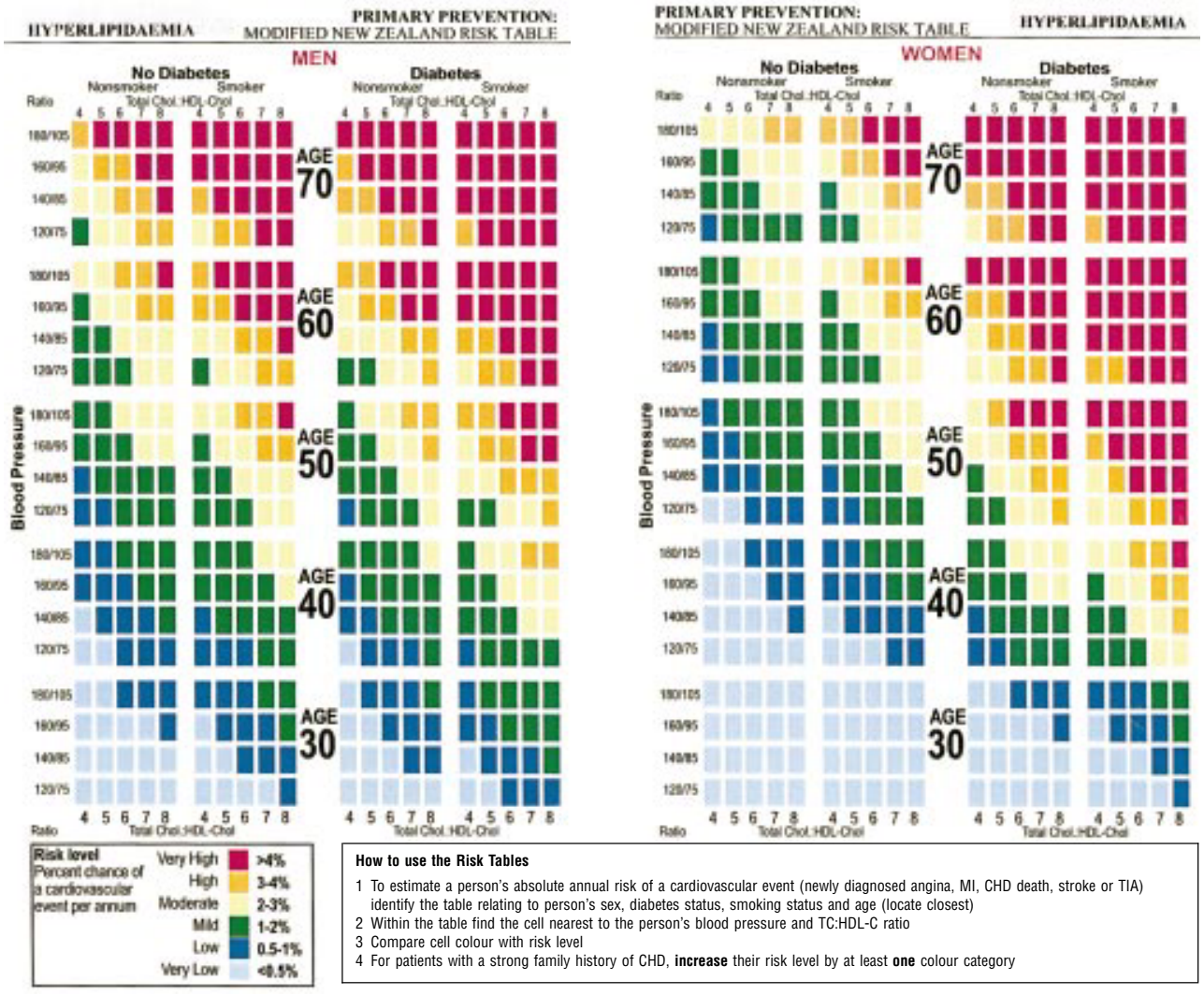

New Zealand tables for estimating absolute risk of developing a cardiovascular event, for men and women (tables are available at http://www.nzgg.org.nz/guide/guide001.htm.)

Refugee families have psychological strengths

EDITOR—As a child psychiatrist who grew up in a refugee family I take issue with Hodes's editorial about refugee children.[1] It views refugees and their experiences through the eyes of a Western psychiatrist. This, unfortunately, means that the picture of illness and disorder portrayed puts an emphasis on Western notions of psychopathology; this gives the impression that there is much serious psychiatric disorder in both the children and their carers. No mention is made of the psychological strengths that refugee families possess and the ability that most have to survive and learn from their suffering.

The same cultural mistake is made in the discussion about the type of help that health and other services can give. The notion of therapy and counselling in different settings is repeatedly put forward. This is the last thing that most refugee families want or need, whether the counsellor is from the refugee community or from the dominant culture. This type of approach to healing is not commonly recognised outside Western societies. It often undermines the families and their culture's own methods and beliefs about healing and help. An Iraqi refugee recently told me how he and many other Iraqi refugees have become more religious since arriving in Britain; he added, "Our faith has helped keep us sane."

More respect and understanding need to be shown to refugees' own cultural background and what they find helpful from professionals. Often it is the simplest things, such as writing a supportive letter to the Home Office or helping them fill in forms, that will make the most positive impact at this time in their lives. Refugees find intrusive the attempt to professionalise and pathologise their experiences. We can learn much more by listening and taking seriously what they tell us is helpful rather than imposing our Western ideas about what we believe they ought to find helpful.

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1 Hodes M. Refugee children. *BMJ* 1998;316:793-4. (14 March.)

People considered virtually unable to walk can walk further and faster than in 1977

EDITOR—Help with mobility for disabled people evolved from invalid cars (Invacars) to the mobility allowance in 1977 and then the "higher rate mobility" component of the disabled living allowance in 1992. Eligibility criteria have remained virtually constant over 20 years, yet the number of recipients has increased dramatically to almost two million.

Distances walked and speeds achieved by four groups studied, and comparison with figures achieved by users of invalid cars

	Distance (m)				Speed (m/s)			
Group	Median	Mean	Range	Comparison with users of invalid cars*	Median	Mean	Range	Comparison with users of invalid cars*
Invacar users	87	154	8-650	—	0.25	0.31	0.04-0.69	—
Year:								
1978	120	161	8-930	NS	0.32	0.41	0.04-1.29	NS
1983	138	195	40-685	P<0.05	0.36	0.40	0.06-0.95	NS
1990	145	209	20-700	P=0.05	0.37	0.42	0.06-0.97	P<0.01
1996	225	318	8-1050	P=0.004	0.44	0.51	0.05-1.46	P<0.01

*Mann Whitney U test.

Over 20 years I have studied five groups of patients considered virtually unable to walk: 36 with invalid cars in 1977; three groups receiving the mobility allowance (34 in 1978,[1 2] 36 in 1983,[2] and 44 in 1990); and 38 receiving the higher rate mobility component of the disabled living allowance in 1996. After a rest they walked at their preferred pace on a level surface until limited by pain, shortness of breath, or distress, or for 12 minutes. The table shows the distance recorded and the average speed to the first stop (or over 12 minutes). The people receiving financial benefit tended to walk further and faster than those with invalid cars. Significant differences were evident from 1983 (table). Representative samples of patients were not available. Although these results in convenience samples may be open to challenge, the trend over time in the mobility of people considered virtually unable to walk is striking. It accords with qualitative observations on much larger numbers and may explain much of the dramatic rise in the number of people receiving financial benefit for disability. The differences were magnified by the change to self assessment with limited professional corroboration when the disabled living allowance was introduced. Medical evidence is again required now; will the trend be reversed in future?

The only specific legal guidance defines virtual inability to walk as being unable to walk less than 50 m without unacceptable discomfort or distress.[3] Only 15% (6/15) of the 1996 sample walked less than 50 m. Roughly 5% of beneficiaries are unable to walk, so less than a quarter of recent recipients may have met these eligibility criteria. If they had been applied rigorously over the years, current expenditure might have allowed higher rate mobility allowance to be awarded to eligible people of all ages.

The changes in mobility over the years probably have many factors: differing diagnoses, the regulations' wording, decisions by social security commissioners, people's difficulties in estimating distance,[4] and the tendency to award benefit for life early in treatable conditions.[5] The relatively normal mobility of considerable proportions of people receiving financial benefit for disability undoubtedly influences the perceptions of others, who no longer see being disabled as a stigma.

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*John Hunter is a member of the Disabled Living Allowance Advisory Board, but these studies were undertaken independently. The views expressed are his own and do not reflect those of the board or the Department of Social Security.

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Crude rates, without standardisation for age, are always misleading

EDITOR—Ten years ago the National Audit Office published a report that attributed the highest coronary mortality in Europe to Sweden.[1] The *BMJ* published a letter that I wrote pointing out that the National Audit Office had committed the error of failing to standardise for age[2]: the Swedes were an ageing population, but after correction for age their coronary mortality was unremarkable. Later the National Audit Office produced a corrected chart for parliament, so that epidemiologists were happier.

Earlier this year a paper by Esmail et al purported to show that the allocation of merit awards was racially biased.[3] My criticism was the same: crude rates are potentially misleading and need to be corrected for age. Through increasing percentage recruitment over time the age distribution of non-white versus white consultants (and indeed of women versus men) cannot be the same. Merit awards are highly dependent on age, so correction for age is essential.

The same accusations of racial bias have also been repeated in a news article by Warden, supported again by crude rates.[4]

Crude rates can be seriously misleading. Are award holders, who spend much valuable time and effort trying to make the system work fairly, going to stand publicly condemned on the basis of naive arithmetic? I know that in these days of tabloid

journalism a story that combines race, sex, money, character assassination, and politics must be difficult to resist, but the *BMJ* is in danger of antagonising many of its senior readers.

Probably, more white than non-white consultants have bifocal reading glasses, and for the same reason that more have merit awards—age distribution. If I am right, lapse of time would decrease the apparent anomaly as age distributions even out. The misleading crude rates could be made similar in the short term only by discrimination in favour of younger non-white and female consultants rather than white male consultants. That would bring the system into disrepute in a different way.

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1 National Audit Office. *National health service: coronary heart disease.* London: HMSO, 1989.
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Career options available to general practitioner assistants

Options are restricted for doctors not wishing to become partners

Editor—Numbers of general practitioners are dwindling, so why is it almost impossible to find a post as a general practitioner assistant? Practices are penalised for taking on assistants instead of partners, because out of hours allowance, basic practice allowance, minor surgery and health promotion payments, cytology and immunisation payments, and postgraduate education allowance are payable only for partners. The BMA exacerbates the situation by refusing to provide guidelines on salaries, quoting job variability as an excuse, even though this does not prevent it from recommending fees for loans.

As I have worked for the NHS for seven years, I deserve a position that provides me with annual, study, and maternity leave and that enables me to contribute to the NHS superannuation scheme. However, I am married and intend to start a family and may have to move because of my husband's job within the next five years.

The only options available to me seem to be to become a general practitioner locum and forsake all my employment rights—as well as most of my job satisfaction—or to take up a nice staff grade job at the local hospital and leave general practice to follow the same path as the dinosaurs.

A disgruntled general practitioner locum
Lancashire

Reply by chairman of General Practitioners Committe's medical workforce subcommittee

Editor—The General Practitioners Committee has now established a non-principals subcommittee to consider and keep under review all matters affecting non-principals and to report with recommendations to the committee. To help non-principals the subcommittee has commissioned the BMA's health policy and economic research unit to survey a sample of the pay and workload of general practitioner assistants using a questionnaire, which will be analysed in due course.

To increase opportunities for salaried service the Department of Health has introduced a new scheme to enable general practices to employ a salaried doctor. Health authorities can reimburse all or part of the costs from cash limited funds for general medical services under an amendment to paragraph 52 of the statement of fees and allowances. The scheme is aimed at improving the career opportunities for general practitioners who prefer not to work as principals and to give health authorities additional flexibility to influence and support local workforce developments and the provision of general medical services. The salaried doctor must be employed under a contract acceptable to both the local medical committee and the health authority.

The General Practitioners Committee is concerned that the scheme has inadequate funding, has no guarantee of continuation (which may deter doctors looking for longer term arrangements), and competes with other demands on the cash limited budget. We have expressed these concerns to the department and are continuing to press for the introduction of further ways of employing salaried doctors to provide properly structured and funded salaried career options. The introduction of such options for doctors wishing to pursue alternatives to the independent contractor status is a priority.

A recent conference on workforce problems facing general practice drew further attention to the difficulties highlighted by the author. The General Practitioners Committee is aware of the concerns of general practitioner assistants and is seeking to ensure that these doctors are not treated unfavourably.

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Search for better inotropic drugs should continue

Editor—In their editorial on drug treatment in heart failure, Steeds and Channer state that "all clinical trials of positively inotropic drugs have either failed to improve symptoms or have increased mortality in heart failure."[1] Even if this statement is allowed, it is a generalisation too far to conclude that "drugs that increase the force of contraction of the failing heart result in increased mortality and…there should be a halt on further development in this direction."

We believe that there are at least two reasons why attempts to develop better inotropic drugs should continue. Firstly, basic studies of heart muscle over the past two decades have shown that there are two cellular routes to improving the strength of the heart: (*a*) increasing intracellular calcium concentrations and (*b*) increasing myofilament calcium sensitivity, resulting in stronger contractions for a given amount of calcium.[2] All clinically tested drugs use the first mechanism either exclusively or predominantly. The second mechanism remains clinically untried. Yet increasing calcium sensitivity has substantial theoretical advantages, especially in relation to energy efficiency, arrhythmogenesis, and efficacy in partially hypoxic muscle[3]—all crucial considerations in the failing heart. Several "calcium sensitisers" are currently under investigation and development.[3]

The second reason is that heart failure can be acute or chronic. In acute heart failure a niche clearly exists for fast acting inotropes with minimal side effects that is not fully satisfied by current drugs. As well as improving symptoms, such compounds would improve the likelihood of surviving an acute event.

Successful drug treatment of chronic heart failure is complex since the heart is already severely diseased once failure becomes apparent. Not surprisingly, it has proved difficult to safely augment the compensatory mechanisms already in play. In future it may become apparent that no inotropic drug possesses a suitable risk:benefit ratio to recommend itself for use in chronic heart failure. On the other hand, calcium sensitisers with an appropriate profile of actions may turn out to be substantially more useful than previously tried inotropes. It is premature (and indeed unnecessary) to write off inotropic treatment for heart failure while a major, independent, and theoretically advantageous inotropic mechanism remains clinically untested.

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1 Steeds RP, Channer KS. Drug treatment in heart failure. *BMJ* 1998;316:567-8. (21 February.)
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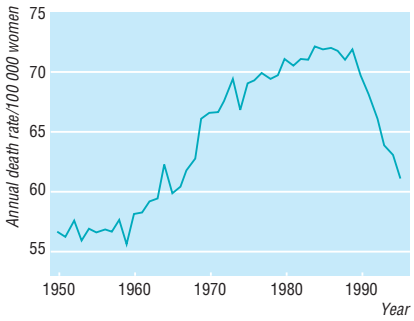
Mortality from breast cancer in UK has decreased suddenly

Editor—Trends in mortality from breast cancer in the United Kingdom are much more encouraging than Wise's news article indicates.[1] Although the age standardised mortality from breast cancer had been increasing steadily ever since the 1960s,



Mortality from breast cancer in United Kingdom, 1950-95 (mean of rates at ages 35-69)

suddenly, in the late 1980s, it flattened out, and then during the 1990s it decreased substantially.[2] Already by 1995 it was 15% lower than in 1985-9, and this rapid decrease seems still to be continuing (mortality statistics for the United Kingdom (1950-95), supplied by the World Health Organisation). Earlier surgery and wider use of systemic treatment,[3] particularly with some years of tamoxifen, must already have been preventing a few thousand deaths from breast cancer in the United Kingdom each year, but this nationwide decrease in mortality is a new and sudden phenomenon that was not apparent until the past decade or so.

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Postnatal health education in Nepal

Study cannot be generalised

Editor—In their study on the effects of postnatal health education on mothers in Nepal Bolam et al conclude that "individual health education for postnatal mothers in poor communities has no impact on infant feeding, care, or immunisation…".[1] This finding cannot be generalised to mothers who do not enjoy the health advantages of the mothers studied by Bolam et al. The effectiveness of health education programmes should be tested among those they are intended to benefit. The authors admit that their group is select, having opted to pay to have their babies in hospital. This reflects a high awareness of health issues and also economic security in a country in which only 7% of births are attended by trained staff and gross national product is $200 (£125) per capita.

Ninety per cent of the deaths that occurred among infants in the study were not preventable, being associated with prematurity and congenital abnormality. Only one death was attributed to respiratory infection and none to diarrhoea; this is remarkable when, as reported by the authors, infant mortality is 98/1000 live births in Nepal. The highly select nature of the women studied is further reflected by the 95% rate of infant immunisation among the group. The authors acknowledge this; they had anticipated rates of 40% and thus miscalculated their sample size. Running a pilot study would have avoided such problems and indicated the need to select a more vulnerable group.

Similarly the statistics on outcome at 6 months reflect a well nourished cohort of Nepali infants; it would be difficult to improve this outcome even if the intervention had improved breast feeding practice. That the rate of breast feeding was below average probably is not a reflection of a lack of awareness of the benefits of breast feeding, but of a more privileged urban lifestyle and various pressures not to breast feed.[2]

However, the mothers displayed good basic health awareness; 93% knew about oral rehydration. The mothers didn't seem to be responsive to learning to count their infant's respiratory rate. We have found that a more easily assimilated message is to "get advice if the baby starts eating less."

Perhaps if the authors had selected a vulnerable group and given more appropriate messages they may have come to a different conclusion. The majority of vulnerable infants have not been immunised, live in poverty, and will never be seen in a hospital. Without tackling poverty we have little to offer these mothers beyond opportunistic counselling and informal education programmes. The authors are right: we need to evaluate our interventions, but we must select appropriate target populations if our conclusions are to be generalisable.

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1 Bolam A, Manandhar DS, Shrestha P, Ellis M, Costello AM de L. The effects of postnatal health education for mothers on infant care and family planning practices in Nepal: a randomised controlled trial. *BMJ* 1998;316:805-11. (14 March.)
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There are no shortcuts

Editor—My wife and I practised for many years in rural India where health education became our priority, therefore I was surprised that the randomised controlled trial by Bolam et al, the field component of which was completed in four months, was expected to give much information on this subject.[1] Four months is far too short a time, and basing the project largely on information supplied by health educators is the wrong approach for this population.

The subject of the nurturing of infants is far more than a cerebral exercise, and the educator's information is unlikely to be accepted initially as superior to that of the mother's peer group and the knowledge of her grandmother.

The authors write that it "might also be argued that mothers in Nepal do not perceive many health workers as purveyors of credible knowledge about motherhood." It can be more than argued; it can be stated. The negative findings of the paper support this. We look on from outside and decide that the poor people of Nepal (or India) are ignorant of what is good for them, but the Nepalese mother will not think that she and her peers are ignorant. She will credit herself with common sense and credit the acquired wisdom of her forebears as fully reliable. Why should she accept automatically suggestions about looking after her baby from an outsider? What you hope she will learn for her first baby may not be appreciated and internalised until she has had her second. Above all she and the people of her village need time to mull over and reconsider their background wisdom and understanding. It will take a number of years.

One of the key messages of the study was that the "efficiency of health education interventions that rely solely on giving people information to bring about change … is unproved …." Surely this was disproved years ago. During our time in India, we did not attempt education of this sort. At hospital education was in groups in the ward. In the villages opportunities for discussion were always sought and arranged. Mothers would quiz and support each other. I do not agree that alternative strategies for health promotion in developing countries have to be more costly, but they are costly in effort and involve much hard slogging and disappointments on the way. There are no short cuts.

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1 Bolam A, Manandhar DS, Shrestha P, Ellis M, Costello AM de L. The effects of postnatal health education for mothers on infant care and family planning practices in Nepal: a randomised controlled trial. *BMJ* 1998;316:805-11. (14 March.)

Quality of health education was not measured objectively

Editor—Bolam et al are to be congratulated for addressing the important issue of the effectiveness of health education in developing countries.[1] As a doctor working in primary health care in Nepal I found their results depressing but not very surprising. The quality of health education offered in Nepal is often poor. It is noteworthy that the authors described measures taken to ensure the quality of the education. However, there are no objective indicators of quality included in the study. We do not know, for

example, whether the women's knowledge improved after the 20 minute educational session. Additionally the setting for the original contact (a maternity hospital immediately after childbirth) and the large amount of information imparted may have contributed to the negative findings.

While new approaches for health promotion based on the methodology of adult learning are undoubtedly needed, it would be a shame to throw the baby out with the bathwater. The case for providing quality health education that is delivered in an appropriate way still needs to be proved.

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1 Bolam A, Manandhar DS, Shrestha P, Ellis M, Costello AM de L. The effects of postnatal health education for mothers on infant care and family planning practices in Nepal: a randomised controlled trial. *BMJ* 1998;316:805-11. (14 March.)

Authors' reply

EDITOR—Three issues have arisen from the correspondence about our study: how representative was our population, what kinds of alternative educational interventions might work, and how should these be evaluated?

We are surprised that Stangroom and Appleby misconstrue our population of mothers as privileged and unrepresentative. They were drawn from an urban setting and they used hospital facilities. One reason for doing this trial was the opportunity presented for health education by delivery in hospital especially since urbanisation increases the proportion of hospital deliveries (now above 70% in the Kathmandu valley[1]). We drew a representative sample from predominantly poor mothers who used the main government maternity hospital where user charges are nominal (15 000 deliveries per year). Our baseline indicators showed that the mothers in our trial were usually smaller than average, more than half were illiterate or had received only primary education, and they had infants with a low mean birth weight. This population is quite different from that attending the hospital run by Stangroom and Appleby's organisation, where there are fewer than 4000 deliveries per year and where user charges are higher and mothers more privileged. Their criticism that health education should be tested among those it is intended to benefit misses the point. This intervention was designed for poor mothers seen in hospital, an important minority group in Nepal. Different strategies are needed in rural areas where contacting mothers is difficult.

We chose to conduct a potentially sustainable educational intervention in a manner recommended by many international agencies. It had little impact. Maybe this is not surprising to an experienced health educator like Thompson but no randomised controlled trials had previously been conducted. Thompson suggests that he knew such methods were valueless and implies that education in groups on the ward and discussions in villages are more effective. Participatory methods at village level might be more effective, especially in rural areas where domiciliary delivery rates are high. But policymakers making decisions about large scale interventions need rigorous evidence of effectiveness not anecdotal reports. They also need information about the costs of such interventions. For this reason we are starting a community based randomised trial in a poor rural district of Nepal to evaluate the impact of a participatory intervention in informal mothers' groups on perinatal and infant care practices and outcomes. We hope others will do similar studies so that health promotion strategies of proven cost effectiveness will receive proper financial and political support.

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1 Bolam A, Manandhar DS, Shrestha P, Manandhar B, Ellis M, Costello AM. Maternity care utilisation in the Kathmandu valley: a community based study. *J Nepal Med Assoc* 1997;35:122-9.

Trials have shown yohimbine is effective for erectile dysfunction

EDITOR—Wagner and Saenz de Tejada state that yohimbine has only a modest effect on psychogenic erectile dysfunction and none on organic erectile dysfunction.[1] This view is supported by the recent American Urological Association guidelines on treatment of organic erectile dysfunction, which state that "the outcome data for yohimbine clearly indicate a marked placebo efficacy."[2] However, organic and psychogenic causes of erectile dysfunction often overlap, which makes differentiation difficult. Our meta-analysis of all double blind, randomised, placebo controlled trials of yohimbine for erectile dysfunction found a significant improvement in patients treated with yohimbine (odds ratio 3.85, 95% confidence interval 2.22 to 6.67).[3] These data, which also relate to men with less well defined causes of erectile dysfunction, suggest that yohimbine is an effective, non-invasive option for initial drug treatment.

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1 Wagner G, Saenz de Tejada I. Update on male erectile dysfunction. *BMJ* 1998;316:678-82. (28 February.)
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Credit cards could be used to indicate availability of cadaver organs for transplantation

EDITOR—There is a willingness in life to donate organs for transplantation after death, but it is the tiny hindrances to translating that willingness into a consent that is legally binding, rather than the availability of cadavers, that has led to severe shortages of organs.

Specific organ donor cards are not easy enough to obtain and carry at all times, so what else, carried by most people most of the time, could double as a donor card? Any signed credit card could both serve its financial function and act as an organ donation card. Lack of consent to organ donation could perhaps be indicated by cutting off a specified corner of the card, until "smart cards" that incorporate medical information become widely available.The current wording for consent needs to be reconsidered to permit elective ventilation before an organ is harvested.[1]

Although spacial and technical problems need to be overcome and card providers need to agree on a common phraseology and mechanism for non-consent, credit cards could radically improve the supply of cadaver organs for transplantation.

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1 Riad H, Nicholls A, Neuberger J, Willatts SM, Sells RA, Jones MA, et al. Elective ventilation of potential organ donors. *BMJ* 1995;310:714-8.

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