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Long-Term Cardio- and Cerebrovascular Events in Patients With Primary Aldosteronism

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Letters

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Data support extended use of levonorgestrel intrauterine systems

EDITOR—The levonorgestrel intrauterine system (Mirena), a useful addition to contraceptive choice, is now approaching its third anniversary.

In the United Kingdom the product licence for the levonorgestrel intrauterine system permits its use for three years. In all other countries where the system is marketed, however, its use is approved for five years. There is good evidence to support this longer duration,¹ and indeed the system seems to remain effective for as long as seven years.² We understand that Schering Healthcare has applied for a two year extension to its current licence, backed by their new data that relates to the new polymer that has been used since 1990.

Many clinicians have asked us if they should conform strictly to the current product licence and replace each intrauterine system on the third anniversary of its insertion. We would strongly advise delaying reinsertion in this time, at least until the expected statement from the Medicines Control Agency.

It is well known that the process of insertion of all intrauterine contraceptives causes problems (short term inconvenience and discomfort followed by new bleeding and pain or expulsion) and also real risks,^{3,4} notably

perforation and upper genital tract infection. These would be unacceptable if it emerges that reinsertions were not necessary.

Users of the levonorgestrel intrauterine system can be reassured that good data are available to support its use for five years. From 15 May, and until any statement concerning a change in the licence, continuation beyond three years is legitimate, at the woman's choice, provided the established criteria for use by named patients are observed—that is, explaining that this is unlicensed use of a licensed product; highlighting the risks and benefits of the proposed non-intervention; obtaining and recording the woman's verbal consent in her case notes; and keeping a separate record of the woman's name and the nature of the unlicensed use.⁵

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Conflict of interest: The Margaret Pyke Memorial Trust is in receipt of research funds from Leiras and from Schering for three ongoing studies of Mirena, and has received research or educational grants from the previous franchisee, Pharmacia and Upjohn. DM and JG received ad hoc consultancy and lecture fees and associated expenses from these and other unrelated pharmaceutical companies.

1 Irving S, El Mahgoub S, McCarthy T, Mishell Jr DR, Shoupe D, Alvarez F, et al. Long term contraception with the Levonorgestrel 20 mcg/day (LNg 20) and the Copper T 380 Ag intrauterine devices: a five year randomised study. *Contraception* 1990;42:361-78.

2 Sivin I, Stern J, Coutinho E, Mattos C, El Mahgoub S, Diaz S, et al. Prolonged intrauterine contraception: a seven year randomised study of the levonorgestrel 20 mcg/day (LNg 20) and the copper T 380 Ag IUDs. *Contraception* 1991;44:473-80.

3 World Health Organisation. *Mechanism of action, safety and efficacy of intrauterine devices. Report of the Who Scientific Group*. Geneva: WHO, 1987. (WHO technical report series 753).

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5 Guillebaud J. Use of licensed products in an unlicensed way. *Contraception today*. 3rd ed. London: Martin Dunitz 1997:94-5.

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during sexual activity may be a weakness of their work.¹ The intensity level of sexual activity is equivalent to that of leisurely walking or strolling,² and an increasing level of energy output, even when amassed during walking, is independently associated with a decreased risk of all cause mortality.³ This failure to adjust for the energy cost of sexual activity may be amplified if, as seems plausible, the more sexually active individuals have a stronger disposition to physical activity per se than their less virile counterparts.

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1 Davey Smith G, Frankel S, Yarnell J. Sex and death: are they related? Findings from the Caerphilly cohort study. *BMJ* 1997;315:1641-5. (20-27 December.)

2 Ainsworth BE, Haskell WL, Leon AR, Jacobs DR, Montoye HJ, Sallis JF, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993;25:71-80.

3 Hakim AA, Petrovitch H, Burchfiel CM, Ross GW, Rodriguez BL, White LR, et al. Effects of walking on mortality among nonsmoking retired men. *N Engl J Med* 1998;338:94-9.

Study did not treat sexual behaviour with the importance it deserves

EDITOR—The article by Davey Smith et al on sexual behaviour and mortality was a disappointment.¹ The topic is important, and the results should at least be correct. I was surprised by the simplistic design, the mechanical interpretation of the results, and the selective use of references—even from the *Holy Bible*.² Although long term follow up studies have to cope with shifts in medical thinking by the time of publication, this should have been addressed in the discussion. Even in the late seventies, during the time of the study's design, psychological understanding of human sexuality was rather more complex than the authors imply. Firstly, not even minimal information on partners or relationships was included. Indeed, marital status was not mentioned and they gave no reason for this, despite numerous published studies on the relation between mortality and marital status before and since the seventies.^{3,4} It is likely that those men who had regular sexual activity in late middle age were either married or in long term relationships. The greater longevity of married compared with unmarried people has been shown repeatedly⁵ and this might be an important confounding factor. Secondly, it was naive to use the term sexual intercourse and orgasm interchangeably. If the authors were mainly interested in the health effect of orgasm, as a purely biological phenomenon, surely masturbation should not have been ignored. Thirdly,

Are sex and death related?

Study failed to adjust for an important confounder

EDITOR—Davey Smith et al report a significant inverse relation between frequency of orgasm and mortality due to all causes and coronary heart disease in men; however, a failure to adjust for the energy expended

it is unlikely that sexual behaviour is static. As Hotopf and Wessely pointed out in their comment, sexual activity is influenced by age, health factors, and psychopathology.¹ To these might be added changes in relationships, loss of spouse etc, which were not addressed, implying that the authors believe one self report measure is adequate to describe a person's sexual activity during an entire lifetime, or at least from the age of 45 to death. Given these shortcomings, the authors should have been more cautious in their conclusions. The only message from this study is that the topic needs further (and more sophisticated) investigation. Inventing new health promotion slogans with numerical imperatives would be premature and should certainly be withheld.

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- 1 Davey Smith G, Frankel S, Yarnell J. Sex and death: are they related? Findings from the Caerphilly cohort study. *BMJ* 1997;315:1641-5. [With commentary by M Hotopf and S Wessely.] (20-27 December.)
- 2 *Holy Bible*. I Corinthians vii, 2-5.
- 3 Berkson J. Mortality and marital status: reflection on the derivation of etiology from statistics. *Am J Public Health* 1962;52:1318-29.
- 4 Goodwin JS, Hunt W, Key R, Samet JM. The effect of marital status on stage, treatment, and survival of cancer patients. *JAMA* 1987;258:3125-30.
- 5 Chandra V, Szklo M, Goldberg R, Tonascia J. The impact of marital status on survival after myocardial infarction: a population based study. *Am J Epidemiol* 1983;117:320-5.

Author's reply

EDITOR—Our report on the association between sexual behaviour and mortality was intended to illustrate the findings that associational epidemiological studies can produce.¹ This information adds to earlier publications on the subject,² which we did make clear in the discussion. The findings have interest beyond this, however, and we would welcome further investigations. We agree with Batty that attention should be paid to physical activity, but conceptual clarity needs to be maintained when this is done. Batty states that we failed to adjust for physical activity, which he considers an important confounder. It is possible that men engaging in lots of sexual activity also participate in other forms of physical activity. Adjusting for energy expended during sexual activity, as suggested by Batty, would not be adjusting for confounding as the physical activity involved in sexual encounters could be the protective factor and, therefore, an integral part of the exposure. Investigation of this issue would be complex. Although Batty explains that the exertion of sexual activity is equivalent to that of leisurely walking or strolling, this would be different for adventurous couplings.

Our article was intended to fit in with the festive cheer of the *BMJ*^{3 4} and to provide an introduction to associational findings in epidemiological studies. Ayton has missed both these points. The majority of our men were married and the results were identical if we restricted the analysis to married men. Our reference to health education was intended to be ironic, not least because the benefits

exceeded those anticipated with most established health promotion programmes. Some readers obviously missed this mild mischief. In future, to spare disappointment at the end of the year perhaps Ayton should avoid the Christmas *BMJ*.

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- 1 Davey Smith G, Frankel S, Yarnell J. Sex and death: are they related? Findings from the Caerphilly cohort study. *BMJ* 1997;315:1641-5. (20-27 December.)
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Cancer in the offspring of radiation workers

Connection between leukaemia and radiation needs to be considered

EDITOR—The results of the study of Draper et al showing an association between worker status of parents and leukaemia in children¹ are unlikely to be attributable to chance. Some exposure to a causative agent, either at the workplace of male radiation workers or in the communities to which they belong, must increase the risk of childhood leukaemia.

Draper et al suggest an oncogenic infectious agent that results from high rates of population mixing as the cause of increased leukaemia risk. Their evidence is based on three factors: an association of population mixing with childhood leukaemia; population mixing in the vicinity of certain nuclear sites; and worker migration between sites. These factors do not explain the excess risk of 80%. A review of all the studies published at that time of childhood leukaemia in areas with high population mixing² gave 50% as best estimate of increased risk, with only one having an estimate as high as 80%.

The effects of population mixing, however, may be diluted in these workers for several reasons. Firstly, only a proportion will have been employed at establishments such as Sellafield. Secondly, the time windows of documented excess mixing near these plants are mostly short relative to the 34 years of this study. Thirdly, radiation worker status of the father before conception of the child is, here, an inaccurate measure of the relevant exposure (which presumably occurs after conception). Nevertheless, given the accumulating evidence that links patterns of exposure to infectious agents with childhood leukaemia,^{2 3} an infectious origin for this excess should be considered.

So, I suggest, should a connection with radiation. Maybe an additional factor that applies to communities of radiation workers has the same effect as population mixing—that is, to favour microepidemics of the putative agent. The possible relevance of immunotoxic properties of ionising radiation in individual people has been proposed.⁴ The

community effect on herd immunity can be much larger than the sum of small individual parts⁵ and could apply here. Immunosuppression of a human population would increase the risk of microepidemics by increasing the probability of infection after a single contact. In addition, for agents that establish latency it would increase the number of infectious people. Mild immunosuppression of the population may be another mechanism in communities that increases the risk of childhood leukaemia.

Case and control fathers¹ should now be linked to National Registry of Radiation Workers records to determine radiation worker status after conception. Also linked, if possible, should be examination of contact between the fathers (employment at the same site at the same time), especially at critical periods such as gestation, the first year of life, and the year preceding diagnosis.

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- 1 Draper GJ, Little MP, Sorahan T, Kinlen LJ, Bunch KJ, Conquest AJ, et al. Cancer in the offspring of radiation workers: a record linkage study. *BMJ* 1997;315:1181-9. (8 November.)
- 2 Kinlen LJ. Epidemiological evidence for an infective basis in childhood leukaemia. *Br J Cancer* 1995;71:1-5.
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Exposure to internal radioisotopes may be responsible

EDITOR—Draper et al find a 1.8-fold excess relative risk for leukaemia in children of male radiation workers and a fivefold relative risk in children of female workers.¹ Since the highest risk does not follow the highest dose they conclude that exposure to radiation is not the cause of the leukaemia and that the Gardner hypothesis is refuted. This conclusion is unwarranted because exposure to internal radioisotopes may be responsible.

Gardner was well aware that the external irradiation studies were at odds with his findings and wrote: "The interpretation [of our findings] in terms of a related causal mechanism is not known... the examination of radionuclides and other occupational exposures is under way."²

Roman et al studied children of radiation workers in west Berkshire and drew different conclusions when they found a 2.2-fold excess risk of leukaemia: "The possibility that the effects could be due to internal contamination by radioactive substances... should be explored."³

This was done by Rooney et al for risk of prostate cancer in nuclear workers.⁴ The excess risk had not followed a simplistic dose-response relation with external film badge doses, and the researchers could have concluded, like Draper et al, that ionising radiation was not the cause of cancer. Instead, they looked at internal contamina-

tion and found significant evidence for enhancement of hazard as a result of internal exposure to a number of isotopes—for example, a relative risk of 14.2 for tritium. This suggests an error of more than 500 times in the current risk of prostate cancer based on Hiroshima.⁵ It is unsafe to assume that the excess risk of leukaemia is any different from the risk of prostate cancer without investigating this further.

In fact, Draper et al's study found an increased relative risk of 2.5 times for radiation workers who had been monitored for internal exposure, compared with 1.6 for those who had not been monitored. If internal radiation is the cause, then the true relative risks are far higher since the controls used by Draper et al were from local populations whose risk of internal contamination is higher than that of the general population.

Finally, Draper et al assert that although the increased risks exist they are small in absolute terms: the risk of 6.5 per 10 000 (an error, presumably they mean per 100 000) increases by 5.4 to 11.9. This means that many children have died because their parents worked in the nuclear industry. No one should be comforted by how low the absolute numbers were.

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- 1 Draper GJ, Little MP, Sorahan T, Kinlen LJ, Bunch KJ, Conquest AJ, et al. Cancer in the offspring of radiation workers: a record linkage study. *BMJ* 1997;315:1181-8. (8 November).
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Combination of antibiotics and non-fatal infections may be responsible for higher number of deaths from leukaemia

EDITOR—Draper et al¹ say that the most likely explanation for the excess of childhood leukaemia at Seascale seems to be infection promoted by population mixing, and they refer their readers to the Kinlen hypothesis.² According to this hypothesis, leukaemia is a rare response to a widespread virus infection that only needs a large influx of people into a previously isolated place to produce an epidemic.

The existence of such viral epidemics is not in dispute. According to the Oxford survey of childhood cancers, however, children who die from leukaemia before they reach the age of 16 are abnormally sensitive to infection throughout their lifespan,³ and, in relation to leukaemia, pneumonia and other serious infections may be either competing causes of death⁴ or promoters of the neoplastic process.⁵

According to the survey, viral epidemics caused by population mixing might reduce

the frequency of leukaemia by adding to the number of children who die before they show any signs of malignant disease. Nowadays, however, antibiotics save lives while non-fatal infections are known to promote cancer. As a result of this combination of factors, epidemics would probably have the opposite effect. The second alternative would not rule out a viral origin for leukaemia, but it offers a much simpler explanation of the Seascale cluster than the one advanced by Kinlen.

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- 1 Draper GJ, Little MP, Sorahan T, Kinlen LJ, Bunch KJ, Conquest AJ, et al. Cancer in the offspring of radiation workers: a record linkage study. *BMJ* 1997;315:1181-8. (8 November).
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- 5 Kneale GW, Stewart AM. Childhood cancers in the UK and their relations to background radiation. *Radiation and Health* 1987;16:203-20.

Authors' reply

EDITOR—Alexander draws attention to the fact that the excess of childhood leukaemia in our study is slightly higher than in most population mixing studies.

A point that deserves to be considered is the difference in design. Previous studies have covered total populations of certain areas, whereas ours concerned the industry in which the mixing was concentrated. It is therefore relevant that a marked excess of leukaemia among the children of men in another (the construction) industry has recently been reported in areas of marked rural population mixing, and it is higher than that in radiation workers.¹ We are not aware of any evidence to suggest that Alexander's immunotoxic hypothesis is relevant to our findings. We agree with the suggestion about examining the links between cases of childhood cancer and radiation workers with exposures after conception; in fact, this has already been under discussion.

Busby and Cato misquote us. Firstly, we did not say that the Gardner hypothesis is refuted, merely that our results do not support it. Secondly, they have changed the tenor of our comment about risks. The issue here is, however, that it is important to present the absolute risks—neither exaggerating the results nor downplaying them—and to avoid the misunderstandings that can occur when only relative risks are quoted for rare diseases. (The denominators for the absolute risks that we quoted are correct.) The possibility that Gardner's results were due to internally deposited radionuclides was considered in detail in the studies by the Health and Safety Executive that we quoted, but no support was found. These and other findings in relation to internally deposited radionuclides are discussed in our paper and in the full report referred to there.

We agree with Stewart that all plausible mechanisms should be considered to explain how any infective agent might influence the incidence of childhood malignancy.

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- 1 Kinlen LJ. High-contact paternal occupations, infection and childhood leukaemia: five studies of unusual population-mixing of adults. *Br J Cancer* 1997;76:1539-45.

Cognitive impairment and survival in very elderly people

Mini-mental state examination may not test cognitive function adequately

EDITOR—Gussekkoo et al hypothesised that mild cognitive impairment as determined by the Dutch version of the mini-mental state examination discriminates subjects with a higher risk of mortality.¹

We have concerns about the validity of using the mini-mental state examination as an adequate test of cognitive function. We have doubts about the reproducibility of test scores if the test is administered by different physicians. In clinical practice the mini-mental state examination is well recognised as being a useful guide rather than an absolute measure of cognitive function; it is a subjective test with variable results.

In particular, we think that defining the scores for mild cognitive impairment and normal cognitive function so close together, at 24-27 and 28-30 respectively, is a potential source of bias. A subject could fall into either category, depending on several variables affecting alertness, such as time of day, timing of drug treatment, stress, and discomfort.

The authors conclude that this test "seems to be useful as a screening instrument for mild cognitive impairment, and its scores may act as an important predictor of survival in very elderly people."¹ Although this conclusion may be valid, we question the usefulness of such a prognostic indicator in clinical practice.

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- 1 Gussekoo J, Westendorp RGJ, Remarque EJ, Lagaay AM, Hereen TJ, Knook DL. Impact of mild cognitive impairment on survival in very elderly people: cohort study. *BMJ* 1997;315:1053-4. (25 October).

Decreased survival with cognitive impairment seems not to be related to comorbidity

EDITOR—Gussekloo et al showed an increased mortality with increasing cognitive impairment in a population of very old subjects.¹ Lower cognitive status in elderly people may also be due to chronic disease, and the risk of death might be entirely due to concurrent chronic diseases rather than to dementia. We investigated the effect of the number of diseases and their severity on mental state and survival in elderly patients.

We obtained data from the evaluation of 650 consecutive elderly patients on admission to a geriatric evaluation and rehabilitation unit over a period of 14 months. A standard protocol was used to assess demographic factors, mental and functional state, and somatic health. Somatic health was evaluated as the presence of 15 of the most common diseases in elderly patients in hospital and their severity. Severity was evaluated by Greenfield's individual disease severity index, in which each condition was graded from 0 (no disease) to 4 (greatest severity of disease).² We excluded patients with terminal or wasting disease (renal or liver failure, malignant neoplasm, diabetes, or chronic inflammatory diseases).

Our population (mean age 79.2 (SD 7.4) years; 456 women (70.2%)) had moderate functional impairment (mean of 2.8 (2.0) functions lost³), mild depression (mean general depression score 13.0 (6.3)⁴), and many diseases (mean 5.1 (2.7)). Overall, 113 patients died within 12 months after discharge (annual death rate 17.4%).

Like Gussekloo et al, we identified four subgroups with decreasing cognitive status by using the mini-mental state examination. The table shows the crude risk of death in Cox's proportional hazard survival analysis. The severity of heart disease of ischaemic or organic origin, heart disease of other origin, respiratory disease, anaemia, malignancy, and renal disease was independently associated with mortality; disability and serum albumin concentration were also associated with greater mortality (data not shown). The table also shows that the association between cognitive impairment and mortality held after all confounders associated with mortality in the bivariate Cox's model were controlled for—that is, age, sex, severity of diseases, disability, and serum albumin concentration.

Gussekloo et al state that mental state may act as an important predictor of

survival in elderly people. Although we did not find a significant association between mild cognitive impairment and mortality (relative risk 1.8 (95% confidence interval 0.7 to 4.8)), our data are in line with their conclusions supporting the need to assess mental state to predict the survival of very elderly people.

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- 1 Gussekloo J, Westendorp RGJ, Remarque EJ, Lagaay AM, Heeren TJ, Knook DL. Impact of mild cognitive impairment on survival in very elderly people: cohort study. *BMJ* 1997;315:1053-4. (25 October.)
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- 5 Folstein MF, Folstein SE, McHugh PR. Mini-mental state: a practical method for grading cognitive state of patients for the clinician. *J Psychiatr Res* 1975;12:189-98.

Authors' reply

EDITOR—The mini-mental state examination is widely used to screen for cognitive impairment in (very) elderly people. We emphasise that 40% of the participants of the Leiden 85-plus study had optimal scores of 28-30 points.¹ As we described earlier, elderly subjects with test scores of 28-30 points tend to have equal scores over a period of three years.² In contrast, subjects with borderline scores of 24-27 points tend to have lower scores over a similar period of time. In our paper we showed that subjects with borderline test scores have a higher risk of mortality.

Whether these subtle differences in scores on the mini-mental state examination are due to differences in cognition is debatable. However, test scores of 24-27 points and of 28-30 points undoubtedly distinguish different people. These findings are confirmed by the observations of Rozzini et al. Their data suggest that the increased risk of mortality in subjects with borderline test scores is not due to comorbidity. The risk estimates were not materially different after adjustment for concurrent chronic diseases diagnosed in hospital. These observations strengthen the role of mental state as an

important risk factor for mortality. Taken together, we can conclude that the mini-mental state examination is a valid instrument to differentiate subjects at higher risk at a cut off score of 27 points.

Forster et al doubt whether the mini-mental state examination has similar predictive properties when used by doctors in clinical practice instead of the two researchers in the Leiden 85-plus study. We are convinced that scores in the mini-mental state examination are reproducible when the instructions of the test are taken seriously.³ Indeed, the predictive value of a score for one subject has to be weighted differently from the predictive value of scores for a group of subjects, but this dilemma holds for every instrument used to screen for subjects at high risk.⁴

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- 1 Heeren TJ, Lagaay AM, van Beek WCA, Rooijmans HGM, Hijmans W. Reference values for the mini-mental state examination (MMSE) in octo- and nonagenarians. *J Am Geriatr Soc* 1990;38:1093-6.
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Medical students may not like statistics, but as doctors they will

EDITOR—In his book review, Wessely finds Sinclair's analysis of medical education in *Making Doctors* depressing because Sinclair reports that medical students regard the psychiatrist as "the lowest form of medical life."¹ Further, they do this because of the very things of which Wessely is rightly proud: psychiatrists' use of a questioning, multidisciplinary, evidence based, patient centred approach.

Wesseley was shocked to read that "statistics is above all the subject most disliked by students."¹ This is no surprise to those of us given the task of teaching it to them. However, statistics teachers can console themselves with the thought that this dislike will not always be there, and that as these doctors progress through their careers they will find statistics of ever increasing relevance and importance. The *BMJ* shows this clearly in the research it publishes, in the large number of statistical articles it carries, and in its statistical refereeing.

Crude and adjusted associations of score on mini-mental state examination with mortality after 12 months in 650 elderly patients

Score	Proportion of deaths	Crude relative risk (95% CI)*	Adjusted relative risk (95% CI)†‡
28-30	5/94	1.0	1.0
24-27	18/194	1.8 (0.7 to 4.9)	1.8 (0.7 to 4.8)
19-23	25/131	3.8 (1.5 to 10.0)	2.6 (1.0 to 6.9)
0-18	65/231	6.2 (2.5 to 15.4)	3.6 (1.4 to 9.2)

*P<0.0001 in test for trend with decreasing score.
†Adjusted for age, sex, severity of disease (ischaemic or organic heart disease, heart disease of other origin, respiratory disease, kidney diseases, anaemia, and malignancy), serum albumin concentration, and disability.
‡P=0.003 in test for trend with decreasing score.

When I was a student, the attitude to my subject was: "Here's to pure mathematics, may it never be any use to anybody." The ultimate goal, now achieved, was the proof of Fermat's last theorem. I soon learnt that this would not take me far in the outside world and that being of use to people, and to doctors in particular, was far more rewarding. In the same way, Sinclair's medical students will learn that not all problems can be solved with a scalpel.

We continue to grow after leaving full time education. Given what Sinclair tells us, this is just as well.

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1 Wessely S. *Medicine and books: Making doctors: an institutional apprenticeship.* *BMJ* 1998;316:713. (28 February.)

Effect of moderate alcohol consumption on Lp(a) lipoprotein concentrations

Reduction is supported by other studies

EDITOR—We agree with Paassilta et al that there may be a relation between moderate alcohol consumption and lower Lp(a) lipoprotein concentrations.¹ The relation between alcohol consumption and cardiovascular mortality is U shaped, with the lowest mortality at an alcohol consumption of 2-4 units (16-32 g) a day.² Several mechanisms contribute to this cardioprotective effect including beneficial increases in high density lipoprotein cholesterol³ and inhibition of platelet aggregation.⁴ However, other factors may be involved. Lp(a) lipoprotein is a recognised independent risk factor for the development of atherosclerosis and, as stated by Paassilta et al, little attention has been directed to the effects of alcohol on Lp(a) lipoprotein.

In 1995 we reported a significant reduction in Lp(a) lipoprotein concentration in a prospective study of 20 healthy volunteers (men and women) given 21 g of alcohol daily for 10 days in the form of red wine (median (range) 186 (15-1420) mg/l v 132 (10-1210) mg/l, $P < 0.001$).⁵ This reduction was not repeated when the same subjects were given white wine, raising the issue of potential differences between various alcoholic drinks. Interestingly, we found no changes in high density lipoprotein cholesterol concentrations.

We have conducted a larger unpublished crossover trial in 50 men comparing the effects of 3 units (24 g) of alcohol a day as red wine or vodka for 14 days on Lp(a) lipoprotein concentrations. Each period of alcohol consumption was preceded by two weeks' abstinence. Both drinks produced a 10-12% decrease in Lp(a) concentration (geometric mean 153 mg/100 ml v 135 mg/l after vodka, $P < 0.001$; 151 mg/l v 136 mg/l after red wine, $P < 0.01$). These results suggest that moderate alcohol consumption results in changes in Lp(a)

lipoprotein which are independent of the type of alcoholic drink consumed. In conclusion, we agree with Paassilta et al that lower Lp(a) concentrations may be one factor conferring lower mortality and cardiovascular benefit in social drinkers.

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- 1 Paassilta M, Kervinen K, Rantala AO, Savolainen MJ, Lilja M, Reunanen A, et al. Social alcohol consumption and low Lp(a) lipoprotein concentrations in middle aged Finnish men: population based study. *BMJ* 1998;316:594-5. (21 February.)
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No effect seen in Australian drinkers

EDITOR—Paassilta et al suggest that a moderate intake of alcohol in Finnish men is associated with a roughly 50% reduction in median Lp(a) lipoprotein concentration.¹ We have published relevant data from a large Australian cohort participating in an ongoing prospective study of cardiovascular disease (2805 subjects ≥ 60 years, average age 70 years).^{2,3}

Current alcohol intake was assessed by personal interview and classified as nil, 10-70 g/week, 80-140 g/week, 150-280 g/week, and > 280 g/week (the last group in men only). The third and fourth groups correspond most closely with the middle and highest third of alcohol intake described by Paassilta et al.¹ Lp(a) lipoprotein concentration was assessed by a sandwich enzyme linked immunosorbent assay (ELISA) with polyclonal sheep antibody raised against purified human apo(a) (TintElize Lp(a) Biopool, Sweden). The table shows median (interquartile range) Lp(a) lipoprotein and mean high density lipoprotein cholesterol concentrations by sex and alcohol intake.

There was no significant relation between Lp(a) lipoprotein concentration and alcohol intake in either sex. We drew

similar conclusions when the data were examined in those below or above 70 years of age. The usual positive relation between alcohol intake and high density lipoprotein cholesterol was confirmed. Paassilta et al examined only 259 men aged 40-60 years. The median Lp(a) lipoprotein concentration in 37 teetotallers seemed high at 206 mg/l, and this may have been a spurious result. Surprisingly, they found no relation between alcohol intake and high density lipoprotein cholesterol concentration. Though we have reported that any alcohol intake in our cohort is associated with reduced coronary risk³ and raised Lp(a) lipoprotein concentration is associated with increased coronary risk,² any link between alcohol intake and Lp(a) lipoprotein concentration seems unlikely.

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- 1 Paassilta M, Kervinen K, Rantala AO, Savolainen MJ, Lilja M, Reunanen A, et al. Social alcohol consumption and low Lp(a) lipoprotein concentrations in middle aged Finnish men: population based study. *BMJ* 1998;316:594-5. (21 February.)
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Reduction is not found in women

EDITOR—Paassilta et al showed a quantitative association between social alcohol consumption and low Lp(a) lipoprotein concentrations in middle aged men.¹ As they indicated, we reported a negative qualitative association between drinking habits and Lp(a) lipoprotein concentrations in men.² We report here the results of a quantitative analysis of the association between alcohol intake and Lp(a) lipoprotein concentration in the Jichi Medical School cohort study. During 1992-5 we collected population based data in rural districts in Japan. The 9532 subjects (3658 men and 5874 women), which included all subjects in our previous report, were divided into five categories by drinking status; non-drinkers, (963 men), former drinkers (140 men), drinkers in the lowest third of alcohol intake (< 107 g/week; 711 men), drinkers in the middle third (107-224 g/week; 942 men), and drinkers in the highest third (> 224 g/week; 902 men). Serum Lp(a) lipoprotein concentrations were measured with an enzyme linked

Median (interquartile range) Lp(a) lipoprotein and mean high density lipoprotein (HDL) cholesterol concentrations by sex and alcohol intake

Alcohol consumption (g/day)	Men			Women		
	No	Lp(a) lipoprotein (mg/l)	HDL cholesterol (mmol/l)	No	Lp(a) lipoprotein (mg/l)	HDL cholesterol (mmol/l)
0	262	90 (40-255)	1.12	739	115 (50-275)	1.37
10-70	425	100 (40-240)	1.16	570	125 (60-310)	1.45
80-140	266	103 (45-280)	1.29	197	125 (45-310)	1.62
150-280	160	88 (45-230)	1.39	42	125 (65-260)	1.70
> 280	97	85 (35-205)	1.42	-	-	-

Mean atherosclerotic risk factors by alcohol consumption for men in the Jichi Medical School study, 1992-5

	Non-drinker		Former drinker		<107 g/week		107-224 g/week		>224 g/week		P value†
	No	Mean	No	Mean	No	Mean	No	Mean	No	Mean	
Age (years)	963	57.5	140	61.3	711	54.8	942	53.9	902	54.9	<0.0001
Body mass index (kg/m ²)	958	22.9	138	23.1	703	23.1	941	22.9	898	22.9	0.74
Lp(a) lipoprotein (mg/l)*	963	135	140	120	711	129	942	128	902	107	<0.0001
Fibrinogen (g/l)	570	2.46	97	2.55	530	2.42	713	2.42	643	2.38	<0.05

*Geometric means were calculated for Lp(a).

†P values for variables except age were calculated after adjustment for age by analysis of covariance.

immunosorbent assay (ELISA) and Lp(a) lipoprotein and triglyceride concentrations were calculated as geometric means. We used SAS software for statistical analysis.

The groups were similar regarding body mass index and total cholesterol concentrations. The mean age of former drinkers was higher than that of men in the other groups (analysis of variance $P < 0.0001$). We therefore adjusted for age in all other analyses. Lp(a) lipoprotein concentration decreased with increased alcohol consumption in men except in former drinkers (table). Fibrinogen concentrations fell with increasing alcohol consumption ($P < 0.05$).

For women the geometric mean Lp(a) concentrations were 148 mg/l in non-drinkers, 144 mg/l in former drinkers, 144 mg/l in lowest third, 136 mg/l in middle third, and 138 mg/l in highest third. There was no significant association between Lp(a) lipoprotein concentration and alcohol consumption ($P = 0.27$). Fibrinogen concentrations decreased with increasing consumption ($P < 0.0001$).

Our analysis shows a negative relation between alcohol consumption and Lp(a) lipoprotein concentrations in men but not in women. These results support Paasilta et al's study and confirm our previous qualitative analysis.

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retrieved in a Medline search are irrelevant.³ Evaluated databases such as the Cochrane database and Best Evidence are more helpful to me than the raw, undigested research that is available from Medline, but even they are not concise enough to use easily to answer a particular clinical question.

Slawson and Shaughnessy suggested that doctors are seeking "patient oriented evidence that matters (POEM)."⁴ When faced with a clinical question I would like information NUGGETS. I want information Now, Up to date, Grounded in Good Evidence, There (on my desk), and Specific to the problem. Where can I find a system that will deliver information NUGGETS?

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1 Osheroff JA, Bankowitz RA. Physicians' use of computer software in answering clinical questions. *Bull Med Libr Assoc* 1993;81:11-9.

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3 Haynes RB, McKibbon A, Walker CJ, Ryan N, Fitzgerald D, Ramsden MF. Online access to Medline in clinical settings. *Ann Int Med* 1990;112:78-84.

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Interventions to treat shoulder pain

Review was overly negative

EDITOR—The systematic review by Green and colleagues of interventions for treating shoulder pain concluded that there is little evidence to support the use of any of the common interventions for the management of shoulder pain.¹ This is a negative message that is likely to inhibit practitioners from treating patients with shoulder pain and to dissuade them from referring these patients to specialists. While we agree with the other conclusions of the study, we disagree with the negative message about treatment for several reasons.

Rheumatologists make decisions about the treatment of musculoskeletal disorders such as shoulder pain based on the duration of the condition, its severity, and a careful examination to define the exact site of the lesion.² It is generally taught, for example, that the injection of corticosteroids will only work if done soon after the onset of any shoulder disorder and if the injection has been precisely localised to the anatomical site of the problem, such as a specific tendon

or bursa within the rotator cuff.³ In the systematic review of the evidence great weight was given to the quality of the studies, but no weight was given to the quality of the clinical input to the studies, or to any of the three clinical criteria described above.

We would agree that the assessment of the value of treatments for shoulder pain is difficult because of the overall poor quality of published studies, the absence of agreed outcome measures, and a lack of uniformity of definitions; some recent rheumatological research has addressed the problem of definitions.⁴ However, the methods used to assess quality in this review are not transparent or validated, and it is unclear on what basis the authors chose to combine studies; this throws into question the validity of their conclusions.

Furthermore, if, as the authors of this and other reviews of interventions to treat shoulder pain contend, the methodological scores for most studies are low, surely it is erroneous to conclude that treatments do not work. The poor quality of the evidence discredits the negative conclusions as well as the positive ones.

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1 Green S, Buchbinder R, Glazier R, Forbes A. Systematic review of randomised controlled trials of interventions for painful shoulder: selection criteria, outcome assessment, and efficacy. *BMJ* 1998;316:354-60. (31 January.)

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Lack of concordance between rheumatologists may render multicentre studies invalid

EDITOR—As Green et al point out the management of shoulder pain is bedevilled by imprecise diagnostic criteria.¹ It is likely that many trials that examine the efficacy of treatment are flawed because treatment has been directed at the wrong part of the shoulder mechanism.

As there are only three joints and two main tendons in the shoulder it ought not be too difficult to agree a simple set of

Information "Nuggets" are not easy to find quickly

EDITOR—An essential ingredient in the practice of evidence based medicine is the evidence, but where can it be found? My recent experience using Medline and Embase suggests that these databases are not the place for a busy general practitioner to seek evidence with which to answer clinical questions. Only 40%-46% of a physician's clinical questions can be answered using Medline,^{1,2} and many of the citations

diagnoses with appropriate signs. But this is not enough. Our own study showed that three consultant rheumatologists who examined the same patients disagreed on the precise diagnosis in over 50% (14/26) of the cases, and when they examined a second group of patients together (so that they agreed on the clinical signs) they still disagreed in nearly 20% (4/18) of the cases.²

Such a lack of concordance among clinicians who are experienced in the management of shoulder problems will render any multicentre study invalid, and may leave any study by an individual clinician open to doubt or criticism.

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- Green S, Buchbinder R, Glazier R, Forbes A. Systematic review of randomised controlled trials of interventions for painful shoulder: selection criteria, outcome assessment, and efficacy. *BMJ* 1998;316:354-60. (31 January)
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Authors' reply

EDITOR—Szebenyi and Dieppe have adopted an overly pessimistic reaction to our paper by misquoting our conclusions. The abstract and the further research section clearly state that “there is little evidence to either support or refute the efficacy of common interventions for shoulder pain.”

The suggestion that careful clinical assessment will identify the problem is questionable. As Bamji points out, one of the important findings of our study (and his own), is a lack of concordance in the labelling and defining of shoulder disorders. This presents difficulties for those performing clinical trials as well as for clinicians who rely on the results to manage their patients. It is also of concern for those who use databases to make inferences about these conditions.¹ The need for formal development and universal adoption of valid and reliable criteria for classifying shoulder disorders has been highlighted previously.²

Teaching students that the injection of corticosteroids will only work if done shortly after the onset of symptoms and if the injection has been precisely localised does not seem to be based on any evidence. One problem is the inability to precisely localise the injection. Previous studies have suggested that as many as 60% of shoulder injections thought to be intra-articular may not be.^{3,4}

The methods we used to assess methodological quality have been well validated and are described in the reference cited in the review.⁵ Complete details will shortly be available from the Cochrane Library Musculoskeletal Group. The overall score of methodological quality was calculated based on an assessment of the study population (selection criteria, randomisation, comparable control group, and complete follow up), description of the intervention, measurement of the effect (blinding of patient

and assessor, outcome measures), and data analysis (including complete presentation of results and consideration of sample size). The criteria used to combine the studies is specified in the methods section. Trials considered for pooling were those in which the same intervention was used in similar populations and in which the same outcome measure was used at similar lengths of follow up.

Further clinical trials are needed to determine optimal treatment strategies for shoulder pain. While such trials have the potential to be bedevilled by problems of diagnostic criteria, unlike Bamji we believe that careful consideration of selection criteria and outcome measures are not insurmountable challenges.

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Suspension of consultant raises serious issues

EDITOR—The recent news item¹ and the subsequent letter and correction² about the hearing of Dr James Taylor before the General Medical Council have not taken account of some serious issues raised by this case, which will affect interventional physicians, cardiologists, and radiologists as well as obstetricians, surgeons, and others.

The accusation had been about consent, not of medical negligence. Negligence forms no part of the hearing. At issue was the extent to which consent is covered in the standard consent form used in NHS hospitals. After lengthy debate by counsel both for the GMC and Dr Taylor, the GMC's legal adviser omitted the strong legal challenge by defending counsel. He relied on his interpretation of two previous, distinctly different, cases reviewed by the Privy Council.

It was accepted that “consent” may not be required if the medical action was “necessary, in the child's best interest, and medically justified.” However, the GMC

panel ruled that “necessary” meant “immediately life-threatening.” It came to this conclusion on the basis of a “Guide to Consent” published by the NHS in 1991, and of legal precedents, which concerned adult patients of unsound or immature mind. The unanimous expert evidence in the present case was that the procedure undertaken had been fully justified, a crucial point totally ignored in the GMC's decision.

In all its hearings the GMC panel acts as both jury and judge—the strictly legal part being in the hands of its legal adviser, who pronounces on legal issues and precedent. His opinion is not questioned by the panel and is not open to challenge. This adviser is part of the panel—that is, not an independent judge.

Decisions at these hearings as to fact should be proved “beyond reasonable doubt.” Yet these proceedings were conducted in a manner that seemed to require the defendant to prove himself to be innocent. The finding of “guilty of serious professional conduct” seems to go against the legitimate and admitted doubt concerning the implications of the consent. If the GMC wished to clarify consent, it could do so by issuing firm regulations (which it has so far failed to do), rather than by penalising one doctor.

Siding with the parents on the issue of consent, the GMC may make it impossible for doctors caring for children to act “first and always” (as the motto of the Great Ormond Street children's hospital states) in the interest of the child, whenever an unexpected and urgent procedure ought to be performed.

As matters now stand, doctors may be prevented from acting in the patient's best interest when the unforeseen arises and requires immediate attention.

There is no appeal to the GMC's decision, other than to the Privy Council. Should there not be an appeals procedure within the GMC?

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- Dyer, C. Consultant suspended for not getting consent for cardiac procedure. *BMJ* 1998;316:955. (28 March.)
- Goodwin JF, Somerville W. Punishment of doctors must fit their crime [correction appears in *BMJ* 1998;316:1677]. *BMJ* 1998;316:1248. (18 April.)

Correction

Punishment of doctors must fit their crime

An authors' error occurred in this letter by Goodwin and Somerville. At the hearing the parents, both in their evidence and through their counsel, accused Dr Taylor not only of having acted without consent but also of actually having proceeded against their (allegedly previously expressed) wish. This part of their action, however, failed (contrary to the statement made in Goodwin and Somerville's letter).