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Lottery of NHS funding is inappropriate

EDITOR—From time to time, additional funds are made available to the NHS—for example, for cancer care or for new chest pain clinics. The distribution of these funds lacks openness, lacks accountability, and does not give good value for money.

When extra cash is made available, bids are invited within a tight time frame, which at times seems to be driven more by media pressure and political expediency than by local health needs. Typically, trusts have only a few weeks to prepare complex bids that require financial, clinical, and technical skill and knowledge of staffing. Rushed consultation and planning inevitably lead to unreal expectations, delays in implementation, and failure to deliver planned benefits.

The short timetable also works against the principle of accountability as the trust board may be informed of a bid only retrospectively. Recently, one regional office allowed 10 working days for trusts to bid for considerable resources for major redevelopment of accident and emergency departments; within five working days it had announced the successful bids. Non-executive directors of trusts, unused to the ways of the NHS, are often astonished at the

way such large sums are distributed. Since there is little or no feedback there is little confidence that bids have been fairly or thoroughly evaluated.

Occasionally, trusts are told before submission that a bid is likely to be successful; this again raises concerns about the fairness of assessment of the bids. Further examples illustrate other concerns. Recently, funding became available for additional intensive care beds, one of the criteria being that beds must be operational by a specific date. Since intensive care nurses are not available nationwide, this has resulted in the transfer of senior nurses from acute services, effectively depleting other departments. Similar difficulties have occurred with the development of new chest pain clinics, whose introduction has come at the expense of other medical patients.

We submit that the current arrangements have many problems: the time frame is inadequate, local priorities are not met, inadequately prepared bids are submitted, evaluation of the bids is neither thorough nor open, and the current arrangements are not giving best value for money. The system that hands down large sums of public money annually seems to be inappropriate in a modern NHS.

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Doctors should not advise adolescents to abstain from sex

EDITOR—In recent years rates of teenage pregnancy and abortions, and the prevalence of sexually transmitted diseases have increased in Belgium and other European countries.¹ Against this background Stammers and Ingham considered whether advising should be an effective response to declining sexual health in teenagers.² Both advocated their opinion with valid arguments, but there is insufficient evidence to justify one of the two opinions.

To determine the needs and expectations of adolescent girls concerning contraceptive use as well as their attitude to healthcare providers we conducted qualitative research with focus groups of 17 year old girls.³ Afterwards a survey conducted

among more than 700 adolescents in schools in Antwerp confirmed the results. The girls' knowledge concerning the daily use and side effects of contraceptives was insufficient. Most of them had a good relationship with their parents, especially their mothers. Nearly 50% of the girls preferred to talk to their mother about contraceptives and sexual health.

Wellings et al also described an increased proportion of girls citing parents as the main source of information.⁴ Other important sources of information are female friends, sisters, and doctors. General practitioners especially play an important part in giving information about the use of contraceptives.

More sexually experienced girls following their mothers' advice used oral contraceptives when they had their first sexual intercourse than girls who did not seek advice at home (55% v 30%, $\chi^2 = 15.71$; $P < 0.005$). Girls who did not seek advice at home displayed unsafe contraceptive behaviour (17% v 9%), and used more emergency contraception (morning after pills) (69% v 31%, $\chi^2 = 4.15$; $P < 0.05$). In both groups, 67% of girls used condoms. Those who followed their mothers' advice consulted gynaecologists more often (22% v 14%, $\chi^2 = 10.93$; $P < 0.025$). Young et al also found that parents play an important part in communication about sexual behaviour.⁵

Healthcare providers should not directly advise adolescents to abstain from sex. They can encourage girls to talk to their parents. Besides, adolescents want an open approach to sexual conduct. In our survey more than 70% of adolescents give a score of 8 or more on a visual analogue scale for the following attitudes from their doctor: the doctor is serene, listening to me, taking time, or showing respect. S/he is answering my questions, but only those I want to discuss. Sexually transmitted diseases and relationships are subjects adolescents prefer to discuss with their parents and friends. Our research supports an open approach and better communication of healthcare providers and parents.

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Emergency contraception from pharmacists misses opportunity

EDITOR—I hope that the predicted reduction in unplanned pregnancies and abortions will result from pharmacy access for emergency contraception, as described by Harrison-Woolrych et al in their editorial.¹ The possibility of risk displacement, however, rendering this move ineffective in the same way as has been postulated for condom use² makes this far from certain. What is certain is that the opportunity for detection of sexually transmitted diseases and reduction of risk has been missed.

A tunnel vision approach to reducing unplanned pregnancy may do nothing to reduce the risk of sexually transmitted diseases and can increase it.³ Many, if not most, women in need of emergency contraception will also be at risk of sexually transmitted diseases. If, in taking a history to explore the need for emergency contraception, a doctor did not also gently explore the risk of sexually transmitted diseases and advise the patient appropriately, I would consider it substandard practice and possibly even negligent. The website pharmacy training programme highlighted in the editorial mentions sexually transmitted diseases only in the context of emergency contraception, not in terms of providing any protection against them. No questions are advised to assess the risk of sexually transmitted diseases, and no information is to be provided on how to obtain further help on diagnosis and management. Even mandatory provision of a simple leaflet mentioning possible risk of sexually transmitted diseases and giving the details of the nearest departments for genitourinary medicine would be better than nothing.

As it is, a woman who obtains emergency contraception from a pharmacy is unlikely to be offered any chance whatsoever of having a concurrent sexually transmitted disease investigated and treated promptly. Rates of sexually transmitted diseases will continue to increase in the United Kingdom yet again as a predictable and direct result of a scheme introduced with insufficient planning and training for pharmacists. When concerns about sexually transmitted diseases were raised by some of those involved in the Manchester pilot scheme, they were simply ignored. The trauma of a diagnosis of chlamydia infection is clearly of little interest to those who want to make buying emergency contraception as easy as buying a toothbrush.⁴

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- 1 Harrison-Woolrych M, Duncan A, Howe J, Smith C. Improving access to emergency contraception. *BMJ* 2001;322:186-7. (27 January.)
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Psychosocial impacts of chlamydia testing are important

EDITOR—The article by Duncan et al about the psychosocial impact of diagnosis of chlamydia infection has identified important issues that need to be considered before implementation of a national screening programme as described in the chief medical officer's report.^{1 2}

We conducted a cross sectional study in a local estate in Nottingham last year to measure knowledge and attitudes of women aged under 25 years attending a family planning clinic before and after a targeted campaign. Selective screening for *Chlamydia trachomatis* was undertaken by using urine testing by a commercial DNA amplification method. Follow up interviews were held at six months to examine attitudes to the result, results of contact tracing, and implications for the patient.

The response rate for the questionnaire was 100% (n=180). Awareness before the targeted chlamydia campaign was reported by 50% of the women. There was an increase in awareness of chlamydia after the campaign, with attendees aged under 20 reporting an 11% increase and attendees aged over 20 reporting a 7% increase in awareness. Female patients aged under 16 were at an increased risk of chlamydia owing to reported sex without a condom ($\chi^2=4.59$, $P=0.03$) and suspecting their partner was having sex with others as well as themselves ($\chi^2=6.74$, $P=0.01$) compared with older attendees. Sixty five women were screened for chlamydia; of these four (6%) had positive test results, and all of them were treated and helped with partner notification. The four women were re-tested and interviewed six months later, although they all had a negative test result, the psychological effects of testing positive for a chlamydia infection were evident.

The women's responses to the positive test result for the chlamydia infection were shock and worry, unhappiness, embarrassment, and surprise. Three of the women felt embarrassed about the need to trace contacts. None the less, two of the girls self referred their partner, one requested provider referral, and one was unable to contact her partners because they were out of the country. All four of the women were worried about the long term effects of chlamydia if left untreated. One was so concerned about the possible inability to conceive in the future, that when she returned for the six month follow up chlamydia test, she also

requested a pregnancy test as she and her partner were trying to conceive. As discussed by Duncan et al in their article, our study, although small, would confirm this is a real issue that necessitates further development and discussion with girls before they are tested for chlamydia infection.

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2 Chief Medical Officer. *Main report of the CMO's expert advisory group on Chlamydia trachomatis*. London: Department of Health, 1998.

Glycaemia and vascular effects of type 2 diabetes

Lowering glucose concentrations may not be of any value in itself

EDITOR—Stratton et al have documented that as glycaemic exposure increases, diabetic complications increase.¹ They conclude that treatment of hyperglycemia will have substantial benefit, a conclusion reiterated by Tuomilehto.² Yet reduction of glycaemic exposure did not have such benefit in the UK prospective diabetes study (UKPDS) randomised trial.^{3 4} The data by Stratton et al suggest that reducing mean haemoglobin A_{1c} concentration by 1% would reduce diabetes related deaths by 21%. Intensive treatment of hyperglycaemia for 10 years in UKPDS reduced haemoglobin A_{1c} by nearly 1% (from 7.9% to 7.0%) yet did not reduce diabetes related deaths significantly.

The conventionally treated group, with greater glycaemic exposure, experienced diabetes related death at a rate of 11.5 deaths per 1000 person years. On the basis of the data by Stratton et al, the intensively treated group should have experienced diabetes related death at a rate of 9.0 deaths per 1000 person years. Intensive treatment was, however, associated with only a non-significant decrease in diabetes related mortality.⁴ Similarly, the data by Stratton et al suggest that intensive treatment would result in significant reductions in adverse outcomes that include all cause mortality, stroke, myocardial infarction, and amputation. Reducing haemoglobin A_{1c} by nearly 1% in the UKPDS, however, was not associated with significant reductions in any of these adverse outcomes.

Treatment that significantly improves glycaemic control therefore does not achieve the predicted benefit. Does this mean that greater glycaemic exposure is a marker for adverse outcomes but not a cause? This would imply that the higher the haemoglobin A_{1c} concentration the more attention needs to be paid to non-glycaemic treatment of diabetic patients, such as controlling blood pressure. Or does it mean that the treatments currently available to lower glucose harm diabetic patients as much as the lowering of blood glucose helps them? McCormack and Greenhalgh may be correct when they say that treatment with metformin improves outcomes in diabetic patients, not necessarily resulting from its glucose lowering effect, but that lowering glucose concentrations in itself is of little to no value in type 2 diabetes.³

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- 1 Stratton IM, Adler AI, Neil AW, Matthews DR, Manley SE, Cull CA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. *BMJ* 2000;321:405-12. (12 August.)
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UKPDS is not a cohort study and analysis is misleading

EDITOR—In their two papers the UK prospective diabetes study (UKPDS) investigators potentially render their analyses invalid.^{1,2} UKPDS was subrandomised into many smaller comparisons with limited sample sizes in the subgroups. The trial provided unique data on treatment outcomes for type 2 diabetes where there had previously been virtually no evidence. It cannot be repackaged as an observational or

epidemiological study by ignoring its design to test treatments.

In the paper by Stratton et al, the major treatments are hardly mentioned and not accounted for in the main analyses.¹ If Stratton et al are conceding that the main glycaemic interventions were ineffective, both on large vessel disease and mortality, and of limited impact on microvascular outcomes if the hypertension factorial component is excluded, then perhaps the pooling of intensive and conventional treatments would be legitimate. That concession seems unlikely, although that is the major result from a meta-analysis.³ So a multiple treatment term would at the very least be required in the observational analysis. Was this tried or was it not significant? Similarly, interaction terms (for example, the effective metformin-obesity arm) would be needed. In most cohort studies, subjects are only included when clinically free of the end points of interest. But in UKPDS over 30% of patients already had target organ signs or damage. What about the impact of weight gain in the analysis (some 2.5-4 kg after taking sulphonylureas and 7 kg after taking intensive insulin over the median 10 years of the trial)?

A comparison of impacts on absolute rather than relative risks in the main trial arms may be useful (table), because detailed numbers needed for treatment to benefit or harm can be calculated. These show the slight absolute impact on any end point achieved by intensive glycaemic interventions with currently available treatment. They have not been discussed by the UKPDS trialists, in promoting results of intensive over merely good glycaemic control that have been packaged as very good news for type 2 diabetes patients but are of limited, if any, added benefit.

In the paper by Adler et al on the impact of systolic blood pressure, antihypertensive treatment was effective in that arm of the trial. A treatment term is adjusted for in the analysis. The complex factorial design where only those with blood pressures ≥ 160 or 90 mm Hg were eligible for that arm of

treatment renders such adjustment too simplistic, probably underestimating the impact of blood pressure, and illegitimate because of allocation to several treatments. The only appropriate analysis would be of the patients not randomised to the hypertension “tight control” arm.

Both these papers present erroneous data, which might have been filtered at the refereeing stage. Withdrawal might be considered.

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- 1 Stratton IM, Adler AI, Neil AW, Matthews DR, Manley SE, Cull CA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. *BMJ* 2000;321:405-12. (12 August.)
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Relation between diabetes and hyperglycaemia and cardiovascular disease has not been resolved

EDITOR—Stratton et al show a relation between glycaemia (measured as glycated haemoglobin) and mortality and various morbid events in patients with type 2 diabetes.¹ The statistical independence of this relation is, however, open to doubt. In multivariate analyses the precision of a variable can affect the outcome. In the UK prospective diabetes study (UKPDS) report, glycaemia is the average of multiple measurements—and is therefore a comparatively precise estimate of the individual's state—while potentially confounding variables are, for the most part, single measures at baseline. In the accompanying paper, by contrast, it is blood pressure that is the precise measure.² Incidentally, given that

Comparison of significant absolute risk reduction (ARR) and numbers needed to treat (NNT, or reciprocal of absolute risk reduction) for different arms of UK prospective diabetes study (UKPDS)

End point	Main intensive glycaemic control trial				Metformin subtrial				Hypertension treatment trial			
	Rate per 1000 person years (No affected)			NNT per year (95% CI)	Rate per 1000 person years (No affected)			NNT per year (95% CI)	Rate per 1000 person years (No affected)			NNT per year (95% CI)
	Intensive (n=2729)	Conventional (n=1138)	ARR		Metformin (n=342)	Conventional (n=411)	ARR		Tight control (n=758)	Less tight (n=390)	ARR	
Any diabetes end point	40.9 (963)	46 (438)	5.1	196 (153 to 272)	29.8 (98)	43.3 (160)	13.5	74 (63 to 90)	50.9 (259)	67.4 (170)	16.5	61 (57 to 74)
Deaths from diabetes	10.4*	11.5*	—	—	7.5 (28)	12.7 (55)	5.2	192 (155 to 254)	13.7 (82)	20.3 (62)	6.6	152 (122 to 201)
Deaths from all causes	17.9*	18.9*	—	—	13.5 (50)	20.6 (89)	7.1	141 (115 to 183)	22.4 (134)*	27.2 (83)*	—	—
Myocardial infarction	14.7 (387)	17.4 (186)	2.7	370 (279 to 551)	11.0 (39)	18.0 (73)	7	143 (117 to 182)	18.6 (107)*	23.5 (69)*	—	—
Stroke	5.6 (148)*	5.0 (55)*	—	—	3.3 (12)*	5.5 (23)*	—	—	6.5 (38)	11.6 (34)	5.1	196 (159 to 257)
Microvascular complications	8.6 (225)	11.4 (121)	2.8	357 (285 to 478)	6.7 (24)*	9.2 (38)*	—	—	12.0 (68)	19.2 (34)	7.2	138 (114 to 178)

*Not significant at 5% level; presented here where relevant for comparisons between subtrials, prespecified end points only.

metformin treatment in the UKPDS appeared to have an effect on cardiovascular end points independent of its effect on blood glucose concentration, should it not have been included as a potentially confounding variable?

Another problem in the interpretation of the results is the inclusion of the lowest glycaemic group which, for the duration of the trial (or up to an event) had average haemoglobin A_{1c} values below 6%, below the upper limit of the authors' normal range (6.2%). A substantial proportion of the individuals in this group probably did not have diabetes as defined by the current guidelines of the World Health Organization or the American Diabetes Association. The fasting plasma glucose criterion used by the UKPDS was >6.0 mmol/l, 1 mmol/l less than that recommended by the WHO and American Diabetes Association. Statistically the point is important, for the lowest glycaemic group contributed the second largest number of person years to the analyses.

The debate on the nature of the relation between diabetes and hyperglycaemia, and cardiovascular disease has a long history, and the UKPDS has not resolved it. Perhaps however, given the substantial beneficial effects of statins and angiotensin converting enzyme inhibitors in secondary prevention and of hypotensive drugs in primary prevention, the debate has become academic. Clinicians will continue to try to control glycaemia to prevent microvascular disease.

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1 Stratton IM, Adler AI, Neil AW, Matthews DR, Manley SE, Cull CA, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. *BMJ* 2000;321:405-12. (12 August.)

2 Adler AI, Stratton IM, Neil AW, Yudkin JS, Matthews DR, Cull CA. Association of systolic blood pressure with macrovascular and microvascular complications of type 2 diabetes (UKPDS 36): prospective observational study. *BMJ* 2000;321:412-9. (12 August.)

Authors' reply

EDITOR—Budenholzer's suggestion that reduction of glycaemic exposure was not beneficial in the UK prospective diabetes study (UKPDS) is incorrect. Intensive blood glucose control by either sulphonylurea or insulin substantially decreases the risk of microvascular but not macrovascular disease.¹ The risk reduction for myocardial infarction (16% for a 0.9% haemoglobin A_{1c} difference; $P=0.052$) was, however, entirely consistent with that seen in the epidemiological analysis (14% for a 1% haemoglobin A_{1c} decrement). The improved risk reductions seen with metformin, above those expected from the glycaemic improvement achieved, have been discussed.² Moreover, we showed in table 3 that the point estimates and 95% confidence intervals for the risk reductions obtained in the clinical trial were not inconsistent with the estimates obtained from the observational analyses of either baseline or updated mean haemoglobin A_{1c} values. For example, the observational

analysis of updated mean haemoglobin A_{1c} concentrations for any diabetes related death showed a risk reduction of 21% (95% confidence interval 15% to 27%) for a 1% decrement in haemoglobin A_{1c}, consistent with the risk reduction achieved in the clinical trial of 10% (–11% to 27%) for a 0.9% absolute difference in median haemoglobin A_{1c} concentration.

Cruickshank's concerns about the possible effects of treatment allocation on the relation of glycaemia and blood pressure to diabetic complications have already been addressed by us in detail. In our first paper we reported that in these models treatment of blood glucose itself had no association with any complication beyond that of mean updated haemoglobin A_{1c}. In the second we showed that treatment of blood pressure had an effect over and above the updated mean systolic blood pressure for stroke, heart failure, and diabetes related deaths. Cruickshank suggests that we consider absolute rather than relative risks and numbers needed for treatment to benefit or harm, which we have already done.¹⁻⁴

When allocation to metformin is included in the model restricted to overweight patients, a significant metformin effect is seen on updated mean haemoglobin A_{1c} only for the any diabetes related aggregate end point ($P=0.044$). Reanalysis of the data, following Jarrett's suggestion, to include updated high density lipoprotein cholesterol and low density lipoprotein cholesterol, together with both updated haemoglobin A_{1c} concentrations and systolic blood pressure, shows the risk relations with glycaemia and blood pressure to be unchanged. The choice of reference category does not influence the gradient of the risk relations that were calculated from continuous data. The large number of people with updated mean haemoglobin A_{1c} values <6%, however, allows us to show the continuity of risk over a wide range of glycaemia.

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1 UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 1998;352:837-53.

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Genetic contribution to osteoarthritis of the hip

Did ethics committee consult specialists?

EDITOR—Lanyon et al studied genetic factors associated with osteoarthritis of the hip, but how ethical is it to subject more than 600 healthy participants (siblings of their original cohort) to pelvic radiography?¹ The authors used radiographs only as a diagnostic tool. Clinical examination using the Harris hip score would have more accurately obtained the diagnosis with the addition of information concerning loss of function and disease severity. Examination of the patient would have detected and excluded patients with rheumatoid arthritis.

Despite the use of radiographs in this study, no information about the morphology of the hip joints was given. It would have been fascinating to measure the degree of femoral head cover, angle of acetabular inclination, and femoral shaft offset, which govern the magnitude and direction of forces, and the degree of pressure concentration in the joint. Such morphological differences exist between races and are believed to account for differences in prevalence of osteoarthritis and hip dysplasia (G Fuji et al, combined congress of the British and Japanese Orthopaedic Associations, London, October 2000).

If Lanyon et al had undertaken a morphological analysis and found no significant variations between the study and control groups (presuming a similar racial breakdown in both groups, although this information is not given), then—rather than biomechanical and morphological factors—the composition of articular collagen (biochemical factors) may be implicated. If the composition of collagen predominates in influencing susceptibility this may focus treatment approaches in the future.

The study succeeds only in measuring the point prevalence of osteoarthritis in two selected groups, a figure more easily obtained by simply comparing the rate of total hip replacement between them. Did the ethics committee consult advice from specialists in musculoskeletal medicine before approval?

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1 Lanyon P, Muir K, Doherty S, Doherty M. Assessment of a genetic contribution to osteoarthritis of the hip: sibling study. *BMJ* 2000;321:479-83. (11 November.)

Genetic contribution needs further investigation

EDITOR—The paper by Lanyon et al on osteoarthritis of the hip adds further weight to the genetic contribution to primary osteoarthritis of the hip.¹

In a 1983 study of 341 patients of both sexes aged 39-86 having total hip replacement in Oxford, matched with a control population of 7072 blood donors from the same geographical catchment area, the relative frequencies of blood groups O and A were found to be reversed in the two groups, group A being commoner than group O in the osteoarthritis patients.² When phenotype frequencies of group O were compared with non-O (A, B, and AB), patients and controls differed significantly ($\chi^2=3.87$). All patients had radiological evidence of degenerative arthritis, with confirmatory histological evidence from examination of the excised femoral heads. All patients within this clearly defined group were included, as blood was routinely taken for cross matching before the operation in all cases. Lanyon et al included patients with both primary and revision hip replacements in their study population, whose composition also depended on response to a questionnaire. Although their index participants were a comparable group to those in the above blood group study, their sibling and urography groups were only defined by radiographic and not clinical evidence of arthritis.

Numerous insults to the hip such as trauma, infection, avascularity, rheumatoid disease, or congenital abnormality are known precursors of secondary arthritis. Such histories were excluded in the blood group study population. The pathogenesis of primary osteoarthritis of the hip remains elusive. Biochemical variations in cartilage metabolism under genetic control may be responsible for susceptibility to arthritic change in certain individuals, and the factors underlying the ABO polymorphism play a fundamental part in the organisation of cell membranes. Further large scale studies will provide more information about the genetic contribution to this common condition, but populations must be carefully specified to include only those in whom there is no known predisposing factor for arthritis, and who have clinical (and ideally histological) as well as radiographic evidence of the disease.

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1 Lanyon P, Muir K, Doherty S, Doherty M. Assessment of a genetic contribution to osteoarthritis of the hip: sibling study. *BMJ* 2000;321:479-83. (11 November.)

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Authors' reply

EDITOR—We defined osteoarthritis of the hip according to the gold standard definition for community epidemiological studies—namely, minimum joint space.¹ This definition is the best single radiographic predictor of hip pain and has been validated in similar populations

to ours. The prevalence of hip pain (ascertained using standard definitions) in participants with structural change accorded with other studies.¹

There is no consensus that acetabular dysplasia is a significant risk factor for osteoarthritis of the hip,^{2,3} and this hypothesis cannot be tested in cross sectional studies. Although there are geographic variations in the prevalence of osteoarthritis, these cannot be assumed to be due to hip dysplasia. For example, osteoarthritis is less common in Chinese than British men, despite similar prevalences of dysplasia.⁴

We recorded acetabular depth, centre-edge angle, and pattern of femoral head migration in all participants. The overall prevalence of dysplasia was similar to other studies.^{2,3} There was no evidence that dysplasia accounted for the difference in prevalence of osteoarthritis between the two groups.

Chambers suggests simply comparing the prevalence of hip replacement, but this strategy would introduce significant bias. Considerable variations exist within the United Kingdom in indications for, and provision of, surgery. There is also likely to be significant familial bias towards surgery among siblings, who may present earlier with hip pain, be referred earlier, or listed earlier if a sibling has required surgery—that is, the behaviour of both doctor and patient may be influenced. Factors that determine selection for surgery—for example, comorbidity, age, pain severity—would also strongly bias the results obtained. Additionally, detecting a familial predisposition to joint replacement may not be informative for the aetiology of less severe hip osteoarthritis in the community, where the main burden lies.

Several strategies were used to limit exposure of the population to radiography. We studied an existing group of participants to avoid exposing a new control group. When possible, existing radiographs (80 siblings) were used. We excluded participants younger than 45 because of their low likelihood of radiographic change.

The study design was appropriate to answer the question posed. The question was of sufficient clinical importance to justify the inconvenience to participants, exposure to radiation, and the expense entailed. We agree that a biased or underpowered study that did not utilise accepted case definitions would not have been ethical.

We agree with Lourie that careful phenotypic description is essential. Our participants have been extensively characterised according to structural change, morphology, hip pain, and risk factors for osteoarthritis and provide a significant resource for further molecular genetic studies.

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In praise of mercury sphygmomanometers

Appropriate sphygmomanometer should be selected

EDITOR—Users of mercury sphygmomanometers are being advised to consider alternatives, but this is causing problems.^{1,2} Currently there is confusion over the advantages and disadvantages of the alternatives. The mercury sphygmomanometer, when used by trained staff, is the gold standard. Aneroid devices are also in widespread use, but they can be knocked out of calibration easily. These devices can be used, provided they are recalibrated every six months, but indications are that this advice is rarely taken.

Automated devices are now readily available. The British Hypertension Society states that for these devices to be acceptable, no more than 25% of measurements should be in error by more than 10 mm Hg and no more than 10% by 15 mm Hg.³ Automated devices have a well accepted role in monitoring changes in blood pressure but a more limited one in determining absolute blood pressure. The combined recommendation of the European Society of Cardiology, the European Society of Hypertension, and the European Atherosclerosis Society is quite clear—automated devices are unsuitable as a routine substitute for the measurement of clinic blood pressure in the diagnosis of hypertension and not appropriate for determining the need for treatment and for assessing treatment efficacy.⁴

Concerns have been expressed to the European Standards Committee for sphygmomanometers that the current degree of clinical accuracy required by the standard for automated devices is inadequate. Some would like to see noticeable improvements, but manufacturers will resist this strongly, simply because better accuracy cannot yet be achieved and, as O'Brien points out, the oscillometric techniques cannot measure blood pressure in all situations.⁵ Clinical users must decide when automated devices are appropriate and when they are not. We should not allow the argument that clinical staff are poor at taking manual measurements to influence decisions. Clinical staff can be trained.

The looming difficulties over the measurement of blood pressure have been clear for some years. Recognising this, at the Freeman Hospital and the University of Newcastle, we developed a manual device in

collaboration with a manufacturer of traditional sphygmomanometers. This modern electronic device is an accurate alternative to the mercury sphygmomanometer, with features to improve measurement technique and to provide automatic recalibration when switched on.

Standards can help by weeding out poor quality devices but they do not recommend which devices should be used. A clinical decision must be made when selecting between manual and automated devices.

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Electronic readings of blood pressure seem to be higher than readings obtained with mercury sphygmomanometers

EDITOR—Lawes writes of how he prefers mercury sphygmomanometers to other blood pressure measuring devices.¹ In view of the forthcoming rules regarding the use of mercury devices I performed a trial of a mercury compared with an electronic (Omron) device.

Patients' blood pressure was checked with the electronic device followed by a mercury sphygmomanometer to avoid the electronic reading influencing the observer. The left arm was used for all readings. Fifty one patients were checked in this way. In three cases the electronic device could not be made to record a measurement.

In the remaining 48 patients the results were as follows. The mean (SD) readings with the mercury thermometer were: systolic 136.8 (23.7) mm Hg and diastolic 70.6 (11.1) mm Hg. The mean readings with the electronic device were: systolic 150.0 (23.1) mm Hg and diastolic 82.5 (13.7) mm Hg.

My partners and I are concerned that the electronic readings are higher than the readings obtained with the mercury sphygmomanometer. Blood pressure recording and the treatment of patients with pressures above 140/80 mm Hg occupy a considerable amount of our time and effort. If we switch to electronic measurement of blood pressure will we be increasing the amount of drug treatment used, and how valid will this be?

I would be interested to hear if others have found that electronic devices give higher readings.

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Marketing studies and scientific research must be distinct

EDITOR—Drew and Davies's trial to assess the effectiveness of *Ginkgo biloba* in tinnitus, which was conducted by post and telephone, raises several important methodological issues.¹ The advantages of such a design are obvious: easy and quick recruitment of patients allowing large sample sizes at comparatively low costs. The disadvantages are exemplified by the study's limitations.¹

As there was no doctor-patient contact the exact diagnoses are not certain, and the outcome measures depict only perceived effects. External monitoring and quality control were impossible, which means that the quality and reliability of the rough data are questionable. The patients' general practitioners could probably not be informed, and serious adverse events or drug interactions were impossible to assess. *Ginkgo biloba* has antiplatelet activity² and thus can lead to serious bleeding—for example, haemorrhagic stroke—and an increase in bleeding tendency when taken concomitantly with oral anticoagulants.^{3,4}

The question is whether the advantages of such a design outweigh its disadvantages. The answer obviously depends on the perspective taken. From the sponsor's commercial point of view the advantages would dominate (provided the trial's result was positive, which in the present case it was not). From a scientific point of view the disadvantages seem decidedly more important. A methodologically weak study does not get more conclusive through increasing its sample size.

A clear distinction should always be made between marketing studies and scientific research; the latter should be given preference in respected journals.

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All NHS consultants must have equal entitlement to awards

EDITOR—The Department of Health has published a consultation document on the NHS clinical excellence scheme.¹ This highlights the relatively few awards given to some specialties, to female doctors, and to doctors from ethnic minorities and the disproportionately large number of awards to honorary or academic consultants. Altogether 9% of the consultants hold posts as honorary

consultants, but they hold 39% of the awards.¹

Although well deserved, these awards were perhaps achieved because of the greater opportunities that these consultants had and the bias in favour of academic achievements: even outstanding contributions to patient care and service to the NHS were considered less worthy of the higher awards. Discrimination is said to account for the differences between the sexes and ethnic differences, but, despite statistics suggesting this, data are inadequate to justify this conclusion.² Awards committees were urged to rectify these discrepancies while still making their nominations entirely on merit.

The lack of recognition for those with direct patient care has prompted the government to act to redress this imbalance, to the satisfaction of many consultants who considered that they had been unfairly treated. The means of achieving redress outlined in the consultation document is, however, contentious. The awards in future will be weighted towards those who contribute most to the NHS, and "the majority of awards will go to those who make the biggest contribution to the delivery and improving of the health services." This implies preferential treatment of some consultants, with serious consequences for academic medicine.

A fair and just system should ensure that all consultants have equal entitlement to awards, with academic, service, and other contributions receiving parallel recognition. The level of the award should be determined by the level of attainment of the objectives and criteria for academic, service, or other activities, perhaps separately stated for the different groups. Facilities and opportunities available to a person should be taken into account.

But a further issue remains. Is it realistic to have weighted or even proportional representation of the different groups if awards are to be granted strictly on merit? For example, is it unreasonable to accept that there might be a degree of preselection, based on ability for entry into some specialties? Factors determining these imbalances should be scientifically evaluated and not assumed before corrective measures are instituted.

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Rapid responses

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