BMJ

Author's response to influenza vaccination: policy v evidence

Tom Jefferson

BMJ 2006;333;1172
doi:10.1136/bmj.39045.403808.1F

Letters

Physical activity to prevent obesity in young children

Negative outcome or protocol problem?

EDITOR—Although the article by Reilly et al on physical activity to prevent obesity in young children emphasises that adherence to protocol was good,[1] an interview with Reilly on the *Today* programme (7.20 am, 6 October 2006) cited the difficulty in getting children to increase their activity as much as required—that is, not a negative outcome, but a problem in achieving sufficient increase to make the intervention effective. Which was the case?

Alastair Michell *professor of comparative medicine*
University of London, Barts Hospital Harvey Institute, London EC1M 6BQ
Bobmichell@Hotmail.com

Competing interests: None declared.

1 Reilly JJ, Kelly L, Montgomery C, Williamson A, Fisher A, McColl JH, et al. Physical activity to prevent obesity in young children: cluster randomised controlled trial. *BMJ* 2006;333:1041-3. (18 November.)

BMI in the *BMJ*

EDITOR—Reilly et al entitle their article, "Physical activity to prevent obesity …" and conclude, "Alternative interventions [to physical activity] to prevent obesity in young children are required."[1] The emphasis on a lack of impact of physical activity on obesity is unfortunate for four reasons.

JEREMY SUTTON HIBBERT/REX

Firstly, the independent variable was not successfully manipulated: the intervention failed to generate differences in physical activity or sedentary behaviour between groups. Why would one expect a "physical activity intervention" which has no impact on physical activity to alter energy balance or "obesity"?

Secondly, the principal outcome measure, body mass index (BMI), is an inappropriate measure of obesity for two reasons.

Studies of obesity should focus on body composition, rather than measures of body weight (BMI). We, like Reilly et al, observed no differences in weight or BMI in supervised exercise studies in obese children.[2] However, dual energy x ray absorptiometry (DEXA) showed significant decreases in central fat mass. Weight and BMI did not concomitantly change because lower limb muscle mass increased. We also observed improvements in euglycaemic hyperinsulinaemic clamps and vascular function with training (A M Thompson et al, 14th annual scientific meeting, Australasian Society for the Study of Obesity, Adelaide, October 2005).[2] A review of DEXA studies in obese children showed similar countervailing impacts of exercise on fat and lean body mass.[3] A conclusion of no impact on obesity based on lack of change in BMI is misleading.

BMI corrects for individuals who are heavy because they are tall, a valid concept in cross sectional population comparisons. However, in within subject longitudinal experiments, change in BMI reverts to a measure of change in weight because interventions do not alter height. Using weight as an index of obesity is flawed (above). In children, change in BMI is not even a valid measure of weight change because height also changes. Reilly et al did not include data on changes in height or weight or on waist girth.

Thirdly, no amount of statistical manipulation alters the fundamental fact that BMI is not a measure of obesity (adiposity) and change in BMI within subjects is not a proxy for change in obesity.

Finally, BMI may be practical, precise, and easy to collect, but these characteristics do not make it a valid measure of obesity in this context.

Research studies fundamentally need to manipulate an independent variable and measure the impact on a legitimate dependent variable. The study of Reilly et al failed on both counts. The paper generated widespread and unfortunate, but understandable, international media attention.[4] Exercise remains a key interventional strategy for the management of obesity in young people.

Daniel J Green *professor of cardiovascular physiology*
d.j.green@ljmu.ac.uk
Tim Cable *professor*
Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool L3 2ET

Competing interests: None declared.

1 Reilly JJ, Kelly L, Montgomery C, Williamson A, Fisher A, McColl JH, et al. Physical activity to prevent obesity in young children: cluster randomised controlled trial. *BMJ* 2006;333:1041-3. (18 November.)
2 Watts K, Beye P, Siafarikas A, Davis EA, Jones TW, O'Driscoll G, et al. Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents. *J Am Coll Cardiol* 2004;43:1823-7.
3 Watts K, Davis E, Jones T, Green DJ. Effect of exercise training in obese children and adolescents. *Sports Med* 2005;35:1-18.
4 Boseley S. More exercise does nothing to stop obesity in youngsters. *Guardian* 2006 Oct 6.

Dietary and behavioural modifications in managing childhood obesity

EDITOR—The article by Reilly et al provided conclusive evidence that other interventions besides enhanced physical activity are necessary for decreasing the BMI (body mass index).[1] The management of childhood obesity needs a multidimensional approach including dietary modifications, behavioural modifications, and physical exercise. Only in conjunction with the former two will physical activity have an impact in reducing obesity. Dietary modifications include avoiding eating in restaurants, decreasing soft drink intake,[2] decreasing portion sizes, avoiding dried and calorie rich foods, and increasing the fibre content of diet. Behavioural modification strategies include educating children and parents about healthy diets, encouraging children to keep food diaries and avoiding habits such as eating while watching television. Reinforcement of these strategies along with regular physical exercise is likely to produce significant results rather than using one approach exclusively. The management of childhood obesity is especially important to prevent complications such as low self esteem,[3] hypertension, hyperlipidaemia, sleep apnoea,[4] slipped femoral epiphyses,[5] and diabetes mellitus.

Besides it needs to be remembered that though rare, there are genetic causes of obesity—such as Alstrom syndrome and Prader Willi syndrome—as well as endocrine causes such as hypothyroidism that need to be excluded before the above mentioned approaches are used.

S Kapoor *resident physician*
UIC, Chicago, IL 60612, USA
skapoor5@uic.edu

Competing interests: None declared.

1 Reilly JJ, Kelly L, Montgomery C, Williamson A, Fisher A, McColl JH, et al. Physical activity to prevent obesity in young children: cluster randomised controlled trial. *BMJ* 2006;333:1041-3. (18 November.)
2 Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001;357:505-8.
3 Davison KK, Birch LL. Weight status, parent reaction, and self-concept in five-year-old girls. *Pediatrics* 2001;107:46-53.
4 Mallory GB Jr, Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. *J Pediatr* 1989;115:892-7.
5 Kelsey JL, Acheson RM, Keggi KJ. The body build of patients with slipped capital femoral epiphysis. *Am J Dis Child* 1972;124:276-81.

Authors' reply

Editor—Michell asks whether our outcomes resulted from inadequate implementation of the intervention. The intervention was well implemented, but marked and sustained increases in physical activity were not observed: the intensity of physical activity sessions may have been less than achieved in the pilot study.[1] Alternatively, children may have "compensated" for increased physical activity by reduced physical activity at other times,[2] though this was not observed in our pilot study.[1] This emphasises the importance of objective measures of physical activity: subjective measures are biased.

Green and Cable, and Kapoor, question the proposed mechanism for our intervention. Increases in physical activity should increase energy expenditure,[3] and we aimed to limit opportunities for eating by reducing sedentary time.[1] Focusing intervention on one or two behaviours has usually been more successful in obesity prevention than the wider strategy suggested by Kapoor.[4]

Our primary outcome was body mass index (BMI) relative to UK reference data from 1990 as an SD score, taking account of age and sex specific differences in BMI. BMI SD score is increasing in British children, reflecting increased fatness across the distribution.[5] Obesity prevention trials examine differences in the trajectory of increasing BMI SD score as a test of the efficacy of the intervention.[4] The BMI SD score is also a practical proxy for body fatness in field studies and is measured with high precision and more direct field body composition measures are inaccurate and imprecise. Body composition estimates from bioimpedance in our trial did not suggest any differences between intervention and control groups, and we found no significant difference between groups in waist SD scores.

John J Reilly *professor of paediatric energy metabolism*
jjr2y@clinmed.gla.ac.uk
James Y Paton *reader in paediatric respiratory disease*
Division of Developmental Medicine, University of Glasgow, Yorkhill Hospitals, Glasgow G3 8SJ

J H McColl *reader in statistics*
Department of Statistics, University of Glasgow, Glasgow G12 8QQ

A Williamson *honorary research fellow*
Glasgow City Council Education Department, Glasgow G1 1HL

Competing interests: None declared.

A more detailed reply is available at www.bmj.com/cgi/eletters/333/7577/1041#149295

1 Reilly JJ, McDowell ZC. Physical activity interventions in the prevention and treatment of childhood obesity. *Proc Nutr Soc* 2002;62:611-9.
2 Wilkin TJ, Mallam KM, Metcalf BS, et al. Variation in physical activity lies with the child, not his environment: the activitystat hypothesis. *Int J Obes* 2006;30:1050-5.
3 Montgomery C, Reilly JJ, Jackson DM, Kelly LA, Slater C, Paton JY, et al. Relation between physical activity and energy expenditure in a representative sample of young children. *Am J Clin Nutr* 2004;80:591-6.
4 Robinson TN. Reducing children's television viewing to prevent obesity: a randomised controlled trial. *JAMA* 1999;282:1561-7.
5 Reilly JJ. Tackling the obesity epidemic: new approaches. *Arch Dis Child* 2006;91:724-6.

Author's response to influenza vaccination: policy *v* evidence

Editor—My analysis was based on 206 studies (several million observations' worth of data) included in systematic reviews spanning some 40 years. The hypotheses by Mandl do not fit some of the evidence in the elderly population.[1] He cannot explain how in years of good matching between vaccine antigenic content and circulating viruses the vaccines fail to prevent deaths from all respiratory diseases in elderly community dwellers (1.32, 95% confidence interval 1.25 to 1.39, 426 668 observations) while at the same time preventing 42% (25% to 55%, 404 759 observations) of deaths from all causes,[2] presumably including deaths from falls, accidental poisoning, accidents, hypothermia, and so on.

Fedson and Nichol deride my choice of example of poor methodological quality of a large number of available cohort studies.[1] The authors of the studies either did not know such details or like Fedson and Nichol thought them irrelevant and would leave a reader to work them out from "official records." Vaccine matching and level of circulating influenza viruses are the most important predictors of vaccine efficacy and effectiveness. The closer the match and the higher the viral circulation, the better the performance of the vaccine.[3] Without such knowledge it would be very difficult to give an honest and reliable assessment of the effects of the vaccine. That is one of the reasons why these studies are of poor quality.

I note with worry their statement that decisions should be made on three of the most notoriously biased sources of information: non-randomised studies, expert opinion, and economic evaluations.[4 5] It is precisely because most comparative evidence on elderly people comes from non-randomised studies that we are left with the question: are the effects we witness due to the vaccines or are they due to confounding? The tone of the response by Fedon and Nichol (lack of vaccines' effect in small children is undoubtedly due to small numbers and my concern over lack of vaccine safety data—a statement from which they omitted the key word "comparative") implies that my review seemed to be questioning a dogma. Heretics like me get short shrift.

I repeat my statement that especially in elderly people, an insufficient number of field trials has been conducted (five, of which only one has been carried out in the past decade) to allow reasonable certainty of the effects of inactivated vaccines. The nature of the evidence from non-randomised designs when analysed critically and exhaustively is weak and contradictory. I repeat my observation that the totality of safety evidence from comparative sources (studies in which a proportion of participants were contemporaneously exposed or not to the vaccines) is tiny in small children (35 observations) and small in the elderly (2963 observations).

Tom Jefferson *coordinator*
Cochrane Vaccines Field, Anguillara Sabazia, Roma 00061, Italy
jefferson.tom@gmail.com

Competing interests: TJ owned shares in Glaxo SmithKline and received consultancy fees from Sanofi-Synthelabo (2002) and Roche (1997-1999).

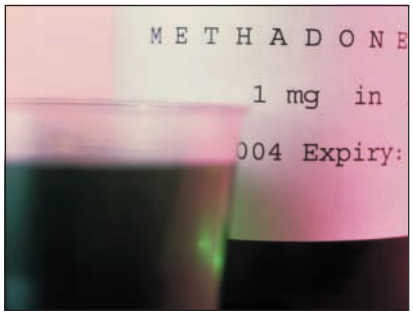
1 Correspondence. Influenza vaccination: policy versus evidence. *BMJ* 2006;333:1020-1. (11 November.)
2 Rivetti D, Demicheli V, Di Pietrantonj C, Jefferson TO, Thomas R. Vaccines for preventing influenza in the elderly. *Cochrane Database Syst Rev* 2006; (3):CD004876.
3 Demicheli V, Rivetti D, Deeks JJ, Jefferson TO. Vaccines for preventing influenza in healthy adults. *Cochrane Database Syst Rev* 2004;(3):CD001269
4 Kunz R, Oxman AD. The unpredictability paradox: review of empirical comparisons of randomised and non-randomised clinical trials. *BMJ* 1998;317;1185-90.
5 Jefferson T, Demicheli V, Vale L. Quality of systematic reviews of economic evaluations in health care. *JAMA* 2002;287:2809-12.

Methadone tolerance testing in drug misusers

Beyond the edge of safety

Editor—The model of methadone tolerance testing Bakker and Fazey describe is not the solution to this problem.[1] They make the assumption that low mean maintenance doses of methadone are a consequence of low starting doses, which are separate issues. The model of induction into methadone treatment recommended in the national clinical guidelines for the management of drug dependence allows for the attainment of adequate optimised maintenance doses.[2] Reluctance on the part of some prescribers (and patients) to increase doses to the recommended range between 60 mg and 120 mg a day is the clinical issue needing further attention.

Bakker and Fazey cite no drug related deaths during tolerance testing as evidence of the accuracy of their patients' estimated dose of methadone required to relieve withdrawal. However, this speaks more for the particular characteristics of the primary care practice involved, its drug dependent clientele, its prescribers, and the therapeutic and professional relationships of the key stakeholders in this model of induction than for the safety of the methadone tolerance testing protocol. It therefore cannot be



CORDELIA MOLLOY/SPL

extrapolated to other drug treatment settings. Neither large, high volume drug treatment services nor small, less experienced settings can afford to invest such confidence in unknown, newly presenting drug users' own predictions of the "right" methadone dose for them.

Even with cautious clinical practice during the first week of treatment, deaths occur, of the order of seven deaths per 10 000 inductions, about double that for heroin users not in treatment.[3] Overall, however, methadone treatment reduces by two thirds drug users' risk of death while in treatment.[4]

Clinicians need to be aware that death during induction while rare, is preventable through cautious and responsible practice that takes account of the known biovigilance data.[5] The practice as outlined by Bakker and Fazey is well beyond such data and if practised on a widespread basis will inevitably result in unnecessary deaths.

We think the publication of this paper, unaccompanied by an independent expert commentary, is an example of poor editorial judgment.

Deborah A Zador *consultant physician in addictions*
South London and Maudsley Foundation Trust, London SE5 8RS
deborah.zador@slam.nhs.uk

Michael Farrell *reader in addiction psychiatry*
National Addiction Centre, Institute of Psychiatry, London SE5 8AF

Competing interests: None declared.

1 Bakker A, Fazey C. Methadone tolerance testing in drug misusers. *BMJ* 2006;333:1056-9. (18 November.)
2 Department of Health, Scottish Office Department of Health, Welsh Office, Department of Health and Social Services Northern Ireland. *Drug misuse and dependence—guidelines on clinical management*. London: Stationery Office, 1999.
3 Zador D, Sunjic S. Methadone related deaths and mortality rate during induction into methadone maintenance, New South Wales, 1996. *Drug Alcohol Rev* 2002;21:131-6.
4 Brugal MT, Domingo-Salvany A, Puig R, Barrio G, Garcia de Olalla P, de la Fuente L. Evaluating the impact of methadone maintenance programmes on mortality due to overdose and aids in a cohort of heroin users in Spain. *Addiction* 2005;100:981-9.
5 Commonwealth Department of Health and Aged Care. Proceedings of an expert workshop on the induction and stabilisation of patients onto methadone: findings of an expert workshop, 28/29 January 1999, Adelaide, South Australia. Monograph Series No 39. National Drug Strategy. Canberra, 2000.

Author's response

Editor—There would be little reason to perform methadone tolerance testing if it didn't seem to attract difficult to engage addicts and if conventional induction were really safe. Unfortunately, slow titration may actually involve a mortality up to seven times greater than untreated addiction.[1] Therefore, even when complying with the guidelines, the risks are not minimal; only the risk of blame is absent. Whether methadone tolerance testing is even riskier remains to be seen. I consider it good practice to discuss overdose risk and its management at the start of treatment, whether using conventional titration or tolerance testing. I have no illusions that useful observation occurs the night after patients have their test dose, but this is too good an educational opportunity to miss.

The risk that patients might not return after a test dose is a real concern, and it would be better to perform the test procedure on site. Unfortunately, bureaucracy makes keeping methadone on the premises unattractive, but we hope to address this soon.

Despite great expansion of treatment facilities and reduction in waiting times, only a minority of addicts currently engage in treatment.[2] It seems that very often patients' motivation is being tested to destruction by requiring numerous assessments before any medication is offered. In this way, only "easy" patients eventually make it through to an actual prescription: the "difficult" ones (who actually cause the most trouble to society and to themselves) are effectively excluded from treatment and, once again, nobody blames the services that make life so difficult for them.

Although early and adequate medication does not address all the issues, it makes it easier to address them later because patients more often keep appointments and engage with counselling once on maintenance. In no other field of medicine would it be considered ethical to test patients' motivation when health and social benefits are so clear cut. To facilitate entry into treatment, I do indeed offer rapid access but also choice: I start as many patients on buprenorphine as methadone. I also offer community detoxification and naltrexone implants.

We doubt that clinicians change their prescribing based only on a relatively small number of cases. However, our experience may be useful in some common situations. For example, the dilemma one faces when a patient fails to collect medication for a few days. Some guidelines recommend that the dose is reduced in this situation, just when withdrawal begins to bite. A tolerance test might be a useful alternative.

Surely the editors deserve credit for publishing this peer encouraged paper, which may stimulate debate and research.

Adam Bakker *general practitioner*
Lisson Grove Health Centre, London NW8 8EG
bakker@nhs.net

Competing interests: None declared.

1 Caplehorn JRM, Drummer OH. Mortality associated with New South Wales methadone programmes in 1994: lives lost and saved. *Med J Aust* 1999:141-55
2 Home Office. *Updated drug strategy 2002*. London: Home Office, 2002.

Refusing to provide a prenatal test: can it ever be ethical?

Time to re-think the autonomy of future individuals

Editor—Dalatycki says that opposition to prenatal testing is based on the ethical principles of confidentiality, and of preserving the autonomy of the future individual (in order that they can make the decision once they have sufficient intellectual and emotional maturity to choose whether to test).[1] This view enjoys widespread support if we are to judge by the quoted guidelines of the International Huntington Association and the World Federation of Neurology[2] and by Dalatycki's observation that all major human genetic societies recommend against testing minors for genetic disorders that have their onset in adulthood and for which no preventive treatment exists.[3 4] I am left wondering why this future autonomy principle is not paramount in the decision of whether to terminate a healthy pregnancy? Termination is surely the ultimate loss of autonomy for a future individual, yet it is legal in this country and also widely accepted.

If we accept that ethics is the science of deciding the right thing to do based on reason then this timely article must prompt a re-think of the ethical principles concerning unborn children? Are they future individuals with rights or not? Ethics introduces the notions of consistency and reproducibility to decision making which makes the accepted views concerning prenatal testing for Huntington's disease and concerning so called "social" termination of pregnancy incompatible. They cannot both be right.

Paul D Kelly *general practitioner principal*
Highfield Surgery, Blackpool FY4 2LD
pdkelly@lineone.net

Competing interests: None declared.

1 Duncan RE, Foddy B, Delatycki MB. Refusing to provide a prenatal test: can it ever be ethical? *BMJ* 2006;333:1066-8. (18 November.)
2 International Huntington Association and the World Federation of Neurology Research Group on Huntington's Chorea. Guidelines for the molecular genetics predictive test in Huntington's disease. *J Med Genet* 1994;31:555-9.
3 Clinical Genetics Society. The genetic testing of children. Working party of the Clinical Genetics Society (UK). *J Med Genet* 1994;31:785-97.
4 American Society of Human Genetics Board of Directors, American College of Medical Genetics Board of Directors. Points to consider: ethical, legal and psychosocial implications of genetic testing in children and adolescents. *Am J Hum Genet* 1995;57:1233-41.

Rights of future children

Editor—Contrary to Duncan and Foddy,[1] I would argue that clinicians who refuse a test in these circumstances are not being paternalistic, they are upholding the rights of a future child over the rights of parents. The right not to know a genetic diagnosis, or to decide for oneself when to have a diagnosis, is very important, particularly in cases where there is no cure for a condition.

Nor is it clear why parents may want this information to assist them in making a decision about whether to have more children. Symptoms of Huntington's disease develop in adulthood, not childhood, so the status of the child would not have implications for parenting. The status of one child has no implications for the status of any future child they may have. The implication is that the parents do not understand risk, or their other reproduction options.

Clinicians should explain carefully the grounds on which they take the decision to refuse testing, in the hope that parents will come to a similar view. There is always a risk of parents manipulating the system, just as

there is with prohibition on social sex selection, but this is not a reason to relax ethical guidelines.

Paternalism is not always wrong, particularly when it comes to genetic information about future people: clinicians often forgo communicating information which they could obtain. We are not provided with genetic printouts at birth, nor are all the genetic characteristics of a fetus communicated to the potential parents. Long may it remain so.

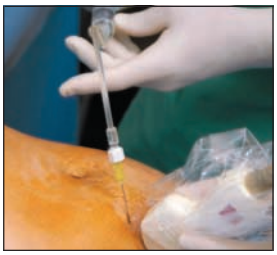
Tom W Shakespeare *research fellow*
Newcastle University, Newcastle upon Tyne NE1 7RU
t.w.shakespeare@ncl.ac.uk

Competing interests: None declared.

1 Duncan RE, Foddy B, Delatycki MB. Refusing to provide a prenatal test: can it ever be ethical? *BMJ* 2006;333:1066-8. (18 November.)

The darker side of medicine

EDITOR—Delatycki seems to suggest that testing is offered to those parents who have yet to make a decision as to whether or not to go through with the pregnancy.[1] In this situation, however, if the test is negative and the parents subsequently choose to have the baby, has not the child suffered the same breach of rights and confidentiality even though the result itself is negative? Surely the ethical implication is the same.



AJ PHOTO/SPL

This makes the idea of judging the relative autonomy of parents versus the autonomy of the child seem rather beside the point. In a system where we have given parents the precedence to decide on the right of the child to exist, it is unrealistic, as Duncan and Foddy have suggested,[1] to set terms on their decision. Despite the breach of the child's rights, this is not where the ethical difficulty stems from—this is no different from other situations when we as a profession have to decide on the ascendancy of rights, such as the rights of the patient versus public interest. Instead, what Delatycki is suggesting is that we provide this test only in cases where it validates the medical profession by allowing it to take further action in the lives of the parents and future family. Where our medical knowledge is powerless to intervene or prevent the inevitability of Huntington's disease, then the knowledge of the possibility of that disease is categorised as potentially harmful and therefore irrelevant to the parents.

This goes a bit further than mere paternalism or abuse of power. It reveals the darker side of medical assumption. The offering of the test is based on the argument that the medical profession has the right to control not just the immediate clinical scenario but the disease entity and its psychosocial impact. Duncan and Foddy cannot entirely avoid this either. Both sides refer to research on the psychological implications, whether for or against testing. This underestimates the value that should be placed on parents themselves having the right to determine how these factors inform their decisions. Knowledge is in our power to divulge, and it is not necessarily up to the profession to define all of its wider impact.

Nushan P Gunawardana *medical student*
University College London, London WC1E 6BT
nushan_g@yahoo.co.uk

Competing interests: None declared.

1 Duncan RE, Foddy B, Delatycki MB. Refusing to provide a prenatal test: can it ever be ethical? *BMJ* 2006;333:1066-8. (18 November.)

Ethics or humanity?

EDITOR—Huntington's disease manifests with cognitive, emotional symptoms, uncontrollable and abnormal movements due to destruction of neurones. There is loss of thinking, planning, speech, and synchronisation; the memory is affected, as is muscle control; psychological imbalances occur, and much more. Such patients face death more often due to complications of the disease than the disease itself; one of those is succumbing to the disorder by committing suicide. It is an adult onset disease, but juvenile Huntington's disease occurs in young children.

In autosomal dominant people the possibility of bearing affected progeny is 50%. Which parent who knows all this and is affected by the disease would want a baby, who might not even live a normal life till middle age?[1] The emotions and concerns of a parent cannot be termed paternalism in a disapproving sense. A baby is a part of the mother's body. She has the right to decide her unborn child's future. Where does she defy or disobey ethics? It is a doctor's moral and ethical duty to explain to the couple all possible consequences of a pregnancy during antenatal follow-ups, and to do this even more carefully if genetic disorders are suspected. The couple should be informed about tests to diagnose disorders in their baby, especially if the baby may be born with an incurable and potentially lethal condition. And the decision to terminate or continue the pregnancy once the outcome is known should also lie with the parents to be.

Neha S Godre *intern*
Lokmanya Tilak Municipal General Hospital, Sion, Mumbai 400022, Maharashtra, India
neha_godre@rediffmail.com

Competing interests: None declared.

1 Duncan RE, Foddy B, Delatycki MB. Refusing to provide a prenatal test: can it ever be ethical? *BMJ* 2006;333:1066-8. (18 November.)

Even clinician led management won't fix the NHS

EDITOR—Although I echo Flook's personal view and have argued similarly for many years,[1 2] I suspect that until governments agree not to set political targets by which healthcare provision is measured, even clinician led management will fail. The NHS was charged originally with providing the best available health care to the whole population, according to need, free at source. In a changing environment with financial constraints, the challenge for us all is to match practice to purpose, and to achieve this, resources must be allocated having prioritised the critical elements necessary to provide a measured response to the perceived need. And so everything turns on the definition of "need" and who defines it.

Too many of the decisions concerning the allocation of medical resources are made for the wrong reasons, using the wrong criteria, by the wrong people. Our profession has abrogated responsibility over these vital decisions to people who should not be making them. We don't allow the care of individual patients to be defined solely in terms of finance, yet we seem content to allow the strategy for caring for large numbers of patients to be decided on this criterion alone.

We need management at all levels in the NHS to be undertaken by doctors trained in the necessary core skills to do the job well. Managing medical resources is part of delivering medical care. Politicians and administrators can ignore detail and seem to get away with it. Doctors and nurses cannot: they talk to patients every day, share their lives and deaths, win their trust and interact in the most intimate ways with people at their most vulnerable and, therefore, they are unlikely to forget the purpose of the NHS and the services it provides. But arguably more important than the need for doctors to enter NHS management, is the need for them to enter politics at all levels and in all parties, so that they can influence NHS policy and strategy. In difficult times, quality of care will be maintained and even improved only if medical resources are managed by clinicians, and NHS policy is influenced and formed by clinicians in government.

Adam R Greenbaum *consultant plastic surgeon*
St Thomas' Hospital, London SE1
adam@elestar.co.uk

Competing interests: None declared.

1 Flook D. Clinician led management can fix the NHS. *BMJ* 2006;333:1077. (18 November.)
2 Greenbaum AR. The place of clinicians in NHS management. *Br J Healthcare Manage* 1995;1:702-4.