We select the letters for these pages from the rapid responses posted on bmj.com favouring those received within five days of publication of the article to which they refer. Letters are thus an early selection of rapid responses on a particular topic. Readers should consult the website for the full list of responses and any authors' replies, which usually arrive after our selection.

GLOMERULAR FILTRATION RATE

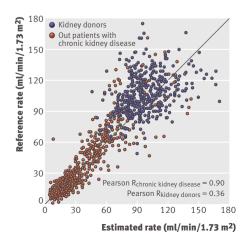
Reporting eGFR has benefits

Giles and Fitzmaurice overlooked one of the main aims of the recent guidelines on chronic kidney disease and did not take into account the accepted definition of stages 1 and 2, or the recommendations on screening.¹² Reducing late referral of people who are heading towards dialysis (and avoiding the associated poor outcome) was one of the intentions of the guidelines.²

The main reason for late referral is that glomerular filtration rate (GFR) can be very low when the serum creatinine is only modestly increased and the severity of the kidney disorder is underestimated. In spite of its shortcomings, eGFR reporting is the best method available to aid interpretation of serum creatinine. Since this was introduced in our unit (together with a programme of education in primary care), the proportion of new patients receiving dialysis who were referred late (defined as within 90 days) has fallen from 38% to 25% (P<0.01).

The diagnosis of stages 1 and 2 does not depend on GFR alone. Stages 1 and 2 refer to people known to have another kidney problem—either functional (proteinuria) or structural (polycystic disease)—and in whom the GFR is at least 60 ml/min (normal or nearly so).

The guidelines did not advocate a screening programme but recommended testing for kidney disease in those at risk, including patients with diabetes and hypertension in whom such testing has



been normal practice for several years. The suggested improvements in sample collection and analysis deserve attention in their own right, irrespective of policies on eGFR reporting.

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Competing interests: RPB was a member of the Chronic Kidney Disease Guideline Development Committee.

- 1 Giles PD, Fitzmaurice DA. Formula estimation of glomerular filtration rate: have we gone wrong? *BMJ* 2007;334:1198-200. (9 June.)
- 2 Chronic kidney disease in adults: UK guidelines for identification, management and referral. London: Royal College of Physicians, 2006.

eGFR in changing drug regimens

Giles and Fitzmaurice did not discuss using estimated renal function to guide changes in the dosage regimens of drugs that are eliminated unchanged by the kidneys, that have active metabolites that are eliminated by the kidneys, or whose pharmacodynamic effects are affected by renal insufficiency.¹ This is particularly important for drugs that have a low therapeutic index.

Recommendations about drug dosage regimens are based on creatinine clearance. This is customarily derived from the Cockcroft-Gault equation for adults² or the Schwartz-Haycock equation for children.³

The eGFR estimated by the modified four variable modification of diet in renal disease (MDRD) equation underestimates true GFR more than the Cockcroft-Gault equation does in younger patients and less in older patients; overall, MDRD underestimates true GFR more than Cockcroft-Gault does.⁴ There are further differences in critically ill patients with burns.⁵ There is currently no information on how to use the eGFR to calculate changes in drug dosage regimens.

Clinical biochemistry laboratories would help doctors if they reported not only the MDRD-derived eGFR in ml/min/1.73 avoid m², but also the Cockcroft-Gault estimated creatinine clearance in ml/min/70 kg, for which only the age and sex of the patient are needed (and not also ethnic group, as for eGFR). General practitioners

LETTERS

could programme the appropriate equations into their computerised records.

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Competing interests: JKA is a member of the Joint Formulary Committee of the *British National Formulary* and the Paediatric Formulary Committee of the *British National Formulary for Children*. However, the opinions expressed here do not necessarily reflect those of other members of those committees.

- 1 Giles PD, Fitzmaurice DA. Formula estimation of glomerular filtration rate: have we gone wrong? *BMJ* 2007;334:1198-200. (9 June.)
- 2 Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron* 1976; 16: 31-41.
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HIV AND PREGNANCY

Are we doing enough?

Gray and McIntyre report that the rates of mother to child transmission of HIV are dramatically reduced by antiretroviral use, caesarean section, and avoidance of breastfeeding.¹ However, none of these effective interventions can take place without awareness of the mother's HIV status.

In the United Kingdom, all antenatal clinics routinely offer HIV testing.² Most mothers accept screening. Two recent cases, however, highlight the deficiencies in the existing system. In 2006 the two infants were diagnosed with HIV within a few weeks of one another. Both mothers had had antenatal screening, and both tested HIV negative.

Current antenatal testing policies fail to take into account ongoing risk exposure. In addition, women who seroconvert during pregnancy are at a greater risk of transmitting HIV to their babies as the maternal viral load is at its highest at seroconversion. An alternative explanation is that both patients were tested during the serological window period. The information leaflet on HIV testing distributed in our antenatal clinic does not include an explanation about the HIV window period, ³ and retesting is not routinely offered to those at higher risk.

Subsequent to these two cases, local antenatal services have altered their HIV testing policies to offer repeat testing of high risk individuals at 32 weeks of pregnancy. Midwives are being advised to consider ongoing risks in all women. Contact tracing as is currently offered to HIV positive women should be offered to high risk HIV negative women as well. High risk women who initially refuse testing in pregnancy should be offered counselling by trained health advisers, with mechanisms in place to offer testing again later in their pregnancy. We see this as a safety net for those let down by the current antenatal system.

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Competing interests: None declared.

- 1 Gray GE, McIntyre JA. HIV and pregnancy. *BMJ* 2007;334:950-3. (5 May.)
- 2 Chief Medical Officer's Expert Advisory Group on AIDS. Reducing mother to baby transmission of HIV. London: Department of Health, 1999.
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GENETICS AND INSURANCE

Effect on premiums is small

Neither Holm nor Ashcroft addresses the quantitative question: how much difference would genetic information make to insurance prices?¹² Would banning insurers from access to genetic tests raise prices by 0.01% or 1% or 100%?

The answer is that it probably makes very little difference indeed. Certainly all estimates of the difference to date, under a variety of approaches and assumptions, have been negligible by comparison with the variations in insurance prices which exist for many other reasons.

To the very minor extent that prices do rise as a result of restricting insurers' access to genetic tests, this may not be a bad thing. In a competitive market, the logical corollary of an increase in insurance prices is an equivalent increase in claim payouts.

The effect of a ban—if there is any measurable effect, which is highly doubtful—is a small redistribution towards people who are affected by actuarially relevant genetic predispositions.^{3 4}

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Competing interests: None declared.

- 1 Ashcroft R. Should genetic information be disclosed to insurers? No. *BMJ* 2007;334:1197. (9 June.)
- 2 Holm S. Should genetic information be disclosed to insurers? Yes. *BMJ* 2007;334:1196. (9 June.)
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RICKETS

Prevention message is not getting through

Ten years ago vitamin D deficient rickets was diagnosed in six children in Manchester, which highlighted the need to implement the government's policy on vitamin D supplementation.¹ In Tayside in the past four months we have diagnosed vitamin D deficient rickets in five infants in an almost identical scenario. None of these children or mothers had received vitamin D supplementation. Their families were unaware of the need for this, despite the UK government recommendations for the universal use of vitamin supplements to all breastfeeding infants to prevent rickets, which have existed for over 10 years.² This recommendation is particularly important for those of Asian, African, Afro-Caribbean, or Middle Eastern origin with reduced exposure to sunlight.³

The NHS Direct website is not specific and is ambiguous about the need for vitamin supplements (www.nhsdirect.nhs. uk/articles/article.aspx?articleId=1122). The recommendations are laid out more clearly as a component of the "Healthy Start" initiative (www.healthystart.nhs.uk), which has replaced the welfare food scheme, but the uptake of vitamins was particularly low when this scheme was last audited. ⁴ None of the affected families we saw is eligible for this scheme as it is not directed specifically at immigrant groups.

The Scientific Advisory Committee on Nutrition has just published a position statement on vitamin D, with particular reference to preventing rickets, which highlights the need for a public health campaign and to supplement infants in high risk groups.⁵ The signs and symptoms of rickets were recognised by the general practitioner in only one of our cases. We must disseminate the message to all health visitors and general practitioners across the UK. Scott Williamson specialist registrar Stephen Greene consultant paediatrician Ninewells Hospital, Dundee DD1 9SY

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Competing interests: None declared.

- 1 Mughal MZ, Salama H, Greenaway T, Laing I, Mawer EB. Lesson of the week: Florid rickets associated with prolonged breast feeding without vitamin D supplementation. *BMJ* 1999;318:39-40.
- 2 HMSO. Department of Health COMA report on weaning and the weaning diet. 1994. Report No.: 45.
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- 5 Scientific Advisory Committee on Nutrition. Update on

MTAS

Response from the BMA

The MTAS debacle is the worst insult visited on the profession by any government in many years.¹ The priority now is to get as fair and transparent a solution as possible, which minimises further damage while also protecting patients.

The BMA will support the estimated 18000 applicants now left hunting for jobs in round 2. We have achieved a small number of extra posts to increase the chances in some of the most competitive areas, but we will lobby the government to find some more. These must be posts with real training and opportunities to progress to consultant (or general practitioner) status.

As round 2 kicks off it must be fair and transparent and contain both ST and FTSTA posts across the specialties and regions. A robust appeals mechanism must be opened forthwith to ensure that those unfairly treated by the system can be identified and given a secure return to training.

We have an assurance from the secretary of state that no one will be left unemployed in England between rounds 1 and 2. We will vigorously support any member made unemployed in this way (telephone 0870 60 60 828). And we are contributing to the independent review of the process under Sir John Tooke.

It is also crucial that the secretary of state heeds our calls, made last month, for a new body to design the future of postgraduate training. This work must start now if this painful episode is not to fester on, further sapping the morale of the profession and having a negative impact on patient care. Sam Everington acting chairman, BMA Council, Jonathan Fielden chairman, CCSC, Michael Rees chairman, MASC, Jo Hilborne chairman, JDC, Hamish Meldrum chairman, GPC, Chris Spencer-Jones chairman, CPHMCH BMA, London WC1H 9JP

Competing interests: None declared.

I Godlee F. The future of specialist training. BMJ 2007; 334;1067-8. (26 May.)