Letters

New UK policy on overseas doctors

What future for international medical graduates in the NHS?

EDITOR—Of 449 applications for six posts on our basic surgical training scheme which started in August 2005, 78% graduated from non-European Union (EU) countries, 61% coming from India. In all, 276 doctors from India applied for a place on a scheme in a district hospital. Anecdotal evidence suggests that only a small proportion of them gained positions on official training programmes (one to our rotation). Many of the rest will have "settled" for trust grade or staff grade posts, while many others will have been trapped in financially unrewarding clinical assistantships.

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Those in trust grade posts make a significant contribution to the overall medical staffing in the NHS. It remains unclear how this gap will be filled. There will not be sufficient numbers of UK graduates willing and able to fill such service posts. In our study only 6% of applicants came from non-UK EU countries.

At this time of crisis[1] the General Medical Council continues to hold Professional and Linguistic Assessments Board (PLAB) examinations in India, Africa, and other countries. This practice constitutes an indirect invitation by the GMC to come to work in the UK. Instead, these young doctors should be fully appraised of the new regulations and discouraged from coming, except in extraordinary circumstances of specific manpower shortages.

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Competing interests: OE is an international medical graduate working in the NHS, and NGR is a consultant who regularly works with international medical graduates.

1 Trewby P, Williams G, Williamson P, Barnes E, Carr P, Crilley J, et al. European doctors and change in UK policy. *BMJ* 2006;332:913-4. (15 April.)

International medical graduates invest £7500 in getting first job

EDITOR—The following is the approximate investment each overseas doctor has to put in before applying for jobs in the United Kingdom:

IELTS (language exam): £150
PLAB1 (medical exam): £145
PLAB2 (medical exam): £430
Provisional GMC registration: £100
GMC limited registration: £290.

As each of these doctors has to purchase a return ticket to their home country (around £500), spends £300 a month on subsistence, spends another £100 a month on job applications, takes one year to get the first job, and additionally has to pay a visa fee of about £400, add another £5700. Moreover, visas are only extended for the period of clinical attachment (1-3 months) and the candidate has to apply again. This amounts to a total of at least £7500 that an international medical graduate would have to spend before securing his or her first job in the United Kingdom. Multiplying this by the presumed 10 000 international graduates that might be affected by the new rules[1] gives £75 000 000 that the UK government is willing to take from international medical graduates from poor or developing countries.

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1 Trewby P, Williams G, Williamson P, Barnes E, Carr P, Crilley J, et al. European doctors and change in UK policy. *BMJ* 2006;332:913-4. (15 April.)

Also affects international medical graduates graduating from UK institutions

EDITOR—The Home Office has stipulated that from April 2006, UK medical school graduates who are foreign nationals will strictly fall under the work permit system on completing foundation training.[1] By the end of Foundation Year 2, I will have been in the UK for eight years—long enough to gain the building bricks of medicine, but not long enough to gain residency rights. I read medicine at Cambridge University with the expectations that I could continue postgraduate training here. Otherwise, what use is a primary medical degree without specialist training, unless one is thinking of leaving medicine, say, to go and work in the city?

If this ruling is not overturned, I suggest that undergraduate deans immediately stop accepting foreign students, regardless of the talent.

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Has far reaching effects

EDITOR—The new rule has affected not only trainee doctors but all the doctors who came to this country in the early 1960s and 70s to fulfil their dream and ambition of getting trained in the United Kingdom.[1] They were given the option of working as general practitioners in underprivileged areas and served the British public where local graduates dreaded to go. The new rules don't affect them, but there is a sense of being not wanted in the country as they favour European nationals. Not only doctors but professionals from other disciplines are quite seriously thinking about continuing in the UK for their careers. The UK is losing thousands of skilled staff, and at what cost?

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1 Khan MAI. Non-European doctors and change in UK policy. *BMJ* 2006;332:914. (15 April.)

Thank God for the American Dream

EDITOR—Trewby et al identify the new nationality based employment policy of the NHS as a short term benefit.[1] I am grateful that the institutionalised bias against foreign physicians in the United Kingdom was evident to me seven years ago when I decided to search for opportunities outside

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India. The US is still a bastion of equality and democracy, treating foreign medical graduates on merit and not being reluctant to hire them. The US shows that hiring people for talent rather than nationality is good for progress as well as ethically sound.

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1 Trewby P, Williams G, Williamson P, Barnes E, Carr P, Crilley J, et al. European doctors and change in UK policy. *BMJ* 2006;332:913-4. (15 April.)

New ethical framework for pharmaceutical physicians

EDITOR—Lenzer highlighted the helping hand that some US news organisations give to pharmaceutical companies in circumventing the Food and Drug Administration's requirements for fair balance in video news releases.[1] An ethical framework of guiding principles for pharmaceutical physicians has recently been published.[2] Pharmaceutical physicians should "ensure that expectations are not inappropriately raised as a result of media briefings" and "be involved in the drafting of any briefings about potential therapeutic interventions provided to financial analysts or to the media." Thus the disproportionate publicity that arose from studies such as ASCOT and ASTEROID—targeted directly at the consumer via the news media—should not occur when other trials are reported.[3 4]

The particularly dubious habit of reporting trials that achieve significance only for their secondary end points (ASCOT, PROACTIVE) with the same vigour and publicity as if the primary end point had been achieved should now also be relegated to history.[3 5] Details of how deviations from this guidance will be managed are necessary if the proposals are to be effective and taken seriously.

Thrown into sharp relief is the paucity of guidance for and regulation of health service physicians whose association and financial dependency on the pharmaceutical industry can seemingly approach that of pharmaceutical physicians. The General Medical Council might be best suited to considering these issues, but until this need is identified pharmaceutical physicians may, ostensibly, be held more accountable than their non-pharmaceutical colleagues. Perhaps it is now time to abandon the artificial dichotomy between pharmaceutical physicians and non-pharmaceutical physicians and recognise that similar strictures should apply to all.

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Competing interests: Over the past 10 years RM has attended educational meetings and received travel grants and honorariums for lectures and advisory boards from a number of pharmaceutical companies, including AstraZeneca, Bayer, Fournier, GlaxoSmithKline, Pfizer, Merck, MSD, and Sanofi-Aventis.

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Role of MRI in diagnosing multiple sclerosis

Magnetic resonance imaging is valuable

EDITOR—In investigating the diagnostic utility of magnetic resonance imaging (MRI) in cases of suspected multiple sclerosis, Whiting et al have evaluated imaging findings reported in many different studies—mainly whether there are any lesions present in a brain scan.[1] This approach does not reflect real life, where neurologists use a more detailed interpretation of MRI abnormalities in the context of the clinical findings to reach a diagnosis. Clinicians deal with many different clinical settings that make the diagnosis of multiple sclerosis more or less likely and also have to consider the differential diagnosis. An early and reliable diagnosis facilitates best management and alleviates anxiety due to diagnostic uncertainty. While the diagnosis of multiple sclerosis is based primarily on clinical manifestations, it is often helpfully—and sometimes crucially—supported by laboratory investigations. When used appropriately, MRI—and sometimes cerebrospinal fluid and neurophysiological (evoked potentials) examination—improves diagnostic accuracy and helps exclude or identify other important conditions.[2]

Appropriate use of MRI in cases of suspected multiple sclerosis involves more than determining whether there is a lesion in the brain and if so how many. White matter lesions have numerous causes, and the correct use of brain imaging to improve specificity in suspected multiple sclerosis will take into account location (Barkhof-Tintore criteria for dissemination in space[3]), activity (gadolinium enhancement), and the appearance of new lesions (dissemination in time, a mandatory requirement in diagnosing multiple sclerosis[3]). The currently accepted brain MRI criterion for dissemination in space has a higher specificity than three lesions for multiple sclerosis versus other neurological diseases.[3 4] Detection by MRI of the characteristic spinal cord lesions of multiple sclerosis is of particular diagnostic value.[5] Because a cord syndrome is the presenting feature of around a half of patients with multiple sclerosis, imaging of this region is often needed to exclude an alternative treatable disorder such as spinal cord compression.

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On behalf of members of the Steering Committee of MAGNIMS, a European network on magnetic resonance in multiple sclerosis.

The members of the steering committee who are coauthors of this letter are Frederik Barkhof, Franz Fazekas, Massimo Filippi, Ludwig D Kappos, Xavier Montalban, Jacqueline Palace, Chris H Polman, Marco Rovaris, Alex Rovira, Nicola de Stefano, Alan J Thompson, and Tarek Yousry.

Competing interests: None declared.

1 Whiting P, Harbord R, Main C, Deeks JJ, Filippini G, Egger M, et al. Accuracy of magnetic resonance imaging for the diagnosis of multiple sclerosis: systematic review. *BMJ* 2006;332:875-84. (15 April.)
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Early diagnosis using MRI and early treatment delays disease conversion

EDITOR—Whiting et al ignore data collected over the past 15 years that show repeatedly that early diagnosis of multiple sclerosis is crucial and that early treatment leads to a far better outcome on numerous measures, immunological and clinical, than late treatment.[1]

In patients with one attack of multiple sclerosis starting immune therapy with interferon beta-1a can delay the patient meeting diagnostic criteria for clinically definite multiple sclerosis compared with untreated patients, and the untreated patients never quite catch-up when they eventually begin immune therapy.[2] At this month's 58th annual meeting of the American Academy of Neurology San Diego another study (BENEFIT) using interferon beta-1b confirms the same point (M S Fredman; C H Polman, scientific sessions).

The management of multiple sclerosis has slowly been moving towards early

diagnosis and treatment because it is the best way yet known to avoid the accumulation of significant deficits in the daily life of patients and to afford them the best quality of life and health possible for the longest possible time. That is why so much effort has been put into magnetic resonance imaging (MRI) studies and early treatment trials.

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1 Whiting P, Harbord R, Main C, Deeks JJ, Filippini G, Egger M, et al. Accuracy of magnetic resonance imaging for the diagnosis of multiple sclerosis: systematic review. *BMJ* 2006;332:875-84. (15 April.)
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Textbook of Pharmaceutical Medicine warns of trial risks

Editor—With reference to the adverse reactions with TG1412 being because of the drug, not the study design,[1] I quote from section 4.8 on minimising risk in the chapter on exploratory development in the *Textbook of Pharmaceutical Medicine* on first time dosing in humans[2]:

"For example, the study design may require administration of intravenous infusions to six volunteers. It may be perfectly feasible to perform these on a single day but it is inadvisable to start all the infusions simultaneously. Drug-related adverse reactions would be likely to occur at the same time in all the subjects, which could be very difficult to manage and put subjects at unnecessary risk. Indeed, it may be wise to stop the study after the first significant adverse reaction has been detected and reconsider the dose, speed of administration or whether to proceed at all."

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Competing interests: None declared.

1 Mayor S. Inquiry into adverse events in trial blames drug, not study design. *BMJ* 2006;332:870. (15 April.)
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Mobile phone use and risk of glioma in adults

Results are difficult to interpret because of limitations

Editor—The UK part of the Interphone study concluded that mobile phone use is not associated with an increased risk of glioma.[1] However, ≥ 10 years ipsilateral use yielded an odds ratio of 1.60 (95% confidence interval 0.92 to 2.76) and contralateral use an odds ratio of 0.78 (0.85 to 1.3).

Only 51% of the cases and 45% of the controls participated. Controls were more

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affluent than non-participating controls and participating cases. Mobile phone use is associated with social class. In our study use of cellular telephones was reported by 48% of the most affluent cases and 36% of the least affluent.[2 3]

Use of cordless telephones was not assessed and in the analysis of laterality the "unexposed" group contained subjects with exposure to microwaves on the opposite side of the head.

In table 3, 13 of the 14 odds ratios are <1.0 and one is >1.0, indicating non-random variation. Patients with brain tumours (cases) may not be best interviewed face to face shortly after their operation because of cognitive behavioural defects such as memory loss and aphasia. The interviewers knew that it was a case under interview.

Our publication on malignant brain tumours on this topic is not cited, though available on 14 July 2005.[4] We found an increased risk for high grade astrocytoma with >10 years' latency. The current publication does not give results for high and low grade glioma separately.[1]

The article cites critics of our studies published even before our results appeared in the scientific literature. Two of the cited reports have never been published in a peer reviewed journal and are not possible to rebut. The third cited report was published in 2000, when our first large case-control study was ongoing and no data had been reported.

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1 Hepworth SJ, Schoemaker MJ, Muir KR, Swerdlow AJ, van Tongeren MJA, McKinney PA. Mobile phone use and risk of glioma in adults: case-control study. *BMJ* 2006;332:883-7. (15 April.)
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4 Hardell L, Carlberg M, Hansson Mild K. Case-control study on the association between the use of cellular and cordless telephones and malignant brain tumors diagnosed during 2000-2003. *Env Res* 2005. doi:10.1016/j.envres.2005.04.006.

Study has many flaws

Editor—In years past Hepworth et al's study would never have been published because a low participation rate would have been cause for rejection.[1] With 51% of cases and 45% of the controls participating there is little reason to believe any of the reported results. There are additional flaws:

- Controls were more affluent controls than cases
- Non-participating controls were more likely than participating controls not to use cellphones[2]
- The reference group was never/non-regular cellphone users. Because this reference did not exclude the users of cordless phones, the reference group cannot be described as unexposed
- Regular cellphone use is defined as cellphone use for at least once a week for six months or more. Regular cellphone use is set to such a minimal standard that few could imagine a finding of risk.

In spite of these flaws, the study reported a 60% increased risk of glioma for regular cellphone use of ≥ 10 years of ipsilateral use.

Interphone studies receive cellphone industry funds isolated from a study. This same conflict of interest issue can be seen in the US government's Food and Drug Administration (FDA) where pharmaceutical companies pay fees for drug approval isolated from specific research projects. It is quite apparent that the FDA has come to see the pharmaceutical industry as their customer, not the American public.[3–5]

If this were a study of the risk of lung cancer from smoking would there be a likelihood of finding a risk of lung cancer from smokers who had smoked at least once a week for six months or more? And, would there be a finding of risk if, as is the case in this study for cellphone use, the lifetime years of smoking for 10 years or more included only 3.9% of the smokers in the study?

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1 Hepworth SJ, Schoemaker MJ, Muir KR, Swerdlow AJ, van Tongeren MJA, McKinney PA. Mobile phone use and risk of glioma in adults: case-control study. *BMJ* 2006;332:883-7. (15 April.).
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Conclusions are questionable

Editor—The study by Hepworth et al has severe shortcomings, including faulty interpretation and unfounded conclusions.[1] Is there really any occupational or environmental factor capable of inducing glioma in a period of 3 to 4 years (the average duration of use of a mobile phone in this study)? Not

even after high doses of therapeutic x rays have such short latencies been observed.[2 3]

Only 5% of cases had used a mobile phone for 10 or more years. Therefore, induction of glioma cannot be studied. Only an effect on tumour development and growth can possibly be detected. As pointed out,[4 5] the case-control design is inefficient to study such effects if the duration of exposure is short.

Furthermore, if an effect on an already premalignant lesion is studied only exposures to that region are exposures at all. Therefore the only relevant analysis is that of laterality. And, surprisingly, this analysis resulted in a significantly increased risk that increased further if longer exposure durations were considered. Hence the only analysis compatible with the natural history of the disease and exposure conditions showed a significantly increased risk. But still the authors conclude that the study found no increased risk of developing a glioma associated with mobile phone use. They point to the fact that the odds ratio for contralateral exposure is below 1 and seem to interpret this as an indication for recall bias. However, this is simply a consequence of their method of analysis and of the significant effect on the ipsilateral side.

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Competing interests: None declared.

1 Hepworth SJ, Schoemaker MJ, Muir KR, Swerdlow AJ, van Tongeren MJA, McKinney PA. Mobile phone use and risk of glioma in adults: case-control study. *BMJ* 2006;332:883-7. (15 April.)
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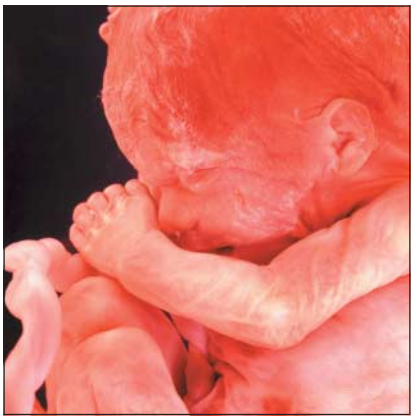
5 Kundi M, Hanson Mild K, Hardell L, Mattsson M-O. Mobile telephones and cancer—a review of epidemiological evidence. *J Toxicol Environ Health B* 2004;7:351-84.

Fetuses can feel pain

Editor—Derbyshire argues against the ability of fetuses to feel pain.[1] He states: "Good evidence exists that the biological system necessary for pain is intact and functional from 26 weeks." He then adopts a definition of pain from the International Association for the Study of Pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage" but concludes that pain is "a conscious experience" rather than "merely the response to noxious stimuli," so a fetus cannot experience pain.

This is a specious argument. There are many examples of the ability of babies of this gestation to feel pain. In the first few moments after birth, even with extremely premature neonates (23-26 weeks), a noxious stimulus—for example, phlebotomy—can cause bradycardia, desaturation, and



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hypertension as a stress response. A neonatologist would seek to relieve this distress with analgesia, and a parent would seek to soothe. Also, as Derbyshire notes, fetal procedures (such as in utero chest drain placement) are increasingly being carried out with analgesia.[2]

The problem lies with his definition of pain and the subsequent development of his argument. If a pregnant woman asks whether the fetus feels pain a conscious rationalisation is not necessarily implied. The *Oxford English Dictionary* instead describes pain as "a strongly unpleasant bodily sensation such as is produced by illness, injury or other harmful physical contact."[3] There is thus a gap between the definition Derbyshire has adopted and his patients' understanding of pain, resulting in ill-informed counselling or, worse (as he encourages in his conclusion), "avoiding a discussion of fetal pain with women." His argument that fetuses cannot feel pain needs correcting.

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Competing interests: None declared.

1 Derbyshire SWG. Can fetuses feel pain? *BMJ* 2006;332:909-12. (15 April.)
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Predictive value of metabolic syndrome is not clear

Editor—Sundstrom et al claim that in their cohort of 50 year old men the identification of the metabolic syndrome (as defined) added to the prediction of total and cardiovascular mortality obtained from classic risk factors.[1]

However, the electronic version of the article clearly shows that this superiority emerges only after about 15 years of follow-up. As most guidelines for therapeutic intervention are predicated on 10 year risk, the observation does not have pragmatic value.

Furthermore, the comparatively poor performance of the classic risk factors seems to be due to the unusually low predictive power of total cholesterol in this cohort, which suggests that the result would not be generally applicable.

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1 Sundström J, Risérus U, Byberg L, Zethelius B, Lithell H, Lind L. Clinical value of the metabolic syndrome for long term prediction of total and cardiovascular mortality: prospective, population based cohort study. *BMJ* 2006;332:878-82. (15 April.)

Clinical research in primary care needs urgent boost

Editor—Let us hope that the cogent arguments advanced by Rothwell in support of more and better clinical research in the United Kingdom begin a serious dialogue that will correct the asymmetries in research funding, kudos, and leadership that have developed over the past 5-10 years.[1] Rothwell's arguments apply with even greater force to the need for a clinical research effort in primary care and in health services research on topics such as the natural history of common diseases; the value of interventions, both therapeutic and preventive; and critically, as Rothwell points out, the difficulties of individualising risk and benefit in a single patient on the basis of large scale trials.

In addition to the clinical research networks described in the new NHS research and development strategy, adequate project and programme funding must be made available to support the research that Rothwell identifies. This includes follow-up studies of large cohorts of patients after the completion of therapeutic trials, health economic evaluations of interventions, studies of the success or otherwise of getting newly proved interventions into practice, research aimed at improving understanding of patients' willingness to accept therapeutic and preventive interventions, and high quality health services research to define the optimum ways of providing new services.

There is an urgency about this. The impending research assessment exercise has led to quick-fix institutional solutions, including playing the research star transfer market and redistributing clinical academic funding to support laboratory based research. Non-clinical researchers underestimate the need for clinical research, partly because they simply can't see it and partly because it is likely to be uncomfortable in terms of their own priorities. They need to understand that translational doesn't simply mean getting the protein out of the test tube into the zebra fish, but getting the therapeutic intervention into the patient and the population.

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Competing interests: None declared.

1 Rothwell PM. Medical academia is failing patients and clinicians. *BMJ* 2006;332:863-4. (15 April.)