LETTERS

Letters to the Editor

Please e-mail letters for publication to Dr Kamran Abbasi [kamran.abbasi@rsm.ac.uk]. Letters should be no longer than 300 words and preference will be given to letters responding to articles published in the *JRSM*. Our aim is to publish letters quickly. Not all correspondence will be acknowledged.

Too little quality; too many doctors

The editorial 'Too little quality; too many doctors' (June 2006 *JRSM*[1]) is incorrect to suggest that the General Medical Council (GMC) had a 'sudden demand for overseas graduates to have a work permit'. The GMC does not have any visa requirements for international medical graduates (IMGs). The Home Office is responsible for setting visa requirements. International medical graduates are responsible for ensuring that they have the appropriate visa when they enter the UK.

Arun Natarajan and Balasubramanian Ravikumar in their editorial (June 2006 *JRSM*[2]) appear to argue that the PLAB test be used as a means to control the movement of international medical graduates. The PLAB test enables IMGs to demonstrate that they have the knowledge and skills required for registration with the GMC. It is not, and cannot be used as, a tool for controlling the number of doctors entering the UK or for determining who should get jobs. To withdraw or ration the PLAB test would deny IMGs the opportunity to demonstrate their knowledge and skills and to compete for jobs. Apart from anything else, this would be unfairly discriminatory and unlawful.

We have consistently argued that IMGs need much better information about potential job and career prospects, in order that they can make properly informed decisions before coming to the UK. We have provided feedback from our surveys of the employment experience of those who have passed the PLAB test; and IMGs who wish to take the test must confirm that they have read our warning statements. There has been a very sharp reduction in applications to sit the test and, as a result, we are reducing the number of test sessions both within the UK and overseas.

We will continue to work with others to ensure that doctors are able to make well informed decisions. My colleagues and I have very considerable sympathy for the plight of doctors already in the UK and we stand ready to contribute, with others, to trying to find a solution to their problems.

Competing interests None declared.

Amanda Watson
Director of Registration and Education, General Medical Council, London, UK
E-mail: pressoffice@gmc-uk.org

REFERENCE

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2 Natarajan A, Ravikumar B. Requiem for international medical graduates. *J R Soc Med* 2006;**99**:272–3

Peer review

Peerless review

Peer review is indeed '. . . a flawed process, full of easily identified defects with little evidence that it works', as Richard Smith concludes in his thoughtful article (April 2006 *JRSM*[1])—but even flawed refereeing is better than not being peer reviewed at all. Increasingly, prestigious journals put submissions through a preliminary triage before they are even allowed to be seen by real scientists. This pre-selection process aims to identify 'sexiness'; however important a paper in terms of advancing its subject, if it is not regarded as sufficiently topical or is too intellectually demanding (perhaps it has some maths) it will be returned with a computer-generated letter to the effect that it would be better 'in a more specialized journal'. Typically, a request for further information about exactly how the paper failed to meet the pre-selection criteria is met with further boilerplate generalities: the process is secret and unregulated.

Of course, journals such as *Nature* and *Science* are under pressure from the huge increase in submissions that has resulted from the whole-scale adoption of impact-factor bibliometrics in the research assistant excersise. But sooner or later scientists are going to ask whether it is worth wasting time on this demeaning and dispiriting ritual, and whether perhaps the internet is now grown up enough that we can cope with web publication—and dispense with the luxury of peer review. Once publication *per se* confers no particular prestige, the cancerous over-publication that afflicts us all will be stopped in its tracks. The metastatistics generated by Google and Amazon demonstrate how one can use the extraordinary power of the internet itself to create systems that prevent one drowning in unreliable information. Would a free-for-all be so very terrible?

R H S Carpenter
The Physiological Laboratory, University of Cambridge CB2 3EG, UK
E-mail: rhsc1@cam.ac.uk

Competing interests None declared.

REFERENCE

1 Smith R. Peer review: a flawed process at the heart of science and journals. *J R Soc Med* 2006;**99:**178–82

Unreliable reviewers

As Richard Smith reports (April 2006 *JRSM*[1]) peer review would be fine if reviewers always gave an impartial, critical assessment when asked—but they may, he indicates, steal ideas, give a good opinion simply because they want to increase work in their field or give a bad opinion because they do not want competition, or other biases.

Long-serving members of research and publication committees will be aware of unreliable reviewers, and one solution to the problem would be to create a blacklist of reviewers found to be unreliable. This should cut down on the problem but would leave open the question of whether there is a better system than peer review.

Competing interests None declared.

Brandon Lush
Lower Hazel, Bristol BS35 3QP, UK
E-mail: brandon.lush@eidosnet.co.uk

REFERENCE

1 Smith R. Peer review: a flawed process at the heart of science and journals. *J R Soc Med* 2006;**99**:178

Conflicts of interest that are bigger than money but never disclosed

Conflicts of interests in academia are indeed a web of intrigue, as eminently portrayed by Smith.[1] There are certain conflicts of interest though, which are married to research and are bigger than money, and yet are never disclosed. Glory of publishing and career progression are two such factors. The purpose of research is to answer a question; but are all researchers honest to this purpose? What happens when research becomes a means for achieving personal gain? Consider the case of young impassioned clinicians, who delve into research. Their goal is to append as much weight to their curriculum vitae as possible by 'getting publications'. How committed are they towards forwarding science? Senior academics are no less prone to such conflicts of interest with elements such as the research assessment exercise lurking in the background. Hunger for authorship, recognition and position can be as corruptive as the apple of Adam. They will, unfortunately, probably remain the bugbears of research, forever.

Competing interests None declared.

Arun Natarajan
DRWF Clinical Research Fellow, Newcastle University, Newcastle upon Tyne, UK
E-mail: Arun.Natarajan1@newcastle.ac.uk

REFERENCE

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Non-traumatic rhabdomyolysis: the emerging role of CYP 3A4 in diabetes mellitus

Gangopadhyay and Ryder (April 2006 *JRSM*[1]) highlight an important message about deranged metabolic control in the setting of diabetes mellitus. Of late, the role of CYP3A4 (a cytochrome-P450 isoenzyme involved in the metabolism of various drugs) is also gaining further momentum, more so in the context of diabetes mellitus.[2] As an illustrative example, amiodarone is a recognised inhibitor of this isoenzyme and many statins are metabolised primarily by CYP3A4, which can result in statin induced non-traumatic rhabdomyolysis in patients with diabetes mellitus.[3,4]

A recent systematic review suggested that the incidence of rhabdomyolysis was 4 times higher for monotherapy with statins like lovastatin, simvastatin, or atorvastatin which are oxidized by CYP3A4 (mean-rate=0.73; 95% CI: 0.64–0.82/million prescriptions), compared to monotherapy with pravastatin or fluvastatin that are not oxidized by CYP3A4 (mean-rate=0.15; 95% CI: 0.09–0.24/million prescriptions, $P<0.001$).[2] In persons taking simvastatin, lovastatin, or atorvastatin, 60% of cases involved drugs known to inhibit CYP3A4, and 19% involved fibrates, principally gemfibrozil; a substrate of CYP3A4.[2] Examples of further commonly used drugs that inhibit CYP3A4 include diltiazem, erythromycin, azole antifungals, ritonavir, cyclosporine and also grapefruit juice.[2] Of note, pioglitazone, clopidogrel and colchicine are also related to CYP3A4 in their metabolism. Patients who received a lipid-lowering medication with a concomitant CYP3A4 inhibitor have been demonstrated to have a 6-fold increased rate of muscle disorders, including non-traumatic rhabdomyolysis.

The risk of arrhythmias with diabetes mellitus is high,[5] and amid the increasing benefits of statins in diabetes mellitus, as well as decreasing threshold to use statins, clinicians should be vigilant about muscle-related complaints, especially in elderly patients on multiple medications. Avoiding or suspending the concomitant use of drugs metabolised through the CYP3A4 system or alternatively, if drug therapy with a potent CYP3A4 inhibitor is inevitable, choosing a statin without relevant CYP3A4 metabolism should be considered. Indeed, the potential for these complications are less commonly perceived in routine daily clinical practice.

Competing interests None declared.

George I Varughese,[1]
John HB Scarpello[1,2]
[1]Diabetes & Endocrinology, University Hospital of North Staffordshire, Stoke-on-Trent ST4 6QG
[2]Keele University, UK
E-mail: georgeiv@doctors.org.uk

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Diagnosing malaria in UK migrants from sub-Saharan Africa

Semi-immune individuals, such as the patient described by Allan and Tahir (April 2006 *JRSM*[1]), present a diagnostic challenge in UK emergency departments due to the relatively uncommon presentation of malaria in these settings. The patient probably had recrudescent *Plasmodium falciparum* infection, caused by persistence of blood forms of *P. falciparum* in small numbers between attacks. Though exposed to infection in her country, the authors did not state if she had clinical infection prior to leaving, if it was treated, or if she took prophylactic medication before or after arriving in the UK.

Widespread anti-malarial drug resistance in Africa makes the possibility of recrudescent infection more significant.

The Health Protection Agency guidelines would thus provide the greatest sensitivity and specificity for diagnosing malaria.[1] Thrombocytopenia which is a relatively common presentation of malaria in Nigerians and other Africans in western countries,[2,3] may be a vital clue to the diagnosis in busy emergency departments.

Competing interests ATA is a Nigerian, and a senior house officer in Accident & Emergency medicine on the GPVTS of the Kent, Sussex, and Surrey postgraduate deanery.

Ayokunle T Abegunde
Directorate of A&E Medicine, William Harvey Hospital ,
Kennington Road, Ashford, Kent, TN24 0LZ
E-mail: abegs@lycos.com

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Bilateral chronic subdural haematoma

The proposition that chronic subdural haematoma (CSH) is largely amenable to surgical treatment[1] can only be substantiated, in the short term, by a national audit of neurosurgical practice, and, in the long term, by a randomized controlled trial. The latter strategy was the one suggested by a leading neurosurgical authority who noted that there were no randomized controlled trials to yield class I evidence supporting any of the treatment options for chronic subdural haematoma; and that the available class II evidence relied on only six trials.[2]

What is even more worrying is the reliance, at least in one instance, on case control studies such as the one dating back to 1992,[3] which was subsequently cited as recently as 1999 to support a proposed management option for CSH.[4] In essence, that option was based on the premise that '. . . there is little point in active treatment over the age of 65 for those who remain in coma (Glasgow Come Scale 8 or less) for more than 6 hours . . .'.[4] Such statements, I maintain, should, at least, be qualified by the acknowledgement that, given the biological diversity of the over 65s, clinical outcomes in that age group might be better predicted by the biological age/physiological reserve index than by chronological age.[5]

Oscar Jolobe
Medical Division, Manchester Medical Society,
c/o John Rylands University Library, Manchester M13 9PP, UK
E-mail: oscarjolobe@yahoo.co.uk

Competing interests None declared.

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Medical theories on the cause of death in crucifixion

I write as one who for many years has taken in the *Shroud of Turin;* and who has visited Turin twice to see this piece of linen bearing on its surface the imprints of a man whose body has been subjected to torture by flagellation, wounding of the head and piercing of the hands and feet. I would comment on the paper by Maslen and Mitchell (April 2006 *JRSM*[1]) entitled 'Medical theories on the cause of death in crucifixion'. The authors contend that there is fair evidence that the shroud of Turin is a forgery.

Before the forgery theory gains acceptance a few facts are worthy of consideration.

The history of the shroud is well documented from the middle of the 14th century, the greatest artist at that time being Giotto who was unable to produce such a detailed and accurate depiction of the human body as is shown on the Shroud. The image of the body is anatomically flawless, its features being particularly well revealed when its light values are reversed as in a photographic negative.

No one has been able to reproduce the image as seen on the shroud which, unlike a painting or photograph, contains three-dimensional information and is made without a single brush stroke

In respect of carbon dating, a peer review scientific paper by Raymond Rogers, retired fellow of the Los Alamos National Laboratory, was published on 20 January 2005.[2,3] The author writes: 'As unlikely as it seems, the samples used to test the age of the shroud of Turin in 1988 was taken from a rewoven area of the Shroud. Pyrolysis-Mass spectrometry results from the sample area coupled with microscopic and microchemical observations prove that the radiocarbon date was not valid for determining the true age of the shroud'.

John Scotson
Retired General Practitioner, Cheshire WA14 2AN, UK
E-mail: jhs@doctors.org.uk

Competing interests None declared.

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Plague, rats and the Bible again

In a letter to this journal[1] I suggested that the 'plague of the Philistines' (I Samuel chapters 5 and 6) was the first documented account of bubonic plague, and remain of that view despite the recent correspondence of Russell.[2,3] He suggests the plague referred to as 'emerods' was dysentery complicated by piles. Russell's view stems from Whiston's translation[4] of Josephus's history of these events as dysentery. However, elsewhere Whiston translates the same word as distemper.

The original Hebrew text uses two words used to describe the plague's pathology namely 'techorim' (tumour) and 'ophel' (boil).[5] The King James version translates both words as emerods, and the New International version translates both as 'tumour'.

The Septuagint translation made in Alexandria in third century BCE from Hebrew into Greek, and St Jerome's translation of this Greek text into Latin, both expand the original Hebrew by stating the tumours were in the groin. (Bubo is derived from the Greek word for groin.) It therefore seems that the 72 Hebrew scholars who made the Septuagint translation were thinking in terms of Bubonic plague—this seems to me to be a sounder basis for a diagnosis than Josephus!

Competing interests None declared.

John P Griffin
Quartermans, Welwyn, Hertfordshire AL6 0SP, UK

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CORRECTION

Thomson DJ, Soni A, Ward M, Jones HW. Simultaneous presentation of myasthenia gravis and mesothelioma. *J R Soc Med* 2006;**99**:259–260

Please note that the wrong CT scan was inserted for Figure 2 of this paper. The correct scan is shown below.

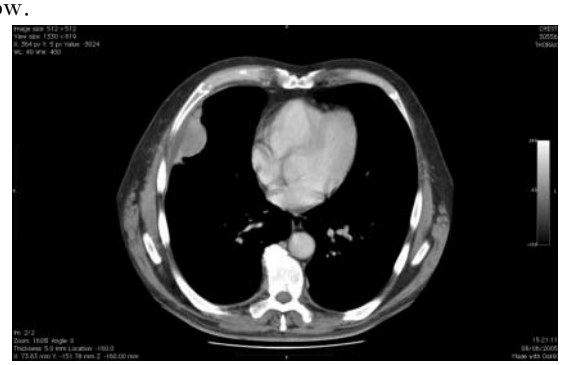

Figure 2 **Thorax computerized tomograph showing pleural mass**