around the world, the topics covered range from regenerative ability in the central nervous system and cerebral reorganisation after stroke to the evidence base for therapy, the use of imaging and the use of technology in rehabilitation. Complications of stroke such as incontinence, visual impairments, balance disorders, aphasia, depression, pain, sleep disorders and cognitive problems are all dealt with separately; however, the paucity of stroke-specific studies means that it is often necessary to rely on research done in other neurological conditions. What is missing is a comprehensive review of the burden of disability following stroke. The introductory chapter on epidemiology, aetiology and avoiding recurrence perversely avoids presenting the data on the prevalence of impairments and disability in cerebrovascular disease which would put the rest of the book into context, and instead discusses issues more appropriate to a text on stroke prevention. Chapters lack a consistent framework and some, such as the one on movement disorders, are disappointing in that they fail to address the management of the eloquently described problems.

Two chapters in particular are worth highlighting because they deal with issues that are common but rarely discussed in the research literature. The chapter on sexual dysfunction brings together information that is of huge importance to patients but seldom discussed with them. From the studies quoted over half of stroke sufferers experience a deterioration in sexual performance or satisfaction leading to discontentment, and yet where are the research studies exploring possible treatments? I was disappointed that the wisdom or otherwise of using drugs, such as sildefanil, after stroke is not even mentioned, as it is one of the questions that I am most often asked by patients when discussing post-stroke impotence. The chapter on 'Depression and fatigue after stroke' likewise covers a frequently neglected symptom, presenting fascinating data on the assessment, epidemiology and association with lesion location. There has however not been a single interventional study in this area despite it affecting up to half of all stroke victims. The final chapter by Donal O'Kelly, giving the patient's perspective, should in my opinion be read first because it contains a vivid description of what it feels like to have a stroke and puts the rest of the book into context.

Despite my few minor quibbles this book that has a great deal to recommend it. Having read it, I have come away feeling that there is an enormous amount about stroke that I didn't know, that there are a huge number of unanswered questions that need more research, and that the services my patients receive after stroke leave much to be desired.

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letters TO THE EDITOR

Please submit letters for the Editor's consideration within three weeks of receipt of the Journal. Letters should ideally be limited to 350 words, and can be submitted on disk or sent by email to: Clinicalmedicine@rcplondon.ac.uk.

International Medical Graduate Training

Editor – I read with interest the articles on improving international graduate medical education in the UK (*Clin Med* March/April 2005 pp 126–32; *Clin Med* March/April 2005 pp133–6).

Whilst I applaud the aims of the proposals and sponsorship schemes. I think it is important not to lose sight of the effect of medical migration on a country's ability to provide healthcare in both the short and long term.

The hospital I work at in Rural KwaZulu/Natal has 10 doctors to serve a population of 250,000 people. Next year five of the doctors are planning to go to the UK after finishing their compulsory year of community service. Admittedly, we will probably be sent some replacement junior doctors who will again leave and emigrate after one year of service. I doubt rural hospitals such as ours will benefit from the return of highly trained physicians who will usually situate themselves in tertiary institutions or private practice, away from the population that needs them most.

I hope the proposed schemes will be tailored to the needs of the country sending graduates for training and that there will be follow-up to ensure that trainees return home and work where they will benefit their community most.

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Rheumatoid arthritis and *Proteus*

Editor – The article by Dubey and Gaffney (*Clin Med* May/June 2005, pp 211–14) may have given the wrong impression to CME readers, as the authors describe rheumatoid arthritis (RA) as 'a disorder of unknown aetiology'.

We have used the concept of 'molecular mimicry', which in the past worked for rheumatic fever, and adapted it for the study of RA. Patients suffering from RA in England were shown to have elevated levels of antibodies to the urinary microbe, *Proteus mirabilis*.[1]

Molecular mimicry has been demonstrated between the 'susceptibility sequence'

EQKRAA, which is found in 90% of patients with RA in the HLA-DR1/DR4 molecules, and the ESRRAL sequence found in the *Proteus* haemolysin molecule.[2] The frequency of HLA-DR1/DR4 in the UK is around 35%, so about every third person in the UK has these genes.

Subsequently we suggested that RA could be a form of 'reactive arthritis' following *Proteus* urinary tract infection and this explains why women are more likely to develop RA than men. In a survey of 1,375 RA patients from 14 different countries, it was shown that RA patients have specific antibodies to *Proteus* when compared to blood donors,[3] and furthermore that isolation of urinary *Proteus* bacteria in RA patients correlates with antibodies to *Proteus* in serum.[4]

These antibodies are specific, since Japanese RA patients have antibodies to *Proteus* but not to *Klebsiella* whilst Japanese patients with ankylosing spondylitis (AS) have antibodies to *Klebsiella* but not to *Proteus* – thus each microbe is a specificity control for the other disease. Similar results have been obtained with Dutch patients.

The RA sera are cytotoxic for sheep red cells coated with EQRRAA or ESRRAL peptides, whilst AS sera are cytotoxic for sheep red cells coated with HLA-B27 peptide sequences.[5] This strongly suggests that RA sera are damaging to joint tissues and that *Proteus* infection of the upper urinary tract is the most probable cause of RA, in the same way that *Klebsiella* infection of the colon is the cause of AS.

At the borderline between the known and the unknown there will always be debate and CME readers should be aware of this problem.

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Diagnosis and management of vertigo

Editor – Halmagyi's very useful review of vertigo (*Clin Med* March/April 2005 pp 159–65) did nevertheless contain some contentious points.

Figure 1 in his article shows a repositioning manoeuvre for benign paroxysmal positioning vertigo (BPPV). Small black particles are seen to drop down inside the posterior semicircular canal. As these otoconia 'fall away from the cupula they create a negative fluid pressure that pulls on the cupula', producing vertigo. What is the evidence for this force? If, say, stones are dropped inside a beaker of water, is there downward pressure on the water surface, increasing the incurvation of the surface film and so decreasing the volume of the water? Common sense and schoolboy physics suggest to me that BPPV is far more plausibly caused by trapped air bubbles moving around inside the bony labyrinth.[1] So what is the flaw in this simple idea?

He suggests that in transient vertebrobasilar ischaemia, unilateral auditory symptoms suggest an aural problem, but 'by contrast, sudden, temporary bilateral hearing loss does suggest brainstem ischaemia', referencing the authoritative team of Lee, Yi and Baloh.[2] This case was of a diabetic woman who, two days previously, had two episodes of transient vertigo with hearing loss in the left ear, which, being unilateral, Halmagyi would characterise as an aural rather than a brainstem problem. Her audiograms with their predominant reversible low-tone loss strike a chord with me, as when I worked with Professor R Hinchcliffe he offered to pay me £10 for every low-tone sensorineural loss I found that was not of cochlear origin. I never did collect any money! Any doubt as to cochlear origin was removed by finding normal stapedial reflex thresholds, and Lee *et al* found the 'prominence of hearing symptoms … best explained by the selective vulnerability of the cochlea to ischaemia'.

In late Meniere's disease, Halmagyi describes the deaf ear that distorts and recruits ('There is no need to shout!'). This muddles two distinct loudness concepts.[3] Recruitment occurs in cochlear deafness wherein soft sounds are not heard, moderate sounds seem quieter and suprathreshold sounds are heard normally, and, as in the case above, acoustic reflex thresholds are unchanged. In contrast, audiosensitivity is where some intense sounds seem unusually loud. This is often an early symptom of Meniere's disease before any shift in pure tone thresholds. It is important to make this distinction or else the audiosensitive patient with normal audiogram will be sent to the neurology or psychiatry department rather than the ear, nose and throat department. Just as it is unhelpful to tell older patients with audiovestibular symptoms and vascular risk factors that they must have had a stroke, so it is to tell younger audiosensitive patients that they must have problems in the brain or, worse, in the mind, rather than having a simple, usually temporary, ear malfunction. Audiosensitivity and slight endolymphatic hydrops are common since they can result from cochlear hypotension from low blood pressure, weight loss, dehydration etc.[3]

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In response

Editor – Dr Gordon makes three thought-provoking points.

Firstly, he says that semicircular canal