LETTERS TO THE EDITOR

Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Clinical skills in final year medical students

Like many older readers (I qualified in 1976), I have been somewhat bemused by Goodfellow and Claydon's report (October 2001 *JRSM*[1]), and the subsequent correspondence.

First of all, we should acknowledge that the students of today are at least as able as their fore- (and in many cases, real) fathers. The 'failings' in the eight core clinical skills (venepuncture, venous canulation, rectal examination, nasogastric intubation, suturing, urinary catheterization, ECT recording and arterial blood sampling) are significant because these, along with lumbar puncture, inserting chest drains and using an ophthalmoscope, are what our 'duties' were as medical clerks and surgical dressers admitting patients. Our generation valued competency as an aim in itself, a view shared by our bosses in sharp contrast to today's views on teaching.

Is what is needed prolonged clinical attachments to anaesthetic rooms as Mr Sado suggests[2] or a 'skills laboratory'? Surely better to allow a return to clinical apprenticeships where students do things, not just observe. Stress is part of medical life, and if trainees find keeping a logbook a bit much, then they should remember that the end-product of their training is to treat fellow humans safely and competently.

As final year students at St George's in the 1970s, we had a month each as 'shadow' house officers in both medicine and surgery. It meant what it said, and we did everything except writing up drugs. On qualifying we were able to start our house-jobs knowing we could cope—and so did our consultants.

In the district general hospitals the students we see are just as keen as we were, and many are all too aware of the practical deficiencies of their training: that's why they come to us; that's why they head for the 'third world' for their electives, so they can learn to be 'useful'.

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Extradural haematoma with sinusitis

Mr Papadopoulos and colleagues (November 2001 *JRSM*[1]) report an extradural haematoma (EDH) 'caused by spread of inflammation beyond the confines of the sinus'. However, there are strong grounds for proposing dural pneumodissection. The key to their case is surely the sudden severe headache. Sinusitis does not explain exacerbation of chronic headache. Also, even if sinusitis causes EDH, it is unclear what initiates the actual bleed, especially with healthy meningeal arteries[2]. This is solved by postulating forceful extradural air entry via a fistula after coughing or sneezing by vigorous males. An 11-year-old boy[3] repeatedly denied head trauma but was not asked about barotrauma. X-rays showed intracranial air and EDH, so dural pneumodissection with slow bleeding from small vessels was proposed. A 30-year-old man[4] had an EDH which decompressed via the ear; air was found inside the EDH. A violent nose-blow can cause or reopen a cerebrospinal fluid (CSF) mastoid fistula, and air can be forced into cerebral ventricles[5]. CSF fistulas are very common, and CSF pooling in sinuses causes sinus overload and infection[6,7]. Ataya's 31-year-old asthmatic[8] presented with sudden exacerbation of headache. No cause for this was given, but the patient was not asked about violent wheezing. An EDH in a 16-year-old girl[9] was associated with ipsilateral maxillary, not frontal, sinusitis, so EDH is not 'always adjacent to the infected region'[1]. Even when it was[3], the bone fistula was in the ethmoid sinus.

The EDH reported by Papadopoulos *et al.* was almost circular, suggesting a strong point source of inward pressure. It may contain two residual air bubbles, common in boys with traumatic fistulas[10]. Traumatic EDHs are commonest near ears and nose where air can be forced in.

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The case report by Papadopoulos *et al.*[1] is a description of complicated frontal sinusitis. This occurs when the infection extends beyond the confines of the bony walls of the sinus involved, and is always the result of an obstructed sinus. It is incorrect to believe that 'prompt drainage of the [resultant] haematoma and a course of antibiotics should lead to complete recovery', unless a drainage pathway to the nose is established, in this case through the frontal

recess and anterior ethmoid sinus. Currently a transnasal endoscopic approach is used for this, but an external fronto-ethmoid approach is also safe. Without such a drainage pathway, further complications are very likely to arise from sinusitis sometime in the patient's lifetime.

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Inappropriate admissions

Despite the limitations of the study due to losses in follow-up, Dr Campbell (December 2001 *JRSM*[1]) reaches an important conclusion: a proportion of patients are inappropriately admitted to hospital. In a similar study[2] we found that 13.4% of admissions to our internal medicine department were inappropriate; and again the main reason (66% in our study *vs* 64% in Dr Campbell's) was the potential for outpatient tests or treatments. Therefore we designed an interventional plan to decrease the inappropriateness. The first step was to create a 'diagnostic orientation consultancy'. In this unit, two specialists in internal medicine have online and telephonic connection with all general practitioners and family physicians in our area, and median delay between this consultation and attendance of the patient is 36 h. The second initiative has been the opening of a 'quick internal medicine consultancy' whereby hospital doctors advise on minor and mild disorders in less than 48 h after referral from any department of the hospital (most are from the emergency department). In 2001, inappropriate admissions had decreased to 9.1%. This may mean that the Appropriateness Evaluation Protocol is not only a tool for measuring the overuse of hospital resources but also an instrument for designing new strategies to avoid such overuse.

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Missing vas

The report by Mr Khan and Mr Novell (November 2001 *JRSM*[1]) raises the important issue of proper clinical examination, no matter how minor surgery is going to be. I had a similar case which was placed on my waiting list without proper examination of the scrotum. One could imagine the distress to both the patient and the surgeon when the procedure is performed under local anaesthesia. As they say, the scrotum should be examined prior to vasectomy. If there is any doubt about the presence of the vas deferens, ultrasound of the scrotum and the kidney should be done.

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1 Khan ZAJ, Novell JR. A missing vas. *J R Soc Med* 2001;**94**:582–3

In the case described by Khan and Novell[1]—a man with a missing vas in whom the ipsilateral kidney was absent but the ipsilateral testicle was present—the primary cause is presumed to be failure of development of the mesonephric (Wolffian) duct. Many years ago I saw failure of development of one of the other pair of genital ducts, the paramesonephric (Müllerian), in a woman[2]. In this instance, the left Fallopian tube was half an inch (1 cm) long and ended blindly. There was no left broad ligament, left ovary or left kidney and no left ureter was seen. The left round ligament appeared normal.

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11 September and after

Future historians will be grateful for Richard Jones' thoughtful report of the attack on the World Trade Center (December 2001 *JRSM*[1]). I too witnessed some of those horrors, on my first visit back to New York since my training concluded there in 1998. Shortly after the towers' collapse, visiting physicians offered to help in the confusion that followed. Of course, the heavy casualties did not come, and sadly there was never a need to press volunteer doctors into service. When my conference was over and I was at last allowed to leave on 13 September (New York airports were closed until that date), the return to my infertility practice in Atlanta revealed how far the ripples from those explosions would reach.

At our office, bookings for new patient appointments for the remainder of September dropped to $<30\%$ of the expected volume. Indeed, in the 10 days immediately after the attacks we registered no new patient appointments at all. Perhaps couples used this time to think about their own present circumstance, rather than to plan for a future baby. Alternatively, for safety reasons they may have decided against flying into the world's busiest airport for the required consult in Atlanta. Within 3–4 weeks of 11 September, this low volume had rebounded, and the year-end numbers actually seem to be higher than last year. But why did this happen?

When we attempted to study this patient scheduling behaviour by questionnaire, results were at variance with what we observed directly in clinic. Our centre's internet site, [IVF.com], continued to register heavy traffic throughout the period corresponding to the terrorist attacks. A poll conducted on that site showed 68% of people 'intended to undergo fertility treatment as if 11 September never happened'. It must be admitted, however, that intent and action are not always parallel; clicking a computer button is far easier than spending US $15 000 for an *in vitro* fertilization cycle. In any case, the putative impact of recent terrorist activity on consumer attitudes to both urgent and elective medical procedures represents a worthy subject for future study.

As one who found myself in Manhattan that historic morning, my helplessness was probably similar to that of witnesses to the *Titanic*'s iceberg collision or the bombing of Pearl Harbor. For Mr Jones and his example of those who were able to help the victims or their loved-ones in New York, Dante's words seem appropriate: *E ciò non fa d'onor poco argomento* (Honour not small shall be thy recompense).

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More on BSE/vCJD

The expectations of promising treatments described in this update by Dr Beale (December 2001 *JRSM*[1]) are offset by his conclusion that 'In the long term, unfortunately, there will probably be no shortage of patients in whom to try these treatments'. In saying this, Beale is accepting, without discussion, the conclusion reached by the BSE Inquiry[2] that 'BSE has caused a harrowing, fatal disease in humans' because they too accept evidence from histopathology, chromatography and experiments in mice that a malfolded prion protein found in the brains of cattle with BSE as proof that it is transmissible to humans in meat and causes variant Creutzfeldt–Jakob disease (vCJD).

This evidence depends entirely upon evidence from laboratories. In the farmyard, dogs, cats and other animals are freely exposed to BSE but, except for rare cases in domestic cats, there is no evidence of comparable encephalopathy. Evidence from national and international surveillance shows no increase in vCJD except in proportion to the increased ascertainment[3,4] which followed identification of cases of CJD in people in younger instead of ageing patients. But this is precisely what Creutzfeldt observed in his first case in 1920, a woman aged 23, which he described in clinical and pathological detail as a unique encephalopathy[5]. Such cases were uncommon because they were identified wrongly (*Unter flasche Flagge*) as multiple sclerosis and other neurological disorders which were symptomatically similar but did not show the neuronal degeneration characteristic of the spectrum of encephalopathies subsequently classified as CJD because, although exceedingly rare, they were observed more often in older patients, as described later by Jakob. Similar encephalopathies, occurring notably in sheep as scrapie, in captive and occasionally in various wild mammals are transmissible (TSEs) within and sometime across species barriers by intracerebral trans-inoculations of brain substance. BSE, the latest example, is transmissible by intracerebral or peritoneal inoculation in mice. If the mice are inbred and transgenic, the lesions resulting from intracerebral injection are similar to those of BSE and vCJD.

The surveillance which extended during the two years and nine months of the BSE Inquiry showed that all the patients with vCJD were homozygous for methionine, as are approximately 40% of the resident population of the UK. Although this could indicate a genetic predisposition to form or to accept malfolded PrP, it does not explain why vCJD is so rare in that part of the same population at highest risk of exposure to BSE, namely workers in farms, abattoirs, butchers shops, kitchens and veterinarians.

Among other points escaping attention in Beale's update, as in the Inquiry, is the entire absence of BSE in suckler-then-grass-fed pedigree herds of Herefords, Angus and other breeds except where there is contact with dairy cattle[3]. This suggests that BSE could be a metabolic PrP disease induced by unnatural forced feeding of animal protein to herbivores, with concomitant stress, or by conveyance of mutable strains of scrapie, or because artificial insemination from limited pools has created a genetic predisposition in dairy cattle to accommodate the novel mutation from scrapie in 1970 which the Inquiry favours presumptively as the origin of BSE. To say the least, these and other uncertainties cast doubt on Beale's endorsement of the scientific prognosis that, among all humans who have ever eaten beef from herds with BSE in or from the UK, there will be (within confidence limits of zero to infinity) no shortage of patients who develop a questionably new, ultimately fatal encephalopathy.

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