common than the council expects. Nobody has ever attempted an examination of the performance of a random sample of doctors in Britain, but such surveys have been done in Canada and found serious deficiencies in between 8% and 15% of almost 900 family doctors and 2% of 380 specialists (R G McAuley, congress on continuing medical education, Los Angeles, 1988).⁹ Then a study of 31 000 random admissions to hospital in New York in 1984 showed that 4% led to adverse events, and in a quarter of those cases the doctors had been negligent.¹⁰ In Britain the first confidential enquiry into perioperative deaths found that 7% of the deaths were solely attributable to surgery and in many more surgical and anaesthetic problems partially accounted for the deaths.¹¹ These pieces of information suggest that poor performance may not be rare and that the GMC machinery may need to have extra capacity built in "just in case."

The second important problem is the relation between the new machinery and audit. It is probably no accident that government pressure for audit and public pressure for more accountability from the GMC have come together, but the timing is unfortunate. Most doctors agree that if audit is seen as an antechamber to the GMC's performance review machinery then audit will not flourish. But inevitably local attempts to manage those who will not participate in audit or who are unwilling or unable to improve poor performance exposed by audit may eventually become entangled with the GMC system. This is something that doctors don't like to contemplate, but they will have to.

The third problem lies in the nature of poor performance. The GMC's proposal talks in terms of retraining those doctors who perform poorly, but managers from any walk of life know that poor performance is rarely managed by a short spell of retraining. Poor performance often has its roots in a combination of psychology and circumstance that is not so easily reversed. Thus some of those entering the GMC machinery may need prolonged retraining that may not succeed, and they may face loss of livelihood. The difficulty and expense of getting poor performers back on track may mean that cash strapped health authorities will be unwilling to foot the bill. They may choose the cheaper option of

dismissal, leaving the council to pick up the bill or deregister the doctor.

And the fourth problem is expense. Doctors are willing to pay for the privilege of self regulation, but they will not be willing to sign a blank cheque. The finances of the GMC are already shaky because of the rapidly rising cost of cases of possible misconduct, and the annual retention fee, which was introduced only in 1970, has more than doubled in the past two years. Now the cost of the new machinery must be added, which is difficult to cost. The president of the GMC told the BMA's council that he hoped that the system would add only about £5-10 a year to the annual retention fee (currently £80), but this estimate is based on the assumption that the NHS will pick up the costs of retraining. This is doubtful in all cases, and there is still the problem of doctors working as long term locums and those working in private practice. Predicting how many doctors will come through the system is also extremely difficult. The costs may be much higher than predicted.

Most doctors, even if they know little of the GMC, believe in self regulation, and we must hope that the GMC can produce a system that will help doctors who fall below acceptable standards without creating a climate of fear that will interfere with raising quality throughout the NHS. This must also be achieved speedily at an affordable price. None of it will be easy.

RICHARD SMITH

Editor, *BMJ*

- 1 Varlaam CF. *The origin and development of the GMC as a sociolegal institution*. London: University of London, 1978. (PhD dissertation.)
- 2 Pyke-Lees W. Centenary of the General Medical Council 1858-1958. In: *The history and present work of the council*. London: General Medical Council, 1958.
- 3 Smith R. Profile of the GMC: 1978 and all that. *BMJ* 1989;298:1297-300.
- 4 Smith R. Profile of the GMC: the hordes at the gates. *BMJ* 1989;298:1502-5.
- 5 Spearing N. Medical Act 983 (amendment). *House of Commons Official Report (Hansard)* 1987 March 3;112:cols 157-8.
- 6 Smith R. Raw emotion from "Raw deal." *BMJ* 1990;301:558-9.
- 7 Berwick DM, Enthoven A, Bunker JP. Quality management in the NHS: the doctor's role. I. *BMJ* 1992;304:235-9.
- 8 Berwick DM, Enthoven A, Bunker JP. Quality management in the NHS: the doctor's role. II. *BMJ* 1992;304:304-8.
- 9 McAuley RG, Henderson HW. Results of peer assessment program of the College of Physicians and Surgeons of Ontario. *Can Med Assoc J* 1984;131:557-61.
- 10 Harvard Medical Practice Study. *Patients, doctors, and lawyers: medical injury, malpractice litigation, and patient compensation in New York*. Boston: Harvard Medical Practice Study, 1990.
- 11 Buck N, Devlin HB, Lunn JN. *Report of the confidential enquiry into perioperative deaths*. London: Nuffield Provincial Hospitals Trust and King's Fund, 1987.

Doctors, patients, and HIV

The risk of transmission from a single inoculation injury with HIV positive blood is 1:275

Recent reports of an HIV infected surgeon working in a British hospital have stirred up anxieties about the transmission of HIV from patients to doctors and from doctors to patients. This week's report from the Royal College of Pathologists should go some way to allay them. *HIV Infection: Hazards of Transmission to Patients and Health Care Workers during Invasive Procedures* provides up to date information on transmission of HIV from patients to staff.¹

During a surgical operation or resuscitation of a critically ill patient the thoughts of the operator are usually focused on the task in hand. Unless there has been a reason to suspect infection in the patient the possibility of exposure to HIV and other bloodborne viruses may not be prominent in the minds of the surgeons, dentists, anaesthetists, or other staff participating in the procedure. Clearly the best approach to preventing occupational infection is to regard blood from any patient as potentially infectious and to adopt "universal precautions" with all patients so that the risk of inoculation injury or mucocutaneous exposure is removed or at least

reduced to a minimum. Despite the concern generated by the spread of HIV infection these incidents occur frequently and are a continuing source of anxiety.

After such incidents health care workers should seek confidential advice and, if necessary, medical follow up and serological testing. Responsibility for these services varies and usually rests with the occupational health service, clinical microbiologists, or virologists. Medical advisers need access to the latest data on occupational transmission and to be able to discuss difficult decisions such as the prophylactic use of zidovudine. The royal college's recent report should help them. Its statistics, which will be updated as necessary, are accompanied by recommendations for reducing risk and an extensive list of references.

Before the recent British case the potential hazards posed to patients from surgeons and dentists infected with HIV-1 came under scrutiny after reports suggested that five patients had been infected through invasive dental procedures performed

by a dentist with AIDS.^{2,4} The Royal College of Pathologists' report provides details of serological testing of patients who had been operated on by HIV infected surgeons, together with estimates of the theoretical risks of transmission. There have been no known instances of transmission of HIV infection from surgeons to their patients. Health care workers who are infected with HIV should not perform invasive procedures if there is a possibility of their blood being transferred to the patient; readers are referred to recent British⁵ and north American guidelines.⁶

A skilled adviser who can balance the risks from inoculation injury against other risks in occupation and in life in general can be very helpful to a member of staff or a patient exposed to HIV infection. The latest follow up studies continue to confirm the rate of transmission from patients to staff members from a single inoculation injury with HIV positive blood as 0.36% or 1 in 275 (upper limit of 95% confidence interval=0.69%). The risk from mucocutaneous exposures is less than this; no seroconversions have been reported from 921 exposures in prospective studies. Reassuringly, extensive studies have failed to confirm evidence of transmission from patients to staff during general ward care and invasive procedures, including dentistry and orthopaedic surgery, even in areas with a high prevalence of HIV.

The Royal College of Pathologists' report does not help in the difficult decision on whether to recommend zidovudine after exposure. As previously detailed in an editorial in this journal⁷ no data on efficacy are available and there are unlikely to be any in the foreseeable future; the low rate of seroconversion after inoculation injury means that a study of the efficacy of prophylactic zidovudine would require the recruitment of many thousands of people. The authors of the report reiterate advice from the United States stating that, after careful consideration of all data then available, zidovudine "could not

be considered a necessary component of post exposure management."⁸ They then record five instances of apparent failure of prophylaxis with zidovudine, in only one of which the time to administration was less than two hours; they also record details of short term toxicity.

The report advises health districts to consider the arguments for and against the use of zidovudine and develop a local policy on its use. It is all too easy to decide, in the absence of data on efficacy, that zidovudine after exposure is not justified, and this decision is much easier to make where the perceived prevalence of HIV is low. If zidovudine prevented a small proportion of cases of nosocomial transmission, perhaps 10% or less, there would be no question about its use. We have, however, no way of obtaining such data. Currently it seems that most British and north American hospitals are providing access to zidovudine in cases of significant percutaneous exposure to HIV infected blood. Evidence suggests that the rate of acceptance is low, but making the drug unavailable would surely be wrong.

D J JEFFRIES

Professor of Virology,
St Bartholomew's Hospital,
London EC1A 7BE

- 1 Royal College of Pathologists. *HIV infection: hazards of transmission to patients and health care workers during invasive procedures*. London: RCPATH, 1992.
- 2 Centers for Disease Control. Possible transmission of HIV to a patient during an invasive dental procedure. *MMWR* 1990;39:489-93.
- 3 Centers for Disease Control. Possible transmission of HIV to a patient during invasive dental procedures—Florida. *MMWR*. 1991;40:21-7.
- 4 Centers for Disease Control. Update: transmission of HIV infection during invasive dental procedures—Florida. *MMWR* 1991;40:378-81.
- 5 UK Health Departments. *AIDS-HIV infected health care workers. Recommendations of the expert advisory group on AIDS*. London: Department of Health, 1991.
- 6 Centers for Disease Control. Recommendations for preventing transmission of human immunodeficiency virus and hepatitis B virus to patients during exposure-prone invasive procedures. *MMWR* 1991;40:1-9.
- 7 Jeffries DJ. Zidovudine after occupational exposure to HIV. *BMJ* 1991;302:1349-51.
- 8 Centers for Disease Control. Public health service statement on management of occupational exposure to human immunodeficiency virus, including considerations regarding zidovudine post exposure. *MMWR* 1990;39:RR-1.

Multiple sclerosis: diagnostic optimism

How magnetic resonance imaging has advanced understanding and research

Great strides have been made in our understanding of multiple sclerosis in the past decade. This new knowledge comes from experimental studies into the mechanism of immune mediated damage in the central nervous system and from magnetic resonance imaging.

The first step was the recognition that abnormalities in the brain in multiple sclerosis are easily identified by magnetic resonance imaging and that the lesions shown by the technique correspond with the plaques within the nervous system.^{1,3} Soon it became clear that there was often a remarkable waxing and waning in size of the areas of abnormality over a matter of weeks.⁴ Moreover, when the enhancing agent gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA) was combined with magnetic resonance imaging some lesions became enhanced while others did not.^{5,6}

These observations became understandable after frequent serial scanning in patients and a comparison of the results with the findings in allergic encephalomyelitis in animals and necropsy studies. Enhancement has been shown to correspond with regions of focal increase in permeability of the blood-brain barrier in association with inflammation.^{7,8} Quantitative magnetic resonance methods have been used to study the nature of the "disappearing" element in the lesion and the structure of the residual scar.

The sequence of events is as follows (reviewed in reference

9). The earliest detectable abnormality is an increase in permeability of the blood-brain barrier. This change may precede the development of corresponding symptoms, though evoked potential recordings done at this stage do show impairment of conduction, probably contributed to by demyelination. Oedema follows and may be extensive. The change in the blood-brain barrier gradually reverses and is usually over after a month. The oedema then resolves to leave a smaller residual scar. The process may be repeated in a ring-like form at the edge of large lesions, which then grow centrifugally.

One of the outstanding characteristics of multiple sclerosis is the near complete clinical recovery that occurs after most early attacks despite persisting evidence of demyelination as shown by evoked potentials. Ultimately many patients develop persistent neurological deficits. The mechanism of these events has long been a puzzle. Magnetic resonance imaging is now helping to solve it.

Serial studies of patients with optic neuritis have shown that the acute symptoms and signs (including pain and visual loss) occur during the inflammatory phase and resolve as the permeability in the blood-brain barrier returns to normal. This implies that inflammation as such plays a part in the pathophysiology of the lesion.¹⁰ These effects may possibly be mediated by cytokines, some of which have striking effects on excitable membranes.¹¹ A reduction in the production of