doctors still complain of the lack of support and supervision by their seniors.⁴ Many of the complaints of long hours would disappear if juniors' expectations of training were met.

It is desirable that our young doctors should not be chronically tired and stressed, but piecemeal patient care and training would be just as deleterious. On call rotas should not exceed every third or fourth night, and no one should be expected to be on call for more than 24 hours at a time, including weekends. More efficient use of cross cover, especially at senior house officer level, would reduce on call rotas, and curtailing the non-clinical duties would lessen the workload.⁵ The government must pay for the projected expansion of the consultant grade⁶ to allow for "more direct" consultant care and for training. Surely it must be time to review the expansion of the non-training subconsultant grades despite the opposition of the juniors: staff doctors and associate specialists should not be denigrated, they have much to contribute towards the service and they would free junior doctors for training. It is a pity that consultant status is perceived as the only form of success, and juniors ought to be reminded that not everyone who wishes to remain in hospital medicine wants or is able to become a consultant.

The BMA survey of junior doctors⁷ shows that, similar to the case in the United States,⁸ the most militant doctors are from the primary care specialists. Is it because they dislike hospital medicine and therefore find the hours more stressful or cannot cope so well?

Are vocation and professionalism considered old fashioned? Are we selecting the wrong students, or have we failed to prepare them for a challenging and demanding career ahead? I strongly support a review of junior doctors' hours, but it behoves the profession to give equal consideration to serious issues such as training, continuing education, and professionalism.

K H MATHESON

West Suffolk Hospital,
Bury St Edmunds IP33 2QZ

- 1 Hunter J. Juniors' hours: measuring the strength of feeling. *BMJ* 1990;301:1008. (3 November.)
- 2 Various authors. Juniors' hours: international overview. *BMJ* 1990;301:830-2. (13 October.)
- 3 Hoffmann J, Fischer A. Juniors' hours. *BMJ* 1990;301:1159. (17 November.)
- 4 Dudley HAF. Stress in junior doctors. I. Stress and support. *BMJ* 1990;301:75-6. (14 July.)
- 5 Turnbull NB, Miles NA, Gallen IW. Junior doctors' on call activities: differences in workload and work patterns among grades. *BMJ* 1990;301:1191-2. (24 November.)
- 6 United Kingdom Health Departments, Joint Consultants Committee, Chairmen of Regional Health Authorities. Hospital medical staffing. *Achieving a balance: plan for action*. London: HMSO, 1987.
- 7 Delamothe T. Juniors favour action over hours. *BMJ* 1990;301:1235. (1 December.)
- 8 Matheson KH. Stress and stress counselling. *Postgrad Med J* 1990;66:738-42.

SIR,—Dr Tony Delamothe reported the results of the recent survey of attitudes of hospital juniors towards hours of work.¹ Interestingly, it was general practitioner trainees and doctors in accident and emergency medicine, paediatrics, and psychiatry who were the most militant. Paediatric posts, particularly ones that include cover of neonatal units, are recognised to be among the busiest of junior hospital jobs, but I do not believe that to be true of psychiatric posts nor of many in accident and emergency departments, in which shift systems already operate. If front line specialties such as obstetrics, general surgery, orthopaedics and trauma, and anaesthetics showed less militancy I suspect that some juniors in these specialties actually think that long hours may be necessary for training.

I do not believe that the excessive hours worked by house officers can be in any way educational, especially if much of their work is repetitive and clerical. But this does not necessarily apply to other

training grades. Much of the emergency work in my own specialty is routine: common conditions being common. By insisting that we reduce our on call commitment we also restrict our chances of being exposed to complex and difficult cases. Even in a specialist children's hospital, for example, there may be a difficult airway problem only two or three times a month, with conditions such as tracheo-oesophageal fistula or diaphragmatic hernia occurring less often. If you are not there you cannot see them.

Before abuse is heaped on me let me say that 72 hours a week is surely enough to gain adequate clinical exposure: longer hours are bound to be counterproductive for all the well recognised reasons. But I am equally convinced that "industrial action" is no way to remedy the current problem. Medicine is not an industry, nor is it yet a business, and there is in any case the example before us of the ambulance service, which achieved nothing despite widespread public support for its action.

But there are other reasons for eschewing action. It has recently been reported that in Watford all non-emergency work is to be suspended from 1 January 1991 to ensure that the health authority does not exceed its current budget pending the introduction of the health service changes in April. This is a disgraceful position forced by a government that is none the less prepared to write off large sums of money to underpin water privatisation. Although the underlying issues are quite different, I believe that it is impossible to express our condemnation of Watford's position if almost half of the junior hospital doctors who replied to the Hospital Junior Staff Committee survey would be prepared to create exactly the same conditions as will apply in Watford by refusing to undertake all but emergency work.

SIMON BRICKER

Countess of Chester Hospital,
Chester CH1 3ST

- 1 Delamothe T. Juniors favour action over hours. *BMJ* 1990;301:1235. (1 December.)

HIV and surgeons

SIR,—We were heartened by the call from Mr David Hamblen and Mr Geoffrey Newton, on behalf of the British Orthopaedic Association, for the implementation of universal precautions throughout the United Kingdom.¹ But our spirits sank when we discovered that they also support the recommendation of the Royal College of Surgeons of Edinburgh for the testing of patients suspected of being infected with HIV.²

Universal precautions means that the blood and certain body fluids of all patients are considered potentially infectious for HIV hepatitis B virus, and other blood borne pathogens. Testing the serum of patients who are suspected of being infected with HIV for the exclusive purpose of applying additional protective measures is not consistent with the concept of universal precautions.

Do Messrs Hamblen and Newton, and indeed the Royal College of Surgeons of Edinburgh, really believe that all, or even most, homosexuals conform to the stereotype of the "raving queen" and are therefore immediately identifiable? Do they appreciate that in 1989 heterosexual contact was the only exposure category in 7.9% of all AIDS cases in the United Kingdom, that this percentage increased to 10.6% for the first half of 1990, and that it is expected to rise even higher with time?³ Did they know that 28% of 52 956 people who had a positive test result at publicly funded sites in the United States between January 1988 and September 1989 were heterosexuals other than intravenous drug users, haemophiliacs, and

recipients of infected blood?⁴ And finally, are they aware of a recent survey in sentinel hospitals in the United States that showed that up to 7.8% of patients (and up to 21.7% of men aged 25-44), whose clinical presentation would not have led health care workers to suspect that they might have an increased likelihood of being positive for HIV antibodies, were, in fact, infected with this virus?⁵

We agree with Dr B G Gazzard and M C Wastell that in those parts of the country where the number of patients infected with HIV is small, the financial implications and added inconvenience for surgeons and other health care workers may preclude the implementation of a pure universal precautions policy at the present time.⁶ A compromise, in the form of a two tier system whereby those patients who are known to be infected with HIV are targeted for additional measures is likely and not unreasonable. We believe, however that testing for HIV based solely on suspicion is untenable.

E M BROWN
S CROSSMAN

Frenchay Hospital,
Bristol BS16 1LE

- 1 Hamblen D, Newton G. HIV and surgeons. *BMJ* 1990;301:1216-7. (24 November.)
- 2 Royal College of Surgeons of Edinburgh. *Statement to fellows on HIV infection and AIDS*. Edinburgh: Royal College of Surgeons of Edinburgh, 1989.
- 3 Communicable Disease Surveillance Centre. *Increase in heterosexual contact AIDS cases*. London: CDR, 1990: (90/28.)
- 4 Center for Disease Control. Publicly funded HIV counselling and testing—United States, 1985-1989. *MMWR* 1990;39:137-40.
- 5 St Louis ME, Rauch KJ, Petersen LR, et al. Seroprevalence rates of human immunodeficiency virus infection at sentinel hospitals in the United States. *N Engl J Med* 1990;323:213-8.
- 6 Gazzard BG, Wastell C. HIV and surgeons. *BMJ* 1990;301:1003-4. (3 November.)

HIV infection in the Edinburgh haemophiliac cohort

SIR,—Dr R J G Cuthbert and colleagues examined various factors associated with progression of HIV infection in haemophiliac patients in Edinburgh.¹ Since February 1986 we have also prospectively studied possible prognostic indicators in a group of 40 haemophiliac patients positive for antibodies to HIV who were attending St James's University Hospital.² Unlike in the Edinburgh cohort no firm date of seroconversion was identifiable, and our follow up was from the time of their first positive test result. We also found the serum concentration of β_2 microglobulin to be a powerful predictor of progression to AIDS in our patients.³

In addition, we found that, based on the lowest recorded count before the development of symptoms, CD4+ cell counts did not distinguish those patients who subsequently went on to develop Centers for Disease Control group IV disease from those who did not. Cuthbert and coworkers also found that single estimations of numbers of circulating CD4+ cells were of limited value and that "a decline to $0.2 \times 10^9/l$ did not necessarily precede symptoms." They state that a downward trend with time is more meaningful and that the mean CD4+ cell counts fell more rapidly in patients positive for HIV antibody with symptoms than in those without symptoms. They do not, however, seem to have applied statistical analysis to support this statement. We make this point as there is some evidence that haemophiliac patients without HIV antibodies have impaired cell mediated immunity as evidenced by impaired delayed cutaneous hypersensitivity,⁴ and decreased CD4+ cell counts have been noted in haemophiliac patients before seroconversion.⁵ This effect may be a direct manifestation of prolonged treatment with factor VIII or exposure to other viruses such as cytomegalovirus or Epstein-Barr virus. Hence a further decrease in CD4+ cell counts in patients positive for HIV antibodies at particular

risk of developing AIDS may not be so clear cut in haemophilic patients. If this was the case identifying other factors that are prognostic assumes greater importance.

We agree with Dr Cuthbert and colleagues that serum IgA titres tend to rise with disease activity in patients who have already progressed to AIDS. We found, however, that high titres were recorded before symptoms developed and that a serum IgA titre of greater than 4.5 g/l (the upper limit of normal for our laboratory) was an independent predictor for the development of AIDS in 14 out of 17 of our patients in whom raised titres were recorded ($\chi^2=9.19$; $p<0.01$). The three year progression rate in seven of these patients who already had raised IgA titres at recruitment was 100% compared with 32% in patients with normal titres at recruitment.

Lastly, we and other workers⁶ have found the presence of p24 antigen to be an important prognostic indicator. We note that p24 antigen was measured in the Edinburgh cohort, though no reference was made to its usefulness or otherwise in predicting disease progression.

G M SMITH
B A McVERRY

St James's University Hospital,
Leeds LS9 7TF

E H COOPER

University of Leeds School of Medicine,
Leeds LS2 9JT

- 1 Cuthbert RJG, Ludlam CA, Tucker J, *et al*. Five year prospective study of HIV infection in the Edinburgh haemophilic cohort. *BMJ* 1990;301:956-61. (27 October.)
- 2 Smith GM, Forbes MA, Cooper J, *et al*. Prognostic indicators for the development of AIDS in HIV antibody positive haemophilic patients: results of a three year longitudinal study. *Clin Lab Haematol* (in press).
- 3 Smith GM, Cooper EH, Hall L, McVerry BA. Serum β_2 microglobulin levels in haemophilics. *Clin Lab Haematol* 1989;11:409-10.
- 4 Brettler DB, Forsberg AD, Brewster F, Sullivan JL, Levine PH. Delayed cutaneous hypersensitivity reactions in haemophilic subjects treated with factor concentrate. *Am J Med* 1986;81:607-11.
- 5 Hollán SR, Füst G, Nagy K, *et al*. Immunological alterations in anti-HTLVIII negative haemophilics and homosexual men in Hungary. *Immunol Lett* 1985;11:305-10.
- 6 Goedert JJ, Kessler CM, Aledort LM, *et al*. A prospective study of human immunodeficiency virus type I infection and the development of AIDS in subjects with hemophilia. *N Engl J Med* 1989;321:1141-8.

MRC freezes new research

SIR,—Dr Richard Smith reports that the Medical Research Council has frozen new research to balance its books.¹ If the council is broke surely what it should economise on is the human genome project rather than original research into the nature and cure of diseases.

Even if most medical conditions are genetically determined, and even if it proved to be wise as well as feasible to tinker with our genetic pool by, for instance, introducing missing genes into the phenotype and so mitigating the effects of natural selection, there is much more scope for modifying environmental effects. For example, the effects of early deprivation on heart disease are likely to prove more important and much easier to modify than genetic defects in the cholesterol receptor.

I get the impression that the direction of the MRC has fallen into the hands of so called pure scientists on the specious argument that in the recent past their kind of science has contributed more to medicine than the plodding work of clinicians. Academic clinicians tend to go along with this out of intellectual deference, with the result that even the systems and neurosciences boards are dominated by laboratory scientists. Why should it be that highly rated environmentally biased units like the Dunn Nutritional Laboratories should have to cut back to pay for yet more molecular biology.

When I was a student I was taught, quite

correctly, that tuberculosis was a genetically determined disease, exposure being nearly universal, but that didn't stop the introduction of isomazid making it a rare disease in our society. I suggest that if the MRC is going to provide medical value for money, it should be headed by an epidemiologist and that it should be reconstituted into boards concerned not with subjects such as physiological systems, neurosciences, and cell biology but with, for instance, infective disease, including microbiology and immunology; degenerative disease; developmental medicine, including obstetrics, paediatrics, and cytogenetics; and neoplastic disease. Each board should be comprised of appropriate clinicians, basic scientists, epidemiologists, and veterinarians. In this way we would get value for money and could put money where it would best pay off in relation to health.

JOHN A DAVIS

Great Shelford,
Cambridge CB2 5JE

- 1 Smith R. MRC freezes new research. *BMJ* 1990;301:1116. (10 November.)

Investigation of adults with haematuria

SIR,—We disagree that Dr John Spencer and colleagues have shown that ultrasonography is better than intravenous urography in investigating patients with haematuria.¹ The apparent advantage was largely limited to detecting bladder tumours, but this is of little relevance as all patients with haematuria should be given cystoscopy. The authors imply that this was not carried out in all their patients and thus cannot give the important false negative rate for ultrasonography.

They state that "the value of ultrasonography in diagnosing bladder tumours is established." But the work they referred to was a small study (not blind) of 17 patients with bladder tumours, which concluded that "further experience is necessary before conclusions can be drawn."² Since then studies of much larger numbers of patients have shown that only 50%-80% of bladder tumours are detected with transabdominal ultrasonography.^{3,6} Furthermore, the detection of tumours less than 0.5 cm is particularly poor at 33%-38%.^{3,7} Dr Spencer and colleagues have not reported detection of any bladder tumours under 0.5 cm; in another study of 79 patients with bladder tumours all 20 false negative results from ultrasonography were for tumours under 0.5 cm.⁵

Most patients in the study by Dr Spencer and colleagues were referred by general practitioners, presumably to exclude bladder malignancy. Ultrasonography clearly is not good enough for this, and cystoscopy is required. The flexible cystoscope allows this to be performed expediently and cost effectively as an outpatient procedure with minimal patient discomfort under local anaesthesia. Cystoscopy has been combined with intravenous urography in our hospital to provide a same day screening service; 6.8% of 102 patients presenting with microscopic haematuria (symptomatic and asymptomatic) were found to have malignancy (T Lynch, personal communication).

This leaves the question of what is the best radiological investigation for assessing the upper urinary tracts and to complement cystoscopic assessment of the bladder. The study by Dr Spencer and colleagues suggests that intravenous urography is better than ultrasonography, and they rightly conclude that a urogram must be obtained in patients with haematuria in whom cystoscopy has shown nothing abnormal. Presumably, therefore, the 81 patients who had normal results would have required additional intravenous urography anyway, making the argument of cost effectiveness of ultrasonography less convincing.

We are concerned that this study may be interpreted as showing ultrasonography as an acceptable alternative to the standard practice of referral for intravenous ultrasonography and cystoscopy of any patient with haematuria. Managing such patients without contemplating cystoscopy is to be strongly discouraged if patients are to receive the benefits of early diagnosis of bladder malignancy.

P S STONELAKE
D M A WALLACE

Queen Elizabeth Hospital,
Birmingham B15 2TH

- 1 Spencer J, Lindsell D, Mastorakou I. Ultrasonography compared with intravenous urography in the investigation of adults with haematuria. *BMJ* 1990;301:1074-6. (10 November.)
- 2 Abu-Yousef MM, Narayana AS, Franken EA, Brown RC. Urinary bladder tumors studied by cystoscopy. *Radiology* 1984;153:223-6.
- 3 Malone PR, Weston-Underwood J, Aron PM, *et al*. The use of transabdominal ultrasound in the detection of early bladder tumours. *Br J Urol* 1986;58:520-2.
- 4 Vallancien G, Veillon B, Charlton M, *et al*. Can transabdominal ultrasonography of the bladder replace cystoscopy in the follow up of superficial bladder tumors? *J Urol* 1986;136:32-4.
- 5 Juul N, Torp-Pederson S, Larsen S, *et al*. Bladder tumor control by ultrasound and urine cytology. *Scand J Urol Nephrol* 1986;20:275-8.
- 6 Davies AH, Cranston D, Meagher T, Fellows GJ. Detection of recurrent bladder tumours by transrectal and abdominal ultrasound compared with cystoscopy. *Br J Urol* 1989;64:409-11.
- 7 Itzhak Y, Singer D, Fischelovitch Y. Ultrasonographic detection of bladder tumors. 1. Tumor detection. *J Urol* 1981;126:31-3.

SIR,—In many ways the paper by Dr John Spencer and colleagues¹ reproduces the flawed arguments of their paper on renal imaging in adult urinary tract infection.¹² Most alarmingly, it fails to present clearly the strengths and weaknesses of intravenous urography versus ultrasonography.

The authors fail to point out that their claim to identify ureteric calculi on plain films cannot be substantiated without intravenous urography to localise the calcification. They misquote Middleton *et al*,³ who stated that plain films with renal tomography are a more sensitive way of detecting renal calculi than is ultrasonography, and fail to point out that radiolucent ureteric calculi cannot be identified either by ultrasonography or plain radiography. They do not mention that papillary necrosis (a cause of haematuria) cannot be diagnosed by ultrasonography, although they do accept that ultrasonography does not provide sufficient anatomical detail to diagnose transitional cell tumours in the upper tract. They also claim that medullary sponge kidney can be diagnosed by ultrasonography—a claim unlikely to be supported by informed renal radiologists.

As with their paper on renal imaging in patients with urinary tract infection the authors totally fail to understand the question posed to the clinician. Rather, this is a play on statistics: What is the likelihood of identifying some underlying cause for haematuria? All practising clinicians know that the return on any imaging procedure in this case will be small. Unlike radiologists the clinician must be able to assure the patient that, with the best available investigation, no anatomical cause for the haematuria has been found. Ultrasonography in this context is recognised to be a second class investigation. Inevitably, because an anatomically demonstrable lesion is unlikely, a small series (and 155 patients must be deemed to be a small series given a less than 5% positivity rate) is unlikely to show the relative merits of intravenous urography versus ultrasonography.

We welcome their aim to reduce exposure to ionising radiation to a minimum but would caution clinicians to examine closely the merits of adopting inferior imaging techniques as opposed to optimum techniques in their clinical practice. With the welcome arrival of critical medical audit, clinicians' choice of investigation must be shown to be in the best interest of the patient and not in the interests of cost paring by departments of