that this has been seen as a regional responsibility. Instead it should be viewed as a duty of the health district or short stay hospital to provide the service. Every district general hospital in Britain should have a computed tomography scanner in the 1990s if it is to practise effective medicine.

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1 Hewer RL, Wood VA. Availability of computed tomography of the brain in the United Kingdom. *Br Med J* 1989;**298**:1219-20. (6 May.)

Junior doctors' hours

SIR,—Dr Nigel F Perks's letter raised some interesting points.[1] I too find it difficult to understand how hours of work as well as numbers of middle grade staff can be reduced without reducing the service commitment.

The often repeated implication that shortening working hours necessarily requires a lengthened training programme, however, is, I believe, incorrect. This assertion is based on my recent experience of a four year residency training scheme at a Canadian teaching hospital. There residents had no service commitment as all cases had to be attended by staff anaesthetists and surgeons. In contrast, the hospital departments have a commitment to teaching the residents. Thus all clinical work was routinely supervised, and the residents were almost always free to take part in the regular organised academic teaching sessions. How many British junior doctors can claim similar postgraduate training opportunities?

The recently published confidential inquiry into perioperative deaths has identified cases of junior doctors taking on cases beyond their competence, often outside "office hours."[2] This does not constitute proper training, and we should not delude ourselves that working excessive hours can compensate for it. With the ever increasing cost of litigation and the complexity of modern medicine the days of the attitude "see one, do one, teach one" should be behind us. It is unlikely to be purely coincidental that the pass rate for the Canadian anaesthetic fellowship examination approaches 80% whereas that for the final examination for fellowship of the College of Anaesthetists is below 20%.

A health service "working for patients" seems to require more fully trained doctors for both improved patient care and junior staff training.

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1 Perks NF. Junior doctors' hours. *Br Med J* 1989;**298**:1314. (13 May.)
2 Buck N, Devlin HB, Lunn JN. *Confidential inquiry into perioperative deaths*. London: Nuffield Provincial Hospital Trust, 1987.

Parable of the £10 000 general practitioner

SIR,—May I correct a false impression that John Warden may have left in his account of general practitioners' evidence to parliament's social services committee?[1]

Mr Warden reported that I was the only one to oppose completely general practice budgets "given [my] position as a rural general practitioner." In fact, I opposed the budgets on two grounds. Firstly, budgets will sour the relationship between doctors and patients; doctors will cease to be the advocates of patients and will become brokers for the state. Secondly, from an analysis of present incentives in the United Kingdom and of health care markets in the United States I believe that there is no evidence that budgets would improve choice or efficiency but that evidence suggests that they would increase administration costs. I referred to my written submission to the committee, which contained a fuller critique of the practical consequences of budgets. (It also exposed how the white paper is based on untested assumptions and showed that the evidence that exists suggests that the proposals will have the opposite effect to the aims the white paper espouses.) I also pointed out that there is little opportunity for competition among general practitioners and among hospitals in many parts of the country, using my own area purely as an example.

I must clarify that my objections to budgets are not parochial but principled. Budgets are an integral part of the government's attempts to introduce a health care market. Two of the general practitioners who spoke in favour of budgets claimed that they could provide a better service to their patients. They may be right (though I believe that they have some rude surprises in store), but the point is that this would not be the case for the whole nation. This is no time for parochialism: we must fight to preserve what is best in the NHS and do so in unity.

I would like to see general practice reformed so that we stop thinking about only the patients in our own patch and start thinking about all patients. The two essential principles of such reform are accountability and planning. Neither budgets nor markets make general practitioners truly accountable because consumer sovereignty does not exist in health care. We need accountability to a body that both represents patients and can assess our work; we need a new type of family practitioner committee whose remit extends beyond the financial policing planned in the white paper and encompasses an assurance of quality.

Budgets would fix general practice in entrepreneurial units; planning would allow it to evolve. General practice has moved from treating problems to caring for the person; from there it has taken on prevention within the practice; the future lies in planning medical care for a population. For this we need coordination on a level higher than the individual practice: we need family practitioner committees that are primary care authorities.

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1 Warden J. Parable of the £10 000 general practitioner. *Br Med J* 1989;**298**:1207-8. (6 May.)

Eye, eye

SIR,—Dr Bashir Qureshi's point about eye to eye contact[1] is a genuine problem faced by doctors from overseas not only in clinical practice but also in examinations.

May I quote my own experience, which is not very different from that of other doctors from overseas? As a Pakistani girl, I was always instructed not to look straight into the eyes of other people, especially parents and teachers, while talking to them. I am thankful that a British well-wisher told me just before I took the primary examination for fellowship of the Royal College of Surgeons that a higher percentage of candidates from Pakistan and India than local candidates fail British examinations. One of the reasons is that Asian candidates when asked a question in an oral examination lower their eyes as a sign of respect despite knowing the answer, which may be interpreted as a sign of not knowing and may not benefit the candidate. I was advised to practise talking while looking into another person's eyes, which was not easy. Furthermore, I was astonished when a German doctor told me that in Germany you would be considered to be telling a lie if you did not look straight into the other person's eyes.

There are several other cultural differences facing young overseas doctors training in Britain. These include, for example, calling older people by their name and holding the hand of a patient of the opposite sex to express sympathy and concern, which are considered to be offensive in most Asian countries but are cultural norms in Britain.

My little experience in Britain makes me realise that, though the science of medicine is the same all over the world, the art of medicine—that is, being a good clinician—is different, being influenced by local customs, manners, and ways of life. Junior doctors coming from overseas to train in Britain would benefit from guidance on these differences. It would help us to understand our mistakes and save us from many embarrassing clinical experiences.

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1 Qureshi B. Eye, eye. *Br Med J* 1989;**298**:1230. (6 May.)

Drug Points

Acute hepatitis and exfoliative dermatitis associated with minocycline

Drs M G DAVIES and P J W KERSEY (Plymouth General Hospital, Plymouth PL4 8NN) write: Minocycline is a tetracycline analogue that is often prescribed for acne and other cutaneous disorders. We recently observed two patients with a similar clinical pattern consisting of a severe self limiting skin eruption and acute hepatic failure, which in one case was fatal. Both patients had received minocycline for a skin complaint a few weeks earlier.

Case 1—A 16 year old boy presented with hidradenitis suppurativa and was treated with minocycline 100 mg twice a day. Four weeks later he became unwell with headache, malaise, fever, and rash. The rash was initially erythematous and patchy and later confluent and exfoliative with oozing over the skin of the face. He developed a sore throat and night sweats. Examination showed an ill young man with an enlarged liver and generalised lymphadenopathy. His throat was red with inflamed tonsils. His skin showed generalised dryness and erythema. He was anaemic with a haemoglobin of 108 g/l, a raised white cell count of $25{\cdot}8\times10^9$/l (66% neutrophils, 20% lymphocytes, 12% eosinophils), and a raised platelet count of 840×10^9/l. A bone marrow examination showed hypercellularity but no other abnormality. Other investigations, including a test for hepatitis B antibodies, gave normal or negative results. During the course of the illness the streptococcal anti DNAase B became positive, suggesting recent streptococcal infection. Liver enzyme activities, which were initially raised, rose further with a maximum γ-glutamyltransferase of 395 IU/l. His skin exfoliated in sheets. Within a month the skin improved and the hepatic enzymes returned towards normal. The lymph nodes and hepatomegaly regressed. His subsequent recovery was uneventful.

Case 2—A 17 year old girl developed an acneiform eruption on her cheeks and neck and was treated with minocycline 50 mg twice daily. Shortly afterwards she went on holiday to Kenya, where she took proguanil and chloroquine and some tanning tablets containing tyrosine, copper, and para-aminobenzoic acid as well as minocycline. She was immunised against cholera, typhoid, and yellow fever. Twenty nine days after starting minocycline and while still taking it she presented with a high fever and a macular rash. Seven days after admission her rash appeared to clear and her temperature returned to normal. She was dis-

charged from hospital but readmitted 24 hours later with relapse. The rash was more severe, exfoliative, and superficially pustular, and her fever became undulant. Investigation showed a mild anaemia (haemoglobin 114 g/l), a very high leucocyte count ($22{\cdot}6\times10^9$, 52% neutrophils), and abnormal liver function values (γ-glutamyltransferase 417 IU/l, aspartate transaminase 182 IU/l, and alkaline phosphatase 560 IU/l hepatic isoenzyme). During her illness the antistreptococcal DNAase B became positive, suggesting a recent streptococcal infection. The skin improved but liver function deteriorated; she went into hepatic coma and was transferred to King's College Hospital, but despite hepatic transplantation she died.

These cases followed a similar clinical pattern. In both continuous treatment with minocycline was followed after 32 days (case 1) and 29 days (case 2) by a severe exfoliative rash that became pustular with an inflammatory element showing features of both toxic epidermal necrolysis and erythema multiforme. The rash improved after 10 days (case 1) and 14 days (case 2). The liver function tests were abnormal at 42 days (case 1) and 37 days (case 2). In both they deteriorated but were showing evidence of improvement, in case 1 at 60 days. A curious feature was the positive antistreptococcal DNAase B, the titre of which rose during the early part of the illness in both cases. This test is much more specific for a streptococcal infection than the antistreptolysin O titre and appears to reflect true recent infection. The role of the streptococcal infection is not clear; it appears to have immediately preceded the illness and may have played a part as a cofactor. It was not solely responsible for the illness as neither a skin eruption of that type nor hepatic failure is a recognised feature of streptococcal infections. The rash was unusual, with features of erythema multiforme and toxic epidermal necrolysis most likely to be seen in a drug eruption. The liver failure might also have been drug induced and appeared to have been so in the absence of positive hepatitis titres. Other infective causes in case 2 were extensively sought and not found.

There is one previous case report of hepatitis associated with minocycline in a patient with an aspiration pneumonia.[1] In that case the pneumonia, from which cultures grew *Staphylococcus aureus* and *Streptococcus salivarius*, had been treated with minocycline and ampicillin. The Committee on Safety of Medicines has received nine reports of hepatic dysfunction associated with minocycline and eight reports of rashes, one of which was described as exfoliative dermatitis.

We believe that there is a case for caution in the use of minocycline for minor skin complaints and would be interested to hear of any other patients with a similar history to our two patients.

1 Burette A, Finet C, Priogine T, De Roy G, Deltenre M. Acute hepatic injury associated with minocycline. *Arch Intern Med* 1984;**144**:1491-2.

Idiosyncratic dapsone induced manic depression

Drs ANDREW J CARMICHAEL and C J PAUL (Skin Hospital, Birmingham B15 1PR) write: We have recently seen a case of manic depressive psychosis induced by dapsone and unrelated to leprosy.

A 37 year old white woman had suffered recurrent attacks of erythema multiforme for seven years, localised to the skin of the arms. Attacks occurred most months, with the lesions spontaneously settling over two weeks. Investigations for an underlying cause, including lupus erythematosus, gave negative results, and she was otherwise well. A more extensive episode of erythema multiforme affecting the face prompted us to start dapsone 100 mg/day in view of our recent experience of response to this treatment.[1]

Twenty four hours after starting the drug the patient had a two day episode of hypomania, with flight of ideas, pressure of speech, marked hyperactivity, and lack of need for sleep. After three more days she entered a depressive state, becoming withdrawn, low in affect, and tearful, with no change in her life circumstances. She gave no personal or family history of psychiatric problems and was taking no other medication. Her haemoglobin concentration was normal and methaemoglobin was undetected. Dapsone was stopped after 10 days' treatment and within a fortnight her mood had returned to normal. The erythema multiforme cleared after a week's treatment with dapsone but relapsed three weeks after discontinuing treatment.

Dapsone associated "psychosis" has been the subject of controversy.[1] The debate has been muddied both by imprecise classification of the psychosis and by all but one[2] recorded case occurring against a background of leprosy, which may itself be associated with psychotic symptoms.[3] Some have suggested that "dapsone psychosis" is dose related,[4] occurs only in those with a background of psychiatric problems,[4] and is therefore partly predictable. In view of the strong temporal relation of our patient's symptoms to dapsone treatment and her recovery after its withdrawal we believe that her manic depressive psychosis was a side effect of dapsone. Rechallenge was not thought to be ethical given the severity of the psychiatric symptoms. This manic-depressive reaction, like the severe agitation in the non-lepromatous patient described by Fine *et al*,[2] appeared after a small dose of dapsone with no predisposing factors and was therefore unpredictable.

1 Durha P, Paul CJ. Continuous erythema multiforme clearing on dapsone. *Br J Dermatol* 1988;**118**:731.
2 Fine JD, Katz SI, Donahue MJ, Hendricks AA. Psychiatric reaction to dapsone and sulfapyridine. *J Am Acad Dermatol* 1983;**9**:274-5.
3 Jopling WH. Antileprosy drugs. *Br Med J* 1971;iv:366.
4 Molesworth BD, Narayanswami PS. Toxic effects of diaminodiphenylsulphone. *Lancet* 1952;i:562-3.

Breathlessness in patients with prostatic carcinoma treated with cyproterone acetate

MR N A GREEN (Norfolk and Norwich Hospital, Norwich) and DR B D W HARRISON (West Norwich Hospital, Norwich NR2 3TU) write: Breathlessness in men with prostatic carcinoma is not uncommon and may be due to coexisting heart or lung disease, pulmonary metastases, fluid retention associated with oestrogen therapy, pulmonary emboli, or anaemia. We describe three patients who complained of breathlessness within a month of starting treatment with cyproterone acetate, none of whom had any of these causes of breathlessness.

The three men with carcinoma of the prostate were all smokers and each had evidence of mild or moderate airways obstruction on pulmonary function tests (table), but none had previously complained of breathlessness. One man (case 3) also had pleural calcification after artificial pneumothoraces for pulmonary tuberculosis 40 years earlier. Each patient noticed and complained of breathlessness on exertion within a month of starting cyproterone acetate. Two were dyspnoeic while undressing. None had wheezed or experienced orthopnoea. Blood gas pressures and pH are also shown in the table. All showed a normal arterial oxygen pressure, a low carbon dioxide pressure, and a high normal pH with a low bicarbonate concentration, which indicated a chronic or compensated respiratory alkalosis. Repeat studies in two of the patients after three and 13 months, while they were still taking cyproterone acetate, showed the persistence of the chronic respiratory alkalosis. Two men stopped cyproterone (table) and noticed an improvement in breathlessness within a few days. Further studies in one man 16 months after he stopped treatment showed that his hyperventilation had ceased and his arterial carbon dioxide pressure and pH were both normal.

Progesterone and progestogens stimulate ventilation and increase the sensitivity of the respiratory centre to carbon dioxide.[1] One of the causes of breathlessness during pregnancy is hyperventilation caused by the increased progesterone secretion,[2] and medroxyprogesterone has been used as a respiratory stimulant in patients with chronic ventilatory failure.[3] Cyproterone acetate acts as an antiandrogen by blocking androgen receptors. It also has progestogenic activity, exerting a negative feedback effect on the hypothalamic receptors, thus diminishing gonadotrophin release.[4]

Our patients attributed their pulmonary symptoms to the cyproterone since all noticed breathlessness soon after treatment started. None had sufficient underlying lung disease or sufficiently abnormal pulmonary function to account for their dyspnoea. All had normal arterial oxygen pressures and evidence of a chronic or compensated respiratory alkalosis consistent with a chronic stimulus to hyperventilation. In one man the dyspnoea and acid base abnormality resolved after the cyproterone was stopped. In another, who declined further tests, breathlessness improved after cyproterone was stopped. In the absence of any other cause of breathlessness we postulate that it was due to the progestogenic effects of cyproterone acetate increasing ventilation in patients with a pre-existing mild to moderate degree of abnormal pulmonary function.

1 Weinberger SE, Weiss ST, Cohen WR, Weiss JW, Johnson TS. Pregnancy and the lung. *Am Rev Respir Dis* 1980;**121**:559-81.
2 Lyons HA, Antonio R. The sensitivity of the respiratory centre in pregnancy and after the administration of progesterone. *Trans Assoc Am Physicians* 1959;**72**:173-80.
3 Sutton FD, Zwillich CW, Creagh CE, Pierson DJ, Weil JV. Progesterone for outpatient treatment of Pickwickian syndrome. *Ann Intern Med* 1975;**83**:476-9.
4 Eaton AC, McGuire N. Cyproterone acetate in treatment of post-orchidectomy hot flushes. Double blind crossover trial. *Lancet* 1983;**ii**:1336-7.

Respiratory and blood gas values in three patients taking cyproterone acetate for prostatic carcinoma

Case No	Age (years)	Cyproterone acetate treatment	FEV_1* (l)	FVC* (l)	Pa_{O_2} (kPa) (11-13)†	Pa_{CO_2} (kPa) (4·8-6·3)†	pH (7·35-7·45)†	Bicarbonate (mmol/l) (22-32)†	Date
1	70	100 mg thrice daily Started Dec 86	3·28 (2·59)	4·84 (3·78)	13·6	3·2	7·428	15·9	18 Mar 87
		Stopped Jun 87			13·1	3·6	7·439	17·9	9 Jun 87
					10·9	4·6	7·433	22·8	31 Oct 88
2	67	100 mg thrice daily Started Nov 86	1·74 (2·46)	3·14 (3·49)	13·6	2·7	7·449	13·7	26 Oct 87
					13·7	3·8	7·437	18·9	14 Nov 88
3	70	100 mg thrice daily Started Jun 88 Stopped Sep 88	2·51 (3.04)	4·45 (4.37)	12·9	3·7	7·450	19·3	3 Aug 88

FEV_1=Forced expired volume in one second. FVC=Forced vital capacity. Pa_{O_2}=Arterial oxygen pressure. Pa_{CO_2}=Arterial carbon dioxide pressure.
*Figures in parentheses are predicted normal values. † Normal range.