years of age and sometimes younger, in addition to those aged over 75 years who are mentioned in the discussion papers. The diagnostic range includes the whole range of psychiatric morbidity, not only the demented.³

Here is a hot spot of new and worthwhile activity, the very essence of the flexible responsive approach health services require. Performance indicators that cannot identify or monitor its dimensions will lead to the withdrawal of resources and the activity will cease.

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1 Performance Indicator Group. *Performance indicators for the NHS*. London: DHSS, 1987.

2 Wattis J, Arie T. Further developments in psychogeriatrics in Britain. *Br Med J* 1984;289:778.

3 Arie T, Jolley D. Making services work: organisation and style of psychogeriatric services. In: Levy R, Post F, eds. *The psychiatry of late life*. Oxford: Blackwell Publications, 1982: 221-51.

Reviewing RAWP

SIR,—The articles by Mr Bevan and Mr Brazier (3, 10, 17 October, p 836, 898, 989) raise the question of how many beds teaching hospitals need. If we assume that regional specialties and centre of excellence referrals can be identified separately then three bed requirements remain—for teaching, research, and local population needs. Obviously each may overlap with some of the others. One may assume that research needs can be met from the other functions. A further confounding factor (partly dealt with in the previous articles) is any excess or unmet need associated with the urban deprivation which is common around many teaching hospitals.

The Resource Allocation Working Party (RAWP) formula assumes that resources for the population can be equated with money, whereas most authorities are looking at ways of keeping a particular service running. When the bulk of the RAWP money comes from the population part of the formula the two things come together.

Many teaching districts have more beds than their population needs and thus they are dependent on a trading position to maintain their beds. Mr Bevan and Mr Brazier have shown that this can lead to some absurd situations and suggest that juggling the formula is unlikely to work and hence seem to imply that the current unsatisfactory situation must continue. An alternative suggestion is to look more closely at why teaching hospitals have all these beds.

Let us assume a separate mechanism in RAWP to fund regional specialties and centre of excellence referrals and also assume that we know the case mix and bed numbers required for these patients. We then have to ask, What is the epidemiology of the medical curriculum? How many cases of what should the average medical student see? What level of certainty is needed on these estimates and hence what size population would produce that many cases for any given number of medical students? From that we could estimate the required number of beds by a simple multiplication of case numbers by length of stay, corrected for occupancy levels and subtracting any beds already filled by suitable regional specialty or centre of excellence patients. Each region then has to find a way of protecting that number of beds for teaching and making sure that a suitable population can get into those beds. Once those calculations are made teaching could be taken off the front end of RAWP. Such a process

could be conducted nationally or within regions and could be done fairly quickly provided everyone remembered that a fairly crude solution is probably all that is needed.

This debate could also open up some useful discussion about why medical students have to learn so much of their craft around beds when it is quite clear that much of the medicine of the next century will be practised in outpatients and in the community and, if the World Health Organisation is to be believed, will have a greater emphasis on prevention.

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Achieving a balance

SIR,—The right way to "achieve a balance" is not that endorsed by the BMA (31 October, p 1152). It is, of course, mathematically obvious that feeding a pool of consultants with average tenure 30 years from a registrar/senior house officer pool occupied for only 10 years will necessarily result in an imbalance between them, which could be corrected within the present constraints only by curtailing the former or expanding the latter—that is, lengthening the training period or shortening the period of service as a consultant, neither of which is desirable.

We should begin by asking what kind of a service is needed for patients before working out a scheme catering for the career aspirations of doctors. The health service suffers from a paucity of ranks (how would an army function that consisted only of lieutenants and generals?), from the pretence that these two ranks represent the trained and the trainees, and from the equation of tenure with the achievement of consultant status with the divide at an age when insecurity usually affects a whole family.

We could reorder the hospital hierarchy into ranks occupied roughly for a decade at each level—10-12 years as senior house officer and registrar, 10-12 years as senior registrar or junior specialist, 10-12 years as senior specialist, and, for some only, a final decade as chef de service or his deputy (the rest retiring or changing roles). It would do no harm if each change of status was sometimes accompanied by a change of health authority; and there would be circumstances in which promotion was not seen as automatic and possibly secured by election.

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Self experimentation in medicine

SIR,—Dr Stephen Lock in his review of a recent book *Who Goes First? The Story of Self Experimentation in Medicine* (17 October, p 968) describes astonishing feats of both courage and recklessness.

We are told that Purkinje performed 35 experiments on himself and Murrell took nitroglycerin some 40 times. While a student in Edinburgh my great grandfather, Dr John Webb Watkins, offered himself to Sir James Young Simpson, who described the anaesthetic properties of chloroform, for "the purpose of experiment, and was the first person to whom he (Simpson) administered it" (Obituary, *Lancet*, 14 February 1903). In his graduation thesis ("A Dissertation upon Anaesthesia in Natural and Morbid Parturition," 1865) he presented a vivid description of sensations experienced during anaesthesia which he observed

"when I have been anaesthetised—now forty or fifty times." Is this a record?

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Living with one eye

SIR,—Professor Calbert I Phillips's entertaining *Personal View* (31 October, p 1133) incidentally confirmed a long held personal suspicion that few ophthalmologists know the full consequences of total loss of vision in one eye. It is true that "Nature gave us two eyes, so there is one to spare," for the loss of an eye is not disabling, as I found out 68 years ago. But the sudden change from binocular to monocular vision does demand a number of adjustments, on which, I suspect, little advice is given. So one learns the hard way how to surmount the real, if small, obstacles now suddenly placed in one's path.

There are several simple things one learns to do and a few one learns not to attempt. Those learning processes must be easier for an 11 year old than they are for individuals in their 70s, but both would be much helped by a little guidance. The loss of visual field is enough to demand extra care crossing the road and to increase the risk of turning into an obstacle on one's blind side—especially a half open door. That loss is immediately increased if spectacles are worn; even the change from a monocle to spectacles as one's aging skin fails to hold the monocle reliably is a limitation.

But Professor Phillips's remark "Stereoscopic vision is of little consequence" makes me wonder if he knows how the one eyed individual gets round the difficulty: reaching nearly to an object quickly, but feeling for the last few inches; always touching the cup or glass rim before pouring into it; constantly turning one's head and watching for kerb edges or raised paving stones; always feeling with one's foot for the height of the first step down; never hitting across the line of a moving ball, whether at cricket, tennis, or squash. To the one eyed golfer all greens are flat. The special requirements of the one eyed driver are more easily appreciated, but the paramount consideration is that he cannot estimate the distance from his near side wing accurately by looking at it. I commend to Professor Phillips F B Brady's little book *A Singular View*. He had to learn the hard way too.

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Drug points

Angiotensin converting enzyme inhibitors and diuretics

Dr BRIAN COOK (Ninewells Hospital and Medical School, Dundee DD1 9SY) writes: With reference to Mr J S Budd's and Dr M A R Houghton's report of an interaction of captopril and dyazide causing hypotension and abdominal pain (5 September, p 612), I would like to draw attention to another drug interaction associated with captopril and combined preparations of potassium sparing and losing diuretics.

We admitted an 85 year old man with painful muscle cramps and dehydration which had confined him to bed for two days. He had longstanding mild congestive cardiac failure and a minor degree of chronic renal failure. He had been treated with Frusene (frusemide 40 mg plus triamterene 50 mg) one tablet a day for some months, but because of worsening of his ankle oedema this had been increased to three tablets and he had been given captopril 25 mg/day some two weeks previously. On admission his

blood biochemical values were: urea 21.2 mmol/l, creatinine 428 µmol/l, sodium 143 mmol/l, chloride 112 mmol/l, potassium 7.2 mmol/l. Muscle cramps due to dehydration and hyperkalaemia were diagnosed and his diuretics and captopril were stopped. He was treated with an insulin-dextrose infusion and calcium resonium. Over the next two days his cramps improved considerably and his serum potassium concentration fell to 4.8 mmol/l. He was restarted on frusemide 40 mg/day and 10 days later was ready for discharge with good control of his heart failure. Biochemical values then were: urea 14.8 mmol/l, creatinine 311 µmol/l, sodium 142 mmol/l, chloride 105 mmol/l, potassium 4.0 mmol/l.

This case highlights the potentially dangerous interaction between angiotensin converting enzyme inhibitors and potassium sparing diuretics and the resultant rise in serum potassium concentration. Administration of captopril to patients taking diuretics causes a rise in serum potassium concentration.¹ This is associated with a fall in circulating aldosterone values² and reduced potassium loss from the distal tubule. Captopril and other angiotensin converting enzyme inhibitors block the production of angiotensin II, which is the main stimulant to aldosterone release. Concurrent administration of potassium sparing diuretics or potassium supplements with captopril has been associated with hyperkalaemia.^{2,3} This was probably the cause in our patient, with his mild chronic renal failure also contributing, as captopril causes a rise in serum potassium values in chronic renal failure.^{1,4} The large dose of potassium sparing diuretic was probably also important. Hyperkalaemia with angiotensin converting enzyme inhibitors and potassium sparing diuretics is well recognised, but with combined preparations of potassium sparing and losing diuretics this interaction may be overlooked. Further confusion may arise with Frusene or similarly named preparations as these could potentially be mistaken for plain frusemide. This interaction may be so serious³ that practitioners should have a clear knowledge of patients' diuretic or other treatment when initiating treatment with angiotensin converting enzyme inhibitors, and this risk should be borne in mind.

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- 2 Textor SC, Bravo EL, Fouad FM, Tarazi RC. Hyperkalaemia in azotemic patients during angiotensin-converting enzyme inhibition and aldosterone reduction with captopril. *Am J Med* 1982;73:719-25.
- 3 Packer M, Lee WH. Provocation of hyper- and hypokalemic sudden death during treatment with and withdrawal of converting enzyme inhibition in severe chronic congestive heart failure. *Am J Cardiol* 1986;57:347-8.
- 4 Zanella MT, Mattei E, Braibe SA, Kater CE, Aizen H. Inadequate aldosterone response to hyperkalaemia during angiotensin converting enzyme inhibition in chronic renal failure. *Clin Pharmacol Ther* 1985;38:613-7.

Does captopril exacerbate psoriasis?

Drs N W HAMLET, M KEEFE, and REBECCA E I KERR (Department of Dermatology, Stobhill General Hospital, Glasgow G21 3UW) write: The most commonly reported side effects of captopril have been rashes.¹ These are most often pruritic, maculopapular, and limited to the upper half of the body but may resemble pityriasis rosea² or take the form of an urticarial erythema with eczematous features leading to erythroderma.³ We have recently seen a 40 year old woman with a 16 year history of psoriasis, which flared dramatically after she took captopril for hypertension. Her psoriasis had previously been a stable plaque type, affecting the scalp, knees, and elbows, with minimal lesions elsewhere, and had never been itchy. In April 1987 she started taking captopril 12.5 mg twice daily for hypertension. She was taking no other drugs. After two weeks she developed a pruritic, scaly eruption, which was typical of guttate psoriasis although unlike the usual plaque pattern of her disease. The distribution was mainly central, affecting the back, buttocks, and posterior thighs. There was no history of sore throat or other illness, and an antistreptolysin O titre was 160 Todd units/ml. Captopril was continued. Outpatient treatment was un-

successful, but inpatient treatment with tar, dithranol, and ultraviolet light cleared the psoriasis in August, and she subsequently remained clear. We cannot be certain that captopril exacerbated her psoriasis, but the time course makes an association plausible. The mode of presentation was consistent with a primary psoriatic reaction and we think it unlikely that psoriasis developed as an isomorphic (Köbner) phenomenon after an ordinary drug rash. There was no need to withdraw the drug, but rashes during captopril therapy can be transient and do not necessarily require cessation of therapy. The possible mechanism of exacerbation is obscure, but rashes from captopril are thought to result from an allergic reaction to the sulphydryl component⁴ or from cutaneous kinin potentiation due to angiotensin converting enzyme inhibition,⁵ although this has been disputed.⁶ We are not aware of published reports of this association, but one of the manufacturers (E R Squibb and Sons Ltd) has received two reports. In one case there was thought to be no causal relation, but the second patient developed severe psoriasis after changing from atenolol to captopril and required admission to hospital and withdrawal of the drug. We have reported our case to the Committee on the Safety of Medicines.

We thank Miss L Gilbert of the drug information unit, Stobhill General Hospital, for reviewing the literature.

- 1 Di Bianco R. Adverse reactions with angiotensin converting enzyme (ACE) inhibitors. *Medical Toxicology* 1986;1:122-41.
- 2 Wilson KK, Hammond JJ, Kirkendall WM. The captopril induced eruption. *Arch Dermatol* 1980;116:902-5.
- 3 Goodfield MJ, Millard LG. Severe cutaneous reactions to captopril. *Br Med J* 1985;290:1111.
- 4 Gavras I, Gavras H. Captopril and enalapril. *Ann Intern Med* 1983;98:556-7.
- 5 Edwards JR, Coulter DM, Beasley DMG, MacIntosh D. Captopril: 4 years of post marketing surveillance of all patients in New Zealand. *Br J Clin Pharmacol* 1987;23:529-36.
- 6 Navis GJ, De Jong PE, Kallenberg CGM, et al. Absence of cross-reactivity between captopril and enalapril. *Lancet* 1984;ii:1017.

Psoriasis as a side effect of β blockers

Drs M KEEFE, N W HAMLET, and REBECCA E I KERR (Department of Dermatology, Stobhill General Hospital, Glasgow G21 3UW) write: Dr Jaakko Savola and colleagues (12 September, p 637) described exacerbations of pre-existing psoriasis or the development of psoriasis in 10 patients receiving β blockers for hypertension or angina pectoris. They comment that angiotensin converting enzyme inhibitors were substituted in the hypertensive patients. A high prevalence of high renin essential hypertension has been claimed in psoriasis,¹ and this seems to be a good reason for using this class of drugs. In a recent study 10 psoriatic patients with hypertension were treated with captopril. Three patients, however, developed reversible heavy proteinuria, and the authors concluded that there should be careful monitoring of urinary protein concentrations when the drug is used in people with psoriasis. Three patients also found that previously resistant skin lesions improved.² The reason for this is unclear, however, and the authors were careful not to make a causal inference. In contrast, we have reported on a patient whose psoriasis flared dramatically after taking captopril (see accompanying report). β Blockers are widely used and have a good safety record. We suggest that doctors should carefully consider the advantages and disadvantages of other drugs before changing their prescribing habits because of coexisting psoriasis.

We thank E R Squibb and Sons Limited for information referred to in this letter.

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- 2 Madeddu P, Ena P, Dessi Fulgheri P, et al. Captopril induced proteinuria in hypertensive psoriatic patients. *Nephron* 1986;44:358-60.

Toxic coma induced by anticholinergic eye drops

Drs J NADAL, V DE LA FUENTE, M ABADIAS, J TORRENT, and F JANÉ (Hospital de St Pau, Avda S

Antoni M Claret 167, 08025 Barcelona, Spain) write: Anticholinergic eye drops are regularly used by ophthalmologists to obtain mydriasis and cycloplegia. The most widely used are those containing atropine or scopolamine. Depending on the intensity and duration of the desired pharmacological effects, one or two drops are instilled in the conjunctival sac.^{1,2} Many side effects including systemic reactions³ may occur as a result of absorption of the topical drug particularly in children.⁴ We describe a serious adverse reaction, which to our knowledge has not been reported.

A 6 year old boy, weighing 22 kg, was brought to the emergency room with a decrease in consciousness. He showed coma with agitation, dilated pupils, dry skin and mouth, facial flushing, a blood pressure of 120/70 mm Hg, and a heart rate of 160 beats/min, which increased progressively, reaching 230 beats/min with multifocal ventricular extrasystoles. Meningismus was absent. During an ophthalmoscopic examination one hour earlier the patient had been given two drops in each eye of an ophthalmic solution containing 2% atropine sulphate, 0.5% scopolamine bromhidrate, and 4% phenylephrine hydrochloride; the amounts instilled were about 3 mg, 0.75 mg, and 6 mg respectively. Atropine intoxication was considered and 0.5 mg of physostigmine salicylate was given in an intravenous infusion over five minutes and repeated five minutes later. Ten minutes later the tachycardia decreased to 115 beats/min with a progressive recovery from peripheral manifestations, although cognitive functions did not recover until 16 hours later. After 48 hours the patient was discharged with a residual mydriasis.

Toxic psychosis and delirium have been reported after routine instillation of eye drops containing anticholinergic agents for refraction.⁵ Systemic absorption probably takes place via the nasolacrimal duct through the nasal mucosa or from swallowed atropine laden tears through the walls of the gastrointestinal tract. Atropine and scopolamine can produce both systemic effects, including confusional states, delirium, and toxic psychosis among other central nervous system manifestations,⁶ and peripheral postganglionic cholinergic effects. In certain sensitive subjects these effects may arise with therapeutic doses.⁷ Recently a toxic psychosis was reported in a 6 year old girl who received an overdose, confirmed by measurements of free scopolamine plasma values from a transdermal scopolamine patch.⁸

The appearance of coma after the administration of a single dose of eye drops together with the fact that these kinds of drugs are widely used for inducing mydriasis and cycloplegia, should alert ophthalmologists to the severe central toxicity of these compounds, even at normal doses and especially in children. Neither the manufacturer (Laboratorios Llorens) nor the Department of Clinical Pharmacology and Drug Surveillance has received reports of such a side effect. We recommend reducing the risk of side effects from ophthalmic application of atropine, for instance by compressing the internal angle of the eye to obstruct the lacrimal duct during instillation of the drops.⁹ In children less concentrated atropine solutions, or other compounds with a lower central toxicity such as cyclopentolate or tropicamide, should be used instead of atropine alone or with scopolamine.

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- 6 Birkhimer LJ, Jacobson PA, Olson J, Goyette DM. Ocular scopolamine induced psychosis. *J Fam Pract* 1984;18:466-9.
- 7 Tune LE, Holland A, Folstein F, Damlouji NF, Gardner TJ, Coyle T. Association of postoperative delirium with raised serum levels of anticholinergic drugs. *Lancet* 1981;ii:651-2.
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