difficult to postulate that the patient had a rare degenerative disorder of the central nervous system, an unexplained defect of calcium metabolism, and a mysterious conjunctivitis.

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¹ Smith, M J, and Hey, G B, *Postgraduate Medical Journal*, 1976, **52**, 86.

² Berlyne, G M, and Shaw, A B, *Lancet*, 1967, **1**, 4.

An unusual case of cerebrovascular disease

SIR,—I would like to raise a few points in connection with the recent clinicopathological conference (17 March, p 727).

The first patient presented with amaurosis fugax, in which it is generally accepted that embolism plays some part. The carotid angiogram showed an irregularity which could have been a source of platelet emboli. It is suggested that the stereotyped nature of the attacks makes embolism less likely, but experimental evidence¹ has shown that emboli may repeatedly go to the same site, owing perhaps to streaming of blood flow. The report mentions the blood pressure inequality between the arms and raises the possibility of subclavian stenosis and "steal," but this is most unlikely with a pressure difference of only 10 mm Hg and no bruits.

Surprise is expressed at the patient's remission while taking only aspirin, but this has well-known antiplatelet effects and is standard treatment for a patient with presumed platelet embolisation. Emboli from an atheromatous plaque may range from microemboli to large pieces and it is possible, in view of the fact that the vertebral arteries were occluded, that one of these caused death by obstructing the blood flow to the brain stem. It would be interesting to have necropsy information about the circle of Willis and associated arteries. This is a situation in which surgery to the carotid bifurcation could have been potentially life saving, as the risk of major embolic complications of such lesions is well known.

In addition, since we do not know whether antiplatelet drugs protect against stroke in patients who have had transient ischaemic attacks, it seems sensible to maintain them on these relatively safe drugs until trials have shown whether or not they are of value.

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¹ Wylie, E J, and Ehrenfeld, W K, *Extracranial Occlusive Cerebrovascular Disease*, ch 3. Philadelphia, W B Saunders, 1970.

Cardioselective beta-blockade and renal function

SIR,—We would like to confirm the observation by Dr R Wilkinson that atenolol has less effect on renal function than non-selective beta-blockers (3 March, p 617).

In a crossover study (n=27) of atenolol and three non-selective beta-blockers in hypertension¹ it was found that at doses producing equal reduction in systolic blood pressure serum creatinine level during atenolol treatment—92 μ mol/l (1.04 mg/100 ml)—was a little, but significantly (P<0.01), lower than during non-selective beta-blockade with

oxprenolol, propranolol, or pindolol—98 μ mol (1.11 mg/100 ml).

A double-blind randomised crossover study of atenolol and identical placebo tablets in eight subjects with mild essential hypertension and normal serum creatinine levels has now been completed. Renal function was assessed during the eighth week of each treatment period by a double-isotope, single-injection technique using ⁵¹Cr-EDTA clearance for the estimation of glomerular filtration rate² and ¹²⁵I-iothalamate clearance for the estimation of renal blood flow.³ Glomerular filtration rate and effective renal plasma flow were below the normal range during placebo treatment (1.18 ml/s and 7.40 ml/s respectively) but did not diminish further to any significant extent on atenolol (1.22 ml/s and 7.06 ml/s). Serum urea was significantly higher on atenolol—6.7 mmol/l (40.36 mg/100 ml)—than on placebo—5.6 mmol/l (33.73 mg/100 ml)—but serum creatinine did not change.

Thus treatment with atenolol would appear not to cause significant reduction of renal function.

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¹ Waal-Manning, H J, *Clinical Pharmacology and Therapeutics*, 1979, **25**, 8.

² Bailey, R R, et al, *Australasian Annals of Medicine*, 1970, **3**, 255.

³ Blaurock, M D, et al, *Nephron*, 1966, **3**, 274.

Peripheral skin necrosis complicating beta-blockade

SIR,—I was interested in the report by Dr R Gokal and his colleagues (17 March, p 721) of peripheral skin necrosis complicating beta-blocker therapy. I have seen two such patients who, like Dr Gokal's patients, had no evidence of large artery disease.

The first, a 54-year-old man, presented in January 1975, having developed painful red toes within days of starting propranolol, 40 mg thrice daily, for hypertension. After three weeks' treatment he was noted to have an ulcerated little toe and bounding foot pulses suggestive of small vessel disease. His toe healed and his vascular symptoms disappeared on his stopping propranolol, and have not recurred.

The second patient, a 66-year-old woman on propranolol, 10 mg thrice daily, for angina, developed peripheral ulceration of a toe some weeks after increasing the dose marginally, to 20 mg twice daily, in February 1977. Although her feet felt cold the foot pulses were strong. The propranolol was stopped and the toe healed. However, the patient restarted propranolol of her own accord two months later as her angina deteriorated, and she has taken as much as 40 mg twice a day up to the present without further skin necrosis.

It thus appears that the absence of clinical peripheral arterial disease provides no guarantee against beta-blocker-induced skin necrosis and that the development of necrosis is not an absolute contraindication against further use of a beta-blocker.

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Drug-induced peripheral neuropathies

SIR,—Dr Z Argov and Professor F L Mastaglia (10 March, p 663), in their useful review "Drug-induced peripheral neuropathies," refer

to hydralazine as an agent responsible for "mild or subclinical" sensory neuropathy in as many as 15% of cases. In this context it is important to mention that side effects of this type (including systemic lupus erythematosus) were reported some 20 to 25 years ago, when hydralazine in high dosage was used, often as the sole agent, in the treatment of hypertension.

More recently, in the United States, hydralazine has been used in doses of up to 200 mg daily, in combination with other agents such as reserpine and diuretics, with very few toxicity problems. Perry reviewed the literature comprehensively in 1973,¹ pointing out that the evidence that late hydralazine therapy might represent a deficiency state was rather meagre.

Now that hydralazine and other vasodilating drugs are being used successfully with β -adrenergic blockers in the management of moderate-to-severe hypertension, it would be a pity if clinicians were misled by data from a previous era. It is the experience of clinicians today that peripheral neuropathy occurs rarely, if at all, when doses of hydralazine in the range of 100-200 mg daily are used. Indeed, no reports of neurological reactions appear in the Committee on the Safety of Medicines register of adverse drug reactions for the period July 1963 to June 1976.

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¹ Perry, H M, *American Journal of Medicine*, 1973, **54**, 58.

Accidental hypothermia and low-reading thermometers

SIR,—This winter has been the coldest since 1963. It is not unreasonable to suppose that there should have been an increase in the number of patients suffering from accidental hypothermia. The condition is common, as was shown in one survey during the relatively mild winter of 1975, when the incidence among the elderly admitted to hospital was 3.6%.¹

In spite of considerable national and local publicity, few cases have been noticed in the new Royal Liverpool Hospital. During the first five days of this year no cases were recorded in the casualty and admissions department. A possible reason for this was the absence of low-reading thermometers in the department. When commissioned, the hospital had an issue of low-reading thermometers but at the beginning of the year none was available on the wards—except in the geriatric department—or in the hospital stores. After discussion with the senior nursing staff the old thermometers are to be phased out and replaced by low-reading thermometers, without additional expense.

It would be interesting to know how many other hospitals do not use low-reading thermometers as a matter of routine. Admittedly, a low-reading thermometer does not guarantee that the mercury will be shaken down, but it may help to detect this lethal condition when the patient is first seen. Those patients suffering from accidental hypothermia do not complain of feeling cold, and the general use of low-reading thermometers in both hospitals and general practice may

improve the detection rate and possibly aid recovery as a result of early diagnosis and subsequent management.

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¹ Goldman, A, et al, *Journal of the Royal College of Physicians of London*, 1977, **11**, 291.

Rugby injuries involving the hand

SIR,—With reference to the letter (10 February, p 414) by Drs R W Nutton and M J Guy, it is not stated what kind of dislocations occurred. The common injury is the posterior one, which is usually no trouble; it is the anterior dislocation that is liable to cause rupture of the middle slip of the extensor tendon. This is not generally realised and is not in many of the textbooks. X-rays are not always taken after this injury, reduction being done on the spot; so it may not be known what type of dislocation occurred. So if after this injury a patient cannot fully extend the proximal interphalangeal joint, then he should be treated at once in a lively splint to prevent the Boutonniere deformity, which is a disabling and difficult condition to treat once it has developed.

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SIR,—With reference to the "Letter from Soweto" (17 March, p 739), allow me the following comments. First of all, I wish to state that I think this article is a reasonably fair account of the situation as it pertains at the present moment at Baragwanath, but certain statements do require revision in the light of recent events and perhaps others require some explanation.

I believe it is now a matter of record that salary discrimination among the medical profession in South Africa will soon be a thing of the past. I in fact expect that when this letter is published this will have already occurred to a large extent.

With regard to the hospital facilities, it is indeed true that there is an enormous demand for hospital beds for the black section of our community. In the light of what I have to say further on, the reason for this may become more apparent. Mr Sakalo is correct in stating that a new hospital has replaced the old Johannesburg General Hospital. What he has not mentioned, and perhaps did not know, is that a 2000-bed hospital is being built at the Medical University of South Africa, and that the University of the Witwatersrand medical faculty and also the University of Pretoria medical faculty in association with the Medical University of South Africa are involved in its implementation and its personnel development.

Finally, allow me to highlight several sentences in Mr Sakalo's final remarks. He states, "Many of the patients are treated for afflictions and diseases resulting from poverty and overcrowding." A little later he remarks "Malnutrition, in particular, is endemic among the black population and a study by the University of Pretoria has shown that 75 black children die of malnutrition every day." These two remarks should perhaps be seen in

the context of some other remarks he makes: "The maternity wards sprawl over 50 hectares of land and cope with a 2.7% birth rate, which is a rate higher than India's"; and a little further on he states, "Even in this modern day, in one of the most advanced black societies, witch doctors still retain a strong hold on the people."

While in no way trying to minimise our concern with the infant mortality rate and malnutrition (primarily a paediatric problem), the concerted effort by all the authorities to inculcate a concern for family planning programmes and for proper infant care and feeding has met with resistance from the major proportion of our black citizens who still cling to the old customs. I nevertheless believe that the programmes which have been instituted by our Department of Health and our faculties must ultimately bear fruit as the sophistication of our black peoples evolve.

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Schizophrenia or obsessive-compulsive neurosis?

SIR,—We were interested to read the case of Dettol addiction described by Dr J S Khan and others (24 March, p 791). The patient's psychiatric symptoms were a fear of acquiring syphilis, associated Dettol drinking to "purify" herself, and a morbid cleansing obsession, including use of Dettol-soaked pessaries. The patient was diagnosed as having a schizophrenic illness. Schizophrenia and obsessive-compulsive neurosis can be difficult to differentiate¹ and recent work² has suggested that resistance is not an essential feature of the latter, making the distinction even less clear. The limited description in the paper would certainly fit an obsessive-compulsive neurosis, which is usually amenable to behavioural treatment.³ If the patient died as a consequence of compulsive cleansing rituals—chronic ingestion of Dettol—this would be almost unique.

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¹ Stengel, E, *Journal of Mental Science*, 1944, **91**, 166.

² Stern, R S, and Cobb, J P, *British Journal of Psychiatry*, 1978, **132**, 233.

³ Marks, I M, Hodgson, R, and Rachman, S, *British Journal of Psychiatry*, 1975, **127**, 349.

Proteinuria at high altitude

SIR,—In your leading article (24 February, p 508) you discuss the physiological changes that occur on ascending to high altitude, with an emphasis on proteinuria. Some of the data reported are open to criticism.

Pines¹ reported studies on proteinuria in the early morning urine specimens of seven people climbing at heights of up to 5890 m. However, the urine protein was measured by an inaccurate method (Bili-Labstix), urine volumes were not taken into account, and there were insufficient data to allow any statistical correlation of proteinuria with altitude, exercise, or the development of acute mountain sickness. Rennie and Joseph² completed a more detailed study of proteinuria at altitude. They showed a good correlation of proteinuria and altitude, but again only early morning

urine samples were tested and an unsatisfactory correction for urine dilution had to be made.

During a recent expedition to the Himalayas³ 17 doctors and scientists collected 24-hour urine samples over a 14-day period during an ascent to 5500 m. In the group as a whole there was no correlation between proteinuria and altitude, although some individuals had an increase in proteinuria at altitude. Proteinuria was of no value in predicting and did not relate to the development of acute mountain sickness.

The effects of altitude on the body and the development of acute mountain sickness are difficult to study because of the large individual variation and problems associated with sample collection. However, progress will not be achieved by inadequate data collection and drawing unwarranted conclusions. For instance, there have been at least five controlled trials at altitude of the drug acetazolamide (which is widely considered to be of value in the prevention of acute mountain sickness); yet not one of these studies was well controlled under routine trekking conditions, and use of this drug cannot therefore at present be recommended.

At a recent symposium, shortly to be published,⁴ many clinical and biochemical aspects of altitude and acute mountain sickness were discussed. However, the mechanisms, early diagnosis, and prevention of the illness remain an open question.

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¹ Pines, A, *British Journal of Diseases of the Chest*, 1978, **72**, 196.

² Rennie, I D B, and Joseph, B, *Lancet*, 1970, **1**, 1247.

³ Birmingham Medical Research Expeditionary Society, October 1977.

⁴ *Postgraduate Medical Journal*, 1979, suppl, in press.

Nutritional intake, adiposity, and diabetes

SIR,—Professor Harry Keen and others (10 March, p 655) appeared surprised that, among their subjects, those with a high energy intake had less adiposity; they also had lower blood sugar concentrations and better glucose tolerance, and would be less likely to develop diabetes. High energy intakes had been associated with high intakes of total carbohydrate.

Professor J N Morris and his colleagues had also been surprised to record that among their subjects high energy intake had been associated with lower incidence of coronary heart disease; this had occurred in men eating more cereal dietary fibre, but not that derived from vegetables and fruit.¹ It would be helpful if Professor Keen gave us information concerning the dietary fibre in his diets: dietary fibre is an essential nutrient.

These findings do not surprise doctors who have worked in tropical Africa and observed a low incidence of obesity, diabetes mellitus, and coronary heart disease among those eating large amounts of lightly processed, high-fibre starch foods.² The dietary fibre hypothesis of diabetes mellitus has two aspects, even if only the first aspect is usually quoted. (1) Dietary-fibre-depleted energy foods tend to produce obesity and mature-onset diabetes in susceptible phenotypes. (2) Large amounts of high-fibre energy foods improve diabetes mellitus and obesity.