

cases of travellers' diarrhoea belonged to serogroups previously associated with infantile gastroenteritis. Indeed, one of our toxigenic strains could not be typed at all. Therefore in screening for ETEC in travellers with diarrhoea not associated with *Salmonella* and *Shigella* we feel that it is important to underline the need to test several colonies of *E coli* isolated from the primary culture, regardless of serotype.

Full details of this work will be published separately.

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<sup>1</sup> Guerrant, R L, *et al*, *New England Journal of Medicine*, 1975, **293**, 567.

<sup>2</sup> Dean, A G, *et al*, *Journal of Infectious Diseases*, 1972, **125**, 407.

<sup>3</sup> Echeverria, P, Blacklow, N R, and Smith, D H, *Lancet*, 1975, **2**, 1113.

<sup>4</sup> Bach, E, Blomberg, S, and Wadstrom, T, *Infection*, 1977, **5**, 2.

### Pituitary suppression in chronic airways disease?

SIR,—We were interested to read the letter "Pituitary suppression in chronic airways disease?" by Dr Pd'A Semple and others (19 May, p 1356). We have been assessing pituitary function in our patients with radiological changes in the pituitary fossa,<sup>1</sup> including also a further group of male hypercapnic patients.

Preliminary results show that in two-thirds of the patients a subnormal serum testosterone level was associated with low or normal levels of serum luteinising hormone (LH) and follicle-stimulating hormone (FSH). Moreover, in these patients the LH response to an intravenous injection of 100 µg of gonadotrophin-releasing hormone was impaired, supporting the suggestion of Dr Semple and his colleagues that abnormal hypothalamic-pituitary function is present in these patients. Basal thyroid-stimulating hormone (TSH) levels and TSH responses to thyrotrophin-releasing hormone were normal.

Thus evidence is mounting of endocrine dysfunction mainly involving hypothalamic-pituitary-gonadal function in these patients, for which there may be a therapeutic dividend.

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<sup>1</sup> Newton, D A G, Bone, I, and Bonsor, G, *Thorax*, 1978, **33**, 684.

### A luxury drug?

SIR,—Some issues of the *BMJ* have carried treble-page colour spreads advertising Timoptol. Never in my 40 years of ophthalmology have we been exposed to such a barrage of salesmanship. Yet from none of the representatives and copious brochures, the various glaucoma symposia subsidised by the manufacturers, or the reports from radio and television and the national and medical press has there been any mention of the one major non-asset, its enormous price.

The manufacturers deserve particular re-

proof for directing their publicity unabashedly at the layman, so that we oculists are now being constantly assailed by patients demanding the new wonder treatment, which they or their friends have all seen, heard, or read about (a little blame, too, to the media for conniving at the propaganda).

We appreciate that research is always costly, but beta-blockers are not so expensive to prepare, particularly in such tiny quantities; yet the cost of Timoptol, even at its cut-rate for NHS hospitals, is about 25 times that of the pilocarpine we use (£5 for a 5 ml bottle compared with about 20p). It was reckoned by the pharmacist of one major eye hospital that, if all our glaucoma patients were changed over from pilocarpine to Timoptol, this would swallow up well over half of our entire annual drug budget. The loss to the taxpayers would be vast, given that there are around a quarter of a million people with glaucoma in the UK.

Timoptol has indeed advantages over pilocarpine in certain cases; it is sad that, if we are to remain solvent, so few will be able to afford this luxury. If only the manufacturers could have spent less flamboyantly on all that advertising, and charged us a little less for those few drops.

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### Heatstroke in a "run for fun"

SIR,—In his letter (20 January, p 197) Dr Tom Bassler describes four cases of death in marathon runners, each of which he ascribes to heatstroke. We would like to point out that in none of these cases is there sufficient evidence to implicate heatstroke as the primary diagnosis, although as three of these athletes died during prolonged exercise the possibility that an elevated body temperature, in contradistinction to heatstroke, may have played some role cannot be excluded.

The first case, reported in detail by Green *et al*,<sup>1</sup> was that of a 44-year-old runner who collapsed, pulseless and apnoeic, after running 24 miles of the 1973 Boston marathon. Cardiopulmonary resuscitation was instituted. On admission to hospital the patient was in ventricular fibrillation and his rectal temperature was 38.4°C, features which are both inconsistent with a diagnosis of heatstroke. In five large series<sup>2-6</sup> of exertion-related heatstroke, involving 297 patients, there was not one reported case of ventricular fibrillation or of cardiac arrest. To support a diagnosis of heatstroke in the patient of Green *et al* there would have to be an explanation of why the rectal temperature on hospital admission was only 38.4°C. In a group of 30 heatstroke victims, a mean rectal temperature of 41.2°C was recorded even half an hour to two hours after the initial collapse.<sup>5</sup> Thus the combination of ventricular fibrillation and a low rectal temperature makes a diagnosis of heatstroke untenable unless further information is forthcoming.

Because sudden death due either to ventricular fibrillation or to cardiac arrest is not a feature of heatstroke, it also follows that heatstroke was not the cause of death in two of the other three cases of death among our South African marathon runners—a 19-year-old athlete who died suddenly during a marathon race and a 47-year-old who collapsed and died in sight of the finish of an eight-mile mountain race. The fourth case, that of a 35-year-old highly trained athlete, has been fully reported,<sup>7</sup> yet Bassler fails to report all the clinical features. For the benefit of your readers, we would like to restate what we consider to be significant features of this case.

A 35-year-old athlete developed chest pain or pain between the shoulder blades or both on six of eleven runs in January 1974. The pain was severe

enough to force him to stop running on a number of occasions. He did not run on the day of his death, choosing rather to go surfing. However, while surfing he became "too breathless" to continue. He left the water and drove home, but within the hour he developed severe precordial chest pain. He was driven to his physician, who referred him to hospital. During the car ride he complained that his chest pain was now worse and that his left hand felt paralysed. He died shortly after admission to hospital. The electrocardiogram showed ST depression in leads 2, 3, and AVF. In the absence of a necropsy, ischaemic heart disease as the cause of death seems likely but cannot be proved. However, on the basis of the clinical history, heatstroke can absolutely be excluded as a diagnostic possibility.

We agree with Dr Bassler that heatstroke is a menace in long-distance running and we did in fact first draw attention to this as early as 1973.<sup>8</sup> By incorrectly attributing all cases of exertional collapse in marathon runners to heatstroke Dr Bassler is not, as he claims, increasing the safety of this sport, because he ignores the dangers imposed by other conditions such as coronary heart disease and hypertrophic cardiomyopathy, both of which occur in marathon runners.<sup>7, 9, 10</sup>

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<sup>1</sup> Green, L H, Cohen, S I, and Kurland, G, *Annals of Internal Medicine*, 1976, **84**, 704.

<sup>2</sup> Malamud, N, Haymaker, W, and Custer, R P, *Military Surgeon*, 1946, **99**, 397.

<sup>3</sup> Barry, M E, and King, B A, *South African Medical Journal*, 1962, **36**, 455.

<sup>4</sup> Kew, M C, Tucker, *et al*, *American Heart Journal*, 1969, **77**, 324.

<sup>5</sup> Shibolet, S, *et al*, *Quarterly Journal of Medicine*, 1967, **36**, 525.

<sup>6</sup> Costrini, A M, *et al*, *American Journal of Medicine*, 1979, **66**, 296.

<sup>7</sup> Noakes, T, *et al*, *Annals of the New York Academy of Sciences*, 1977, **301**, 593.

<sup>8</sup> Noakes, T D, *South African Medical Journal*, 1973, **47**, 1968.

<sup>9</sup> Noakes, T D, Rose, A G, and Opie, L H, *British Heart Journal*, in press.

<sup>10</sup> Noakes, T D, *et al*, *New England Journal of Medicine*, in press.

### NHS security beds

SIR,—Your leading article on Butler-type regional security units (16 June, p 1585) has given encouragement to those of us who publicly opposed the setting up of these units, and who favoured instead the concept of the "simple staff-intensive units."

Mr S Quinn, nursing officer of the Lyndhurst Unit at Knowle Hospital, Fareham (the first and, so far as I know, the only open-door, simple staff-intensive unit in existence), discussing the efficacy of the unit, has said, "...inquiries in February indicated that virtually no patients with mental illness were going to prison in Wessex who should certainly be in a hospital. There are none at present, to our knowledge, being held up in special hospitals for want of a bed in Wessex. Regional health authorities are no longer receiving complaints that beds cannot be found for those difficult mentally ill patients, though there is still a problem with the subnormal patients."

If this is the case, what a tragedy that such specialist open-door forensic units were not set up many years ago—a tragedy both in terms of the suffering of the patients waiting for transfer in grossly overcrowded Broadmoor and also in terms of the harm done to relationships between those working in the special