if it was so, this would be the longest latency period recorded. Moreover, unlike other cases occurring after surgery,⁵ no communication between the abscess and the previous field of surgery was demonstrated.

I wish to thank Mr J Spencer for his permission to publish this case.

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¹ *British Medical Journal*, 1973, **1**, 365.

² Minocha, V R, et al, *Australian and New Zealand Journal of Surgery*, 1975, **45**, 66.

³ Emmons, C W, et al, *Medical Mycology*, 2nd edn, p 76. Philadelphia, Lea and Febiger, 1970.

⁴ Davies, M, et al, *British Journal of Surgery*, 1973, **60**, 18.

⁵ Adar, R, et al, *Israel Journal of Medical Science*, 1972, **8**, 143.

Treatment of acute paracetamol poisoning

SIR,—Some of the statements in your leading article on the treatment of acute paracetamol poisoning (20 August, p 481) need qualification.

(1) Oral methionine has been recommended for the routine treatment of paracetamol overdose by the poisons unit at Guy's Hospital for all cases reported since 1974.¹ During 1975 they received 755 inquiries relating to paracetamol poisoning. Methionine treatment was recommended for all patients reputed to have taken more than 15 g of paracetamol (presenting within 10 hours of ingestion). The total number given methionine is not recorded, but the results from 36 treated cases admitted to the liver unit at King's College Hospital and to the Guy's Hospital intensive care unit have been reported by Crome *et al*.² Thus, although the use of cysteamine has been more widely reported than that of methionine, the adoption of methionine as the recommended treatment of paracetamol poisoning in the south of England has led to its widespread use.

(2) Cysteine and methionine increase the amount of glutathione in the liver. However, cysteamine does not,³ but appears to act mainly through inhibiting a cytochrome P450 mixed-function oxidase, thus reducing the oxidation of the paracetamol to the toxic intermediate.

(3) The relationship between hepatic damage and paracetamol half lives was originally reported by Prescott in 1971.⁴ However, a more accurate method of predicting possible hepatic damage based on the pharmacokinetics of paracetamol has been designed and reported.⁵ This method relies on rapid and accurate methods of blood level estimation of paracetamol, such as the blood paracetamol estimation kit,⁶ a technique for determining paracetamol levels on the ward not mentioned in your article.

(4) Cysteamine injection is not commercially available. Cysteamine is rapidly oxidised in solution to cystamine, but stable formulations of cysteamine injection have been developed.⁷⁻⁹ Nevertheless, as these formulations have not been developed commercially the preparation requires a pharmacy with a sterile products production unit. (Prescott *et al*¹⁰ use a freeze-dried preparation prepared jointly by the pharmacy and the blood transfusion unit.) Thus cysteamine injection is not available in all hospitals. Methionine is readily available as powder or tablets and, as

it can be given orally, poses no such pharmaceutical manufacturing problems.

The report of the Royal College of Physicians of London symposium on paracetamol and the liver¹¹ lists the majority of these comments and gives the rationale behind the development of the treatments for acute paracetamol poisoning.

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¹ Volans, G N, *Journal of International Medical Research*, 1976, **4**, suppl 4, p 7.

² Crome, P, et al, *Journal of International Medical Research*, 1976, **4**, suppl 4, p 106.

³ Andrews, R S, et al, *Journal of International Medical Research*, 1976, **4**, suppl 4, p 39.

⁴ Prescott, L F, et al, *Lancet*, 1971, **1**, 519.

⁵ Recommended Management of a Case of Paracetamol Overdose, *Journal of International Medical Research*, 1976, **4**, suppl 4, p 157.

⁶ Kendal, S E, Lloyd-Jones, G, and Smith, C F, *Journal of International Medical Research*, 1976, **4**, suppl 4, p 83.

⁷ Prior, F G R, *Pharmaceutical Journal*, 1974, **1**, 578.

⁸ Brouwers, J R B J, and Vermeij, P, *Lancet*, 1976, **2**, 965.

⁹ Prior, F G R, *Lancet*, 1977, **1**, 315.

¹⁰ Prescott, L F, et al, *Lancet*, 1974, **1**, 588.

¹¹ Symposium on Paracetamol and the Liver: Overdose and its Management, *Journal of International Medical Research*, 1976, **4**, suppl 4.

Ventricular arrhythmias caused by prenylamine

SIR,—With reference to the paper by Dr R Puritz and others (3 September, p 608) we would like to comment that their recommendations are in line with our own, which stress that prenylamine should be used with caution in patients taking certain other cardioactive drugs or drugs likely to induce hypokalaemia.

Our data sheet states that "prenylamine should be used with caution and in reduced dosage when combined with negative inotropic drugs—for example, β -adrenergic blocking agents, quinidine, etc, or drugs likely to produce hypokalaemia. Isolated reports indicate that on rare occasions such combinations are capable of producing paroxysmal ventricular tachycardia or fibrillation. Under such circumstances a marked increase in the duration of the Q-S or Q-T interval above the rate-adjusted norm is an indication of the need to withdraw treatment with prenylamine."

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Skeletal scintigraphy in carcinoma of the breast

SIR,—Your leading article (3 September, p 592) does well to draw attention to the need for a diagnostic imaging strategy in the evaluation of metastatic breast cancer.

Skeletal isotope scanning followed by conventional radiology of abnormal areas so demonstrated¹ is a realistic and economic approach. Patients with positive scans and negative radiographs then pose a problem. False-positive isotope scans of the spine may be due to degenerative joint disease of intervertebral or costovertebral origin or osteophyte formation. Plain radiographs may be unhelpful.

This dilemma is the subject of an ongoing study in this department² employing computed tomography (CT) with the EMI whole-body scanner. CT enables a precise analysis to be made of horizontal cross-sections 8 or 13 mm in thickness. Preliminary observations³ on 30

patients with breast cancer and positive isotope scans of the spine suggest that differentiation by CT between metastatic and degenerative joint disease even when the two are coincident in the spine is readily made. Lytic deposits unsuspected on conventional radiographs are demonstrable by CT.

It would seem that where whole-body CT is available it could be used in the "positive scan, negative radiograph" situation and certainly unnecessary bone biopsy avoided.

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¹ Roberts, J G, et al, *Lancet*, 1976, **1**, 237.

² Best, J J K B, et al. In preparation.

³ Isherwood, I, and Best, J J K B, Third International Symposium on Computer Tomography, Miami, April 1977.

Hygiene in NHS hospitals

SIR,—We have read the letters from Dr G A J Ayliffe and Mr B J Collins (2 July, p 48) and Mr G Aston and Dr H Gordon (27 August, p 579) with interest and would like to draw attention to the fact that for a considerable time Cuckfield and Crawley Health District has been aware of the importance of kitchen hygiene.

For a number of years past an open invitation has been extended to environmental health officers of the three local authorities within the health district boundary to visit, without prior notice, any of the hospital kitchens within the district for inspection purposes. After each visit a report is forwarded to district headquarters. A working party, consisting of the medical officer for environmental health, general administrator (support services), the local authority environmental health officer concerned, the unit catering manager, the unit administrator, and a representative of the works department, meets to discuss and where possible implement recommendations of the report. By this process not only is progress made towards achieving a high standard of kitchen hygiene but the local authority environmental health officer is aware of any financial constraints which might delay the early implementation of any of the recommendations. The essential rapport between officers of differing authorities and disciplines is also beneficially maintained.

BASIL RAE BURN

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Bell's palsy and herpes simplex

SIR,—Herpes simplex virus (HSV) has been suggested, on theoretical grounds, as a possible cause of Bell's palsy.^{1,2} I am able to find only one report of clinical HSV infection associated with Bell's palsy and that describes an HSV type II infection in a man with genital and oral ulcers immediately after the development of facial palsy.³ The present report describes a case of primary herpes stomatitis followed by mild Bell's palsy.

A European male aged 36 became unwell with a high temperature, multiple generalised mouth ulcers, and cervical lymphadenopathy. There was no suggestion of hand, foot, and mouth disease (more correctly, hand, foot, mouth, and bumps-a-