hypoxia and decreased renal perfusion so that urea formation is increased and excretion hindered. This could lead to a moderately elevated plasma urea and slightly elevated plasma creatinine, which might account for the apparently inferior discriminating power of the urea:creatinine ratio observed by your contributors when compared with the individual measurements of urea and creatinine.

Incidentally, the correlation between plasma urea and creatinine in the 350 unselected patients may have been adversely affected if the authors included patients with keto-acidosis. It is well known that acetoacetate and acetone interfere in the Jaffé reaction for creatinine³ and the plot of urea versus creatinine showed a number of points with seemingly disproportionate high creatinine concentrations.

K WIENER

Department of Pathology,
North Manchester General
Hospital

¹ Wiener, K, *Clinica Chimica Acta*, 1976, **73**, 45.

² Wiener, K, *Clinica Chimica Acta*, 1977, **76**, 243.

³ Cook, J G H, *Annals of Clinical Biochemistry*, 1975, **12**, 219.

Treatment of acute paracetamol poisoning

SIR,—As a postscript to your leading article on the treatment of acute paracetamol poisoning (20 August, p 481) and the letter it provoked by Mr F G R Prior (24 September, p 829) commenting on the use of cysteine, methionine, and cysteamine, we wish to report what we believe to be the highest recorded plasma paracetamol level in a fatal case of self-poisoning.

An 18-year-old girl with a history of depression and previous overdose was admitted in an unconscious state having taken an unknown quantity of Paramol 118 (paracetamol 500 mg and dihydrocodeine 10 mg) tablets 12 hours previously. She was unresponsive to pain and had dilated pupils, and was cyanosed and breathing poorly, and severely hypotensive. Plasma paracetamol level was 1082 µg/ml.

She was treated with intravenous cysteamine, ventilation, and intravenous fluids. Her blood pressure improved and she began to respond to pain. On day 2, the plasma paracetamol level had fallen to 617 µg/ml. The serum aspartate aminotransferase, which had been 250 U/l on the day of admission, had risen to 644 U/l. Serum albumin remained at 23 g/l and the prothrombin activity was 20% of normal.

Twenty-four hours post admission, she was breathing spontaneously with normal blood gases; however, at 40 hours post admission she developed spontaneous ventricular fibrillation and the post-resuscitation electrocardiogram showed myocardial damage. Necropsy showed small haemorrhages in the visceral pericardium, bronchopneumonia of the lower lobes of the lungs, and central and midzonal liver necrosis.

This case demonstrates the clinical effects of hypoalbuminaemia, a bleeding state, liver cell damage, and electrocardiographic evidence of myocardial damage as noted by Maclean *et al*¹ in severe cases of acute paracetamol poisoning.

In Prescott's group of patients,² in which the use of cysteamine was first reported, the highest plasma paracetamol level was 542 µg/ml; but this was only four hours after ingestion. In other papers we have examined which dealt with earlier attempted methods of treatment, we have not found higher levels than that reported by Prescott—which leads us to believe that our case is exceptional in this regard. The case described also highlights

the problem of how to manage severe paracetamol overdose presenting late after ingestion but before overt hepatic failure.

GORONWY JONES
PETER THOMAS

Neath General Hospital,
Glam

¹ MacLean, D, *et al*, *Lancet*, 1968, **2**, 849.

² Prescott, L T, *et al*, *Lancet*, 1974, **1**, 588.

Primary treatment of prostatic cancer

SIR,—The leading article describing the management of primary prostatic cancer (24 September, p 781) adequately covered the present position and outlined the difficulties faced by clinicians in deciding which therapy, if any, to recommend. It would appear that the choice lies between doing nothing, radical surgery, radiotherapy, or cytotoxic therapy. All three therapies carry a definite morbidity and one of the side effects is immunosuppression, and they are suggested for elderly men whose immune competence is on the wane.¹

There is an alternative that demands the controlled trials suggested in your editorial; it carries little morbidity and might stimulate a specific immune response to the tumour. By 1967 it had been demonstrated² that prostatic tissue contained specific antigens to which antibodies could be produced by cryosurgery. Interest in cryoprostectomy was stimulated in the hope that similar results could be obtained in prostatic tumours. Perhaps because the equipment then available was rather daunting, the control of freeze poor, and the cryodestruction of tissue less well understood, few centres persisted. Those that did consistently produced good relief of obstruction.^{3 4} The question of immunostimulation remains open but there is enough encouragement to justify further clinical trials.^{5 6}

Modern liquid nitrogen equipment is easy to use, cell kill can be assured within defined limits, and patient acceptance is high. The present position and methods have been summed up by Wilkinson.⁷ Perhaps some of the money still available for cancer research should be channelled into giving a thorough trial of one of the few treatments combining good local effect with specific immunotherapy. Perhaps then your editorial would be a little less gloomy and more positive.

PETER J DOYLE

Hardgate,
Dunbartonshire

¹ Ablin, R J, *Urology*, 1975, **5**, 317.

² Yatorno, C, *et al*, *Immunology*, 1967, **12**, 395.

³ Sesia, G, *et al*, *Panminerva Medica*, 1975, **17**, 390.

⁴ Ablin, R J, *et al*, *Cryobiology*, 1971, **8**, 271.

⁵ Ablin, R J, *et al*, *Cryobiology*, 1974, **11**, 218.

⁶ Ablin, R J, *et al*, *Oncology*, 1975, **32**, 127.

⁷ Wilkinson, F O W, *Practical Cryosurgery*, ed H B Holden. London, Pitman Medical, 1975.

SI units

SIR,—We are fed up with SI units. We write on behalf of our colleagues at this hospital to ask if there are others who, like us, have not found any advantage in using the new units, which only cause frustration and confusion.

Surely it is not too late to admit having made a mistake and to return to the traditional units which were in use two years ago. It would be wrong for a minority group to do this alone, so may we, through you, ask if there are others who think as we do and would be

prepared to join us in reverting to the old units?

J STEPHEN LAW
J M STANSFELD

Dryburn Hospital, Durham

Arthritis and primary biliary cirrhosis

SIR,—We should like to support the suggestion of Dr D L Child and his colleagues (27 August, p 557) that an association between arthritis and primary biliary cirrhosis is more frequent than has hitherto been recognised.

In a personal series of 137 cases (six male) of primary biliary cirrhosis collected from over the West of Scotland there were 10 (7.3%) cases of seropositive rheumatoid arthritis and one of a biliary arthropathy. Rheumatoid factor was present in 73 (64%) out of 114 patients tested compared with 17 (16%) out of 108 age- and sex-matched controls ($P < 0.001$). However, the R3 titre (Warner Laboratories) was $\leq 1/64$ in 72% of the positive cases. Therefore the test for rheumatoid factor was mainly weakly positive and its immunoglobulin class was not identified. These figures enhance the association previously reported in two other series of primary biliary cirrhosis, in one of which¹ articular symptoms were recorded in 4% of 47 patients (rheumatoid factor positive in 24%) and in the other² rheumatoid arthritis was present in five of 100 patients.

The evidence for occult liver disease in patients with primary rheumatoid arthritis is scanty. In Glasgow,³ of 216 patients with rheumatoid arthritis, 23 (10.6%) had hepatomegaly, 12 (5.6%) bromsulphalein retention, and seven (3.2%) a positive mitochondrial antibody. Liver biopsy was performed on only seven members of the group, showing primary biliary cirrhosis in two, fatty change or mild chronic inflammatory cell infiltrates in three, and normal appearances in two. Further studies of liver histology in rheumatoid arthritis recently carried out in Glasgow have shown mild non-specific pathological changes to be the rule, perhaps brought about by drug therapy.

P MILLS
R N M MACSWEEN
G WATKINSON

Departments of Medicine and
Pathology,
Western Infirmary, Glasgow

¹ Golding, P L, Smith, M, and Williams, R, *American Journal of Medicine*, 1973, **55**, 772.

² Sherlock, S, and Scheuer, P J, *New England Journal of Medicine*, 1973, **289**, 674.

³ Webb, J, *et al*, *Annals of the Rheumatic Diseases*, 1975, **34**, 70.

Sunlighting in medicine

SIR,—Professor D N Baron (22 October, p 1089) should be so lucky only to have 3½ hours' clinical work to do each day. Out in the sticks I rarely do less than 6 hours' actual work with patients each day, and frequently 12 hours. I am a member of seven committees, of which I chair two, to say nothing of the innumerable subcommittees they spawn. In case my (restricted) salary, for 31½ hours per week, should be too much I am on call for a further 54 hours a week. And I know many consultants across the country with considerably greater responsibilities.

Is this full "daylighting"?

A E CARTER

King Edward Memorial Hospital,
London W13