have died as a result of skateboarding in America, but how many million skateboards are in use over there?

My impression, for what it is worth, is that skateboarding is a lot safer than rugby or boxing, and infinitely safer than travelling in a car or on a motorcycle. Skateboards are like cars: they are not dangerous, but the people who use them may be. Why must doctors always be seen to be promoting the negative rather than the positive in people's lives? We live in a culture of isolation and boredom; we should be trying to do something about that before we condemn activities out of hand without the information necessary to back it up.

S GLASCOE

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[1] *Daily Telegraph*, 1 September 1977.

Switching off

SIR,—I write to protest against the practice of asking relatives for permission to disconnect life support systems in cases where brain death has been diagnosed. This is both illogical and cruel and in many cases may prove to be counterproductive.

It is illogical because the relatives are not involved in the decision to put the patient on life support. Nor indeed could they be. The decision to initiate life support is a medical one and by the same token the decision to disconnect is a medical one. Fortunately, we now have clear guidelines on the diagnosis of brain death, which are contained in the statement issued by the honorary secretary of the Conference of Medical Royal Colleges and their Faculties in the United Kingdom on 11 October 1976.[1]

It is cruel because relatives are placed in an agonising dilemma by being asked to agree to the switching off. They do not have the medical knowledge to make a scientific judgment so they are, in effect, being asked to make a moral judgment. But in asking for a moral judgment at such a time we are placing them on the horns of a dilemma. If they say yes, they may feel they are taking a life. If they say no, they may feel they are unnecessarily prolonging death or indefinitely prolonging a useless life. Without full medical knowledge they cannot give a reasoned answer and whichever answer they give they will be left with doubts. What the relatives need is support in their distress and confidence in the judgment of their medical advisers. Our duty is to make up our minds and then quietly and sympathetically inform the relatives that life support will be withdrawn because we have determined by a battery of tests that the brain is dead.

Asking the relatives for permission to switch off is likely to be counterproductive both to the patient and to others. If permission is refused and a useless life is prolonged then an intolerable burden will be placed on the relatives. Moreover, the unnecessary postponement in switching off may result in such physical deterioration of the patient that his kidneys can no longer be used to save another life.

In conclusion, I would like to emphasise that I am not suggesting that the relatives should not be *involved* in the decision to withdraw life support. I am suggesting that they should not be asked to make the decision. We as a medical profession should have the courage of our convictions and recognise that it is our responsibility to make the decision on the medical evidence.

A W FOWLER

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[1] *British Medical Journal*, 1976, **2**, 1187.

The neonatal electrocardiogram and unexpected death in infancy

SIR,—We do not agree with the negative opinion expressed by Professor J L Emery (24 September, p 833) on the value of a major ECG screening study to investigate the possible relationship between cardiac arrhythmias and unexpected infant deaths. The population in Sheffield was selected and relatively small. Furthermore, no information was given on the type of ECG or the duration of the rhythm strips carried out.

We have demonstrated, using 24-hour monitoring, that some infants with normal screening ECGs have serious cardiac arrhythmias. We still consider a prospective multicentre study essential to determine whether cardiac arrhythmias occur regularly in infants throughout the country, whether they are dangerous, and finally whether they can cause sudden death.

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Oxytocin and neonatal jaundice

SIR,—Several UK centres have reported a recent increase in neonatal jaundice.[1 2] Oxytocin or some as yet unidentified obstetric practice has been held responsible.[3 4] However, there is some doubt that a genuine increase in the incidence of neonatal jaundice has occurred since serial figures[1 5] are few and the incidence of jaundice increases with the proportion of neonates whose plasma bilirubin is measured. We have sent questionnaires to paediatricians in Europe (E) and North America (A) asking their recent experience of jaundice rates.

Sixty-six members of the European Society for Paediatric Research were circularised; 52 replied. A random 1 in 5 sample of the American Pediatric Society, total 135, were also circularised; 100 replied. Thirty-nine of the Europeans and 51 of the North Americans were in active practice in neonatology. The majority, 66% (E) and 60% (A), thought there had been no increase in neonatal jaundice since 1970. Only 23% (E) and 24% (A) thought jaundice had increased. The remainder were uncertain. The respondents' estimate of the present percentage of neonates having a peak plasma bilirubin concentration of 206 μmol/l (12 mg/100 ml) or greater in the first 7 days after birth varied between 2 and 30%. The averages of all the replies, 14·2% (E) and 12·6% (A), were similar to the published incidence in unselected series.[5] We asked also about the use of phenobarbitone in late pregnancy for the prophylaxis of neonatal jaundice. Only 13% (E) and 8% (A) paediatricians used this occasionally; none used it routinely.

If oxytocin were icterogenic then we would have expected most paediatricians in North America and Europe to have noticed some increase in neonatal jaundice. The fact that they have not casts further doubt on this association.

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[1] Campbell, N, *et al*, *British Medical Journal*, 1975, **2**, 548.
[2] Sims, D G, and Neligan, G A, *British Journal of Obstetrics and Gynaecology*, 1975, **82**, 863.
[3] Beazley, J M, and Alderman, B, *British Journal of Obstetrics and Gynaecology*, 1975, **82**, 265.
[4] Chew, W C, and Swann, I L, *British Medical Journal*, 1977, **1**, 72.
[5] Lewis, P J, *Therapeutic Problems in Pregnancy*, p 141. Lancaster, Medical and Technical Publishing, 1977.

Sealing of wounds with vacuum drainage

SIR,—Most surgeons these days make use of vacuum drainage with zimmer bottles or similar apparatus and many surgeons no doubt have learnt the value of sealing these wounds and the emergent drainage or drains with such adhesive films as Opsite or Steridrapes. These substances are, however, expensive and I have found that simple household Cling, so useful in the kitchen, makes a most satisfactory substitute.

In view of the lack of sterility, it is best to cover the wound and emergent drain or drains with a small strip of gauze or ribbon gauze. The whole area is then generously sprayed with Onibuctane and the film of Cling spread over. This technique is of especial help when a vacuum tube has to be replaced from without in through a new stab wound. The Cling gives rise to a new and most effective vacuum seal.

C PATRICK SAMES

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Raised plasma urea concentration

SIR,—Surprise was expressed by Professor D B Morgan and others (8 October, p 929) on finding heart failure to be the commonest cause of a raised plasma urea in a general hospital. In a study which I undertook on subjects suffering from acute myocardial infarction, 50% had abnormally high plasma urea concentrations and 41% had high creatinine concentrations at some time during their stay in hospital.[1] Since such cases constitute a substantial proportion of hospital admissions, the observation made by these workers is perhaps not so surprising after all, and presumably their congestive heart failure group would include subjects of this type.

Urine estimations on a small number of patients in my investigation indicated that while glomerular filtration was sometimes decreased, as judged by creatinine clearance, urea excretion over 24 hours tended to be normal or high. Although the two studies are not strictly comparable, being designed for different purposes, some similarities do exist. For example, in both cases mortality was associated with the highest levels of plasma urea, and my results suggested that daily plasma urea measurement might be helpful in monitoring progress and prognosis in acute myocardial infarction.[2]

Professor Morgan and his associates list decreased glomerular filtration rate as one of three general causes of a raised plasma urea, and in heart failure this may result from poor renal perfusion. Hence the uraemia occurring in this condition seems likely to have poor circulation as its primary cause, but with a mixed secondary aetiology of tissue breakdown due to stagnant tissue

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 ketoacidosis. It is well known that acetoacetate and acetone interfere in the Jaffé reaction for creatinine[3] and the plot of urea versus creatinine showed a number of points with seemingly disproportionate high creatinine concentrations.

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[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 overdosage presenting late after ingestion but before overt hepatic failure.

GORONWY JONES
PETER THOMAS

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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 demonstrated[2] that prostatic tissue contained specific antigens to which antibodies could be produced by cryosurgery. Interest in cryoprostatectomy 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.

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[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?

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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 which[1] articular symptoms were recorded in 4% of 47 patients (rheumatoid factor positive in 24%) and in the other[2] 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.

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[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?"

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