tical solution. Mr. Illich foresees a cataclysmic collapse of our present institutionalized values and Dr. Bradshaw envisages a similar end. One would like to think that this particular era of *La condition humaine* will end "not with a bang but with a sigh."

There may be many small but vital pockets of hope and resistance standing out in the Western world. Let us hope that some of these have among their number "men of medicine."—I am, etc.,

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Starving Children before Operation

SIR,—Your leading article on this subject (27 July, p. 213) was both timely and stimulating. Thomas[1] has proposed an effective and practical solution to the problem of hypoglycaemia in infants and small children. This would be practicable for afternoon lists but must have severe limitations for those treated as day patients on morning lists. For over two years it has been a regular practice in this hospital to encourage patients to drink up to one hour prior to receiving premedication before general anaesthesia, using metoclopramide to stimulate emptying of the stomach. A series of over 400 cases has been performed safely.[2] Since the publication of your article this technique has been adapted to infants and young children as a means of raising the blood sugar during operation. The following case report will be instructive.

A child 18 months old weighing 11·1 kg was admitted as a day case for orchidopexy. At 8.30 a.m. the blood sugar was 35 mg/100 ml, though there were no signs of hypoglycaemia. The child was given 125 ml of 20% glucose flavoured with orange and metoclopramide 2·5 mg was injected intramuscularly. At 9.30 a.m. 10 mg of hyoscine *N*-butylbromide was injected intramuscularly with 500 IU of hyaluronidase. A smooth and uneventful induction of anaesthesia with nitrous oxide, oxygen, and halothane followed two minutes later. The operation ended at 10.10 a.m., when a second blood sugar estimation gave a level of 165 mg/100 ml. Three days later a blood sample was taken to assess the normal blood sugar of the child and the reading then was 85 mg/100 ml.

It seems probable that general anaesthesia administered during hypoglycaemia (blood sugar < 40 mg/100 ml)[3] is attended by real risks to the patient of serious permanent brain damage.[4] This case may point the way to a technique in which this risk may be completely avoided and which is at present being investigated in this hospital.—We are, etc.,

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1 Thomas, D. K. M., *British Journal of Anaesthesia*, 1974, **46**, 66.
2 Fry, E. N. S., *Anaesthesia*. In press.
3 *British Medical Journal*, 1971, **3**, 130.
4 Meldrum, B. S., *British Medical Journal*, 1972, **1**, 379.

Treatment of Status Epilepticus with Sodium Valproate

SIR,—We were very interested in the paper by Drs. P. M. Jeavons and Jean E. Clark (15 June, p. 584) describing the encouraging results of treatment with sodium valproate in patients with epilepsy. They did not mention the use of this drug in status epilepticus and we would like to report here the treatment of such a case with sodium valproate.

A girl aged 17 years had a history of grand mal epilepsy since the age of 5 and was accustomed to having one to three fits per day in spite of careful supervision and adjustment of conventional anticonvulsant regimens. On several occasions she had been admitted in status epilepticus. This had usually responded, though sometimes only after 12-24 hours, to intravenous diazepam and/or paradehyde with intramuscular phenytoin and phenobarbitone, together with appropriate supportive therapy as outlined in your leading article (19 February, p. 460). In December 1973, however, status epilepticus failed to respond either to the usual measures or to general anaesthesia with endotracheal intubation and assisted respiration in the intensive care unit. After six days in status she appeared to be dying, but she was then given dexamethasone, whereupon her fits diminished and she recovered during the next three to four days. It was several weeks before she regained her former mental and physical state and she continued to have daily fits in spite of medication (with phenytoin, phenobarbitone, and diazepam).

In June 1974 she was again admitted in status epilepticus. She was treated initially with the usual measures, but as she failed to improve after 12 hours she was given dexamethasone in view of her apparent response on the previous occasion. An infusion of chlormethiazole edisylate (Heminevrin) by a subclavian line was also started and this prevented continuous attacks but had to be maintained at a dose which kept her unconscious except for brief periods during which E.E.G. monitoring showed generalized epileptic activity. After 10 days in this state sodium valproate 100 mg four times a day was started and the dose was increased gradually to 600 mg four times a day over five days, the tablets being dissolved in water and the solution passed into the nasogastric tube. During this period it was possible to stop the chlormethiazole gradually, the number of fits diminished as the dose of sodium valproate was increased, and the E.E.G. improved. She then made a remarkably rapid recovery, having been in status for virtually 15 days. Sodium valproate 600 mg four times a day was continued with a reduced dose of her previous anticonvulsant drugs; she had several days free from fits and was discharged home. On review six weeks later she had remained very much better, having had relatively few attacks and no side effects from the drugs.

We hope that sodium valproate will continue to help in the long-term treatment of this patient's epilepsy, as in many of the cases reported by Drs. Jeavons and Clark. However, we believe that this is the first report of sodium valproate being used in the treatment of status epilepticus. The patient had failed to respond to the supportive measures and drugs generally used together with steroids and chlormethiazole over a period of 10 days, and she recovered only when treated with increasing doses of sodium valproate during the next five days. It therefore seems worth assessing the potential value of this drug now in other cases of status epilepticus.

We thank Drs. T. Healy and A. Hodsman and the nursing staff of the intensive care unit at this hospital for their help with the supportive treatment of our patient, and we are grateful to Reckitt and Colman for the supply of sodium valproate (Epilim).

—We are, etc.,

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MICHAEL ESPIR

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Price of Prostatectomy

SIR,—The article by Mr. S. Argyrou and others (24 August, p. 511) prompts us to write briefly to describe our experience with cryosurgery for prostatic obstruction at Northwick Park Hospital.

Of 16 patients with acute or chronic retention of urine, selected for cryosurgery because severe cardiopulmonary disease made them unsuitable for major surgery or general anaesthesia, 13 left hospital after about 10 days relieved of obstruction and the three failures were subsequently successfully treated by conventional means. So far, no patient has relapsed after periods extending up to 18 months. The operation was carried out under sedation and local anaesthesia with a liquid N_2O-cooled probe designed and made in the bio-engineering division of the Clinical Research Centre. The use of N_2O instead of liquid N_2 makes the instrument and the operation much simpler and safer and avoids the sometimes disastrous complications that have occurred with liquid N_2-cooled probes.[1]

We intend to publish a detailed account of our procedure and results and to carry out a planned clinical trial on a wider range of patients, but meanwhile we would suggest that cryosurgery using N_2O may well be found to offer an acceptable alternative to transurethral resection, with all its advantages and without the disadvantage of requiring expensive equipment and a skilled and experienced operator. An instrument based on our design is being made and sold by Spembly Ltd., Newbury Road, Andover, Hants.—We are, etc.,

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1 *British Medical Journal*, 1971, **2**, 5.

Swimmers' Ears

SIR,—Your leading article (27 July, p. 213) gives an excellent superficial summary but if taken seriously could lead to needless restrictions on those whose recreation is swimming or scuba diving. With the scanning technique of rapid reading the focusing of attention on the last sentence, which states that "when external otitis occurs, diving must be forbidden until the skin has returned to normal," could lead many people being denied their recreation. It is hard to see how this is reconciled with your statement in the first paragraph that a fleeting application of water can hardly cause any harm even when an active eczematous process is present, or the statement by Wright and Alexander,[1] whose work you quote at length. They write: "Swimmers with otitis externa were able to continue swimming with only a limited interruption in their daily routine," and of their divers, all of whom developed external otitis, none ceased their 14 to 16 daily (8-12 hours) excursions into the water and only in a few instances did treatment fail to obtain a sterile ear canal.

For those whose recreation is scuba diving, none expose themselves to the extent that the above divers did, and far from prophylaxis being difficult in divers, as you claim, it is in practice very simple and effective. It is the competitive surface swimmers who suffer more from external otitis than scuba divers[2] because they spend much more time in the water, and in temperate zones the majority of scuba divers wear a hood, which prevents the flow of water in and out of the external ear canal. prophylaxis in the susceptible person begins with the use of 5% acetic acid in rectified spirit[3] after immersion, to dry the skin and maintain its normal acidity and also for its bactericidal and fungicidal properties.