A girl aged 17 years had a history of grand mal epilepsy since the age of 5 and was accustomed to having one to two fits per day in spite of careful supervision and adjustment of conventional anti-convulsant regimens. In several occasions she had been admitted in status epilepticus. This had usually responded, though sometimes only after 12-24 hours, to intramuscularly injected diazepam with intramuscular phenobarbitone, together with appropriate supportive therapy as outlined in your leading article (19 February, p. 460). In December 1973, however, status epilepticus persisted to the use of usual measures or to general anaesthesia with endotracheal intubation and assisted respiration in the intensive care unit. Nine days in status she appeared to be dying, but she was then given dexamethasone, whereupon her fits diminished and she recovered completely during the next five days. It was several weeks before she regained her former mental alertness and has had no further convulsions. She was discharged home 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 chlorothiazide emulsion (Hemide, Astra) over four hours and the day it was started and this prevented continuous attacks but had to be maintained at a dose which kept her conscious for the most part, except for brief periods during which E.E.G. monitoring showed generalized epileptic activity. After 10 days of this regimen a blood sample was taken and the solution passed into the nasogastric tube. During this period she was treated so as to stop the chlorothiazide gradually, the number of fits diminished as the dose of sodium valproate was increased, and the dose was then reduced. She then made a remarkably rapid recovery, having been in status for virtually 15 days. Sodium valproate 600 mg four times a day was started, increased with reduced dose of her previous anticonvulsants; 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 of convulsions but no drug side-effects.

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. Javons 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 chlorothiazide over a period of 10 days. However, 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 (Epileptin).

We are, etc.,

A. R. MANSHIRE

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 about our experience of cryosurgery for prostatic obstruction at Northwick Park Hospital.

Of 16 patients with acute or chronic retention of urine, selected for cryosurgery because of physical inability to manage a catheter and for a major surgical intervention, 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 treatment up to 18 months. The operation was carried out under sedation and local anaesthesia with a liquid N-O-cooled probe designed and made in the biocryosurgery division of the Clinical Research Centre. The use of N,O instead of liquid N makes the instrument and operation much simpler and safer and avoids the sometimes disastrous complications that have occurred with liquid N-cooled probes. Our results have been confirmed by several authors using similar equipment but they do not always suffer from the 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 that "the first principle of all in the application of water can hardly cause any harm even when an active eczematous process is present, or the statement by Wright and Alexander, whose work you quote in full." 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 easier for competitive divers to suffer more from external otitis than scuba divers4 because they spend much more time in water, and in temperate zones the majority of scuba divers wear a hood, which prevents the flow of water and out of the external ear canal. Prophylaxis in the susceptible person begins with the use of 5% acetic acid in rectified spirit5 after immersion, to dry the skin and maintain its normal acidity and also for its bactericidal and fungicidal properties.