ECT with curare

SIR,—In an obituary notice (9 July, p 130) in tribute to the late Dr J A Hobson it is observed that Dr Hobson "was also the first or second psychiatrist to use curare with ECT (he and Dr Aaron Gillis published papers almost simultaneously)."

In fact the paper by Hobson and Prescott¹ appeared in the same issue of the *BMJ* as did the paper by Gillis and Webster.² It should be noted, however, that Dr Hobson's technique included the now well-accepted³ use of thiopentone. The omission of thiopentone incidentally gave us the opportunity to describe² what was perhaps the first case of "awareness during operation."

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¹ Hobson, J A, and Prescott, F, *British Medical Journal*, 1947, 1, 445.

² Gillis, A, and Webster, D D, *British Medical Journal*, 1947, 1, 455.

³ Royal College of Psychiatrists, *British Journal of Psychiatry*, 1977, 131, 261.

Intrathoracic tracheal tumour presenting as asthma

SIR,—The article by Dr D Isaacs and others (19 November, p 1332) highlights a rare problem, that of differentiation of early tumour-related upper airways obstruction from simple asthma. In the patient described the opportunity for early intervention was lost when a diagnosis of asthma was made. Information obtained from measurement of maximum respiratory flow rates and flow volume curves might have been useful had some clinical suspicion or abnormality on spirometry suggested more than simple asthma, prompting further investigation. The article points out that spirometry results showing very low values for FEV₁/FVC with no symptoms at rest should alert the physician.

In addition, however, a distinction between spirometry tracings in upper and lower airways obstruction may sometimes be possible, as is mentioned in certain texts,¹ a uniformly slow expiratory flow with consequent straight-line pattern suggesting the former. Assessment by inspection of the spirometry tracing in addition to measurement of absolute values for FEV₁/FVC still has a place in respiratory investigation.

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¹ Burrows, B, Knudson, R J, and Kettel, L J, *Respiratory Insufficiency*, p 68, Chicago, Year Book Medical Publishers, 1975.

Meningeal leukaemia in lymphoid blast crisis of chronic myeloid leukaemia

SIR,—We greatly appreciated the short report of Dr R K Woodruff and others (19 November, p 1325).

Their experience is further exemplified by a 13-year-old girl who, 14 months after being diagnosed as having Philadelphia-chromosome-positive chronic myeloid leukaemia (CML), presented in acute blastic crisis. The blasts were morphologically and antigenically lymphoid. She responded to vincristine and prednisolone induction and was maintained on 6-mercaptopurine and methotrexate. But persistent severe thrombocytopenia

($\leq 20 \times 10^9/l$ ($\leq 20\,000/mm^3$)) for three months led to modification of chemotherapy and delay in central nervous system (CNS) prophylaxis. She relapsed after four months but was again responsive to prednisolone and vincristine. Her next relapse after a further three months was in the CNS only (vomiting, vertical headache, visual blurring, and blast count of $1.6 \times 10^9/l$ ($1600/mm^3$)) in the cerebrospinal fluid (CSF). Intrathecal methotrexate ($10\,mg/m^2$) given weekly for three doses cleared the CSF of cells, but severe thrombocytopenia ($\leq 10 \times 10^9/l$ ($\leq 10\,000/mm^3$)) unresponsive to platelet infusions led to a conversion to craniospinal irradiation (2400 rads in 15 fractions to the cranium plus 1000 rads in 10 fractions to the spine). Symptomatically she remained well for three more months until a further haematological relapse. At the time of writing she is again in haematological but more significantly CNS relapse.

This case underlines the severe risk of intractable meningeal and intracerebral ($\pm$ spinal) disease in acute lymphoid conversion of CML. However, the marked thrombocytopenia (presumably as a feature of stem-cell disorder) deterred us from lumbar punctures. Hypersplenism and presumed platelet antibody development negated the value of "random-donor" platelets. She also lacked a suitable "close-match" family member from whom to obtain platelets. Yet this thrombocytopenia may have been critical in the development of CNS leukaemia.¹

Craniospinal irradiation was not sufficient to deal with the problem. We would in the future look for any cells reactive to anti-acute lymphoblastic leukaemia serum at presentation of any case of CML. Their presence would indicate a need for early, full CNS prophylaxis involving intrathecal medication and irradiation plus continuing surveillance before megakaryocyte reserves have been destroyed.

We wish to acknowledge Dr George Janossy's kind help with repeated cell-typing of this patient,

O B EDEN

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¹ West, R J, et al, *British Medical Journal* 1972, 3, 311.

Hyponatraemia associated with hydrochlorothiazide treatment

SIR,—I feel it would be beneficial to publicise four cases of hyponatraemia in elderly women associated with diuretic preparations containing hydrochlorothiazide which resulted in confusional states.

Three patients aged 69, 89, and 71 who had been on long-term Moduretic therapy (one tablet a day in the first two cases and two in the morning in the third), either for hypertension or control of congestive cardiac failure, were admitted during the period June–October 1977 to this hospital with confusional states and all were shown to have low plasma sodium concentrations (112–128 mmol/l), which, after stopping the diuretic therapy, rose within a few days to normal levels. The Moduretic preparation contains 50 mg of hydrochlorothiazide per tablet. In two of these three patients there was an accompanying hypokalaemia (2.9–3.0 mmol/l).

The fourth patient, aged 81, had been on long-term Direma therapy, two tablets once a day, each tablet containing 25 mg of hydrochlorothiazide, and was admitted in an incoherent state with general weakness,

equivocal plantar responses, and generalised hyporeflexia. On admission a plasma sodium level of 114 mmol/l was found which rose within three days to 133 mmol/l after cessation of diuretic therapy. The chloride level in this patient showed an accompanying rise from an initially low level. In none of these patients was it necessary to continue with diuretic treatment.

Perhaps it is necessary to consider carefully the choice of diuretic in the older age groups owing to this risk with the thiazide-containing preparation and I would be grateful for anyone else's observations of similar instances.

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Prevention of divers' ear

SIR,—We share the view expressed in your leading article "Divers' ear" (29 October, p 1104) that what may be a mere nuisance for summer swimmers is an incapacitating illness for saturation divers. In pressure chamber experiments with warm, moist atmospheres similar to those you describe¹ we found that otitis regularly interfered with the function of the divers. Pain and other symptoms often led to the administration of sympathomimetic amines, antibiotics, and analgesics including codeine.

This letter is meant to call attention to a highly successful means of prevention.² Divers' ear can be prevented by the use of 2% acetic acid in aluminium acetate solution each morning and evening and following each wet dive. The head is tilted to one side and the canal gently filled with the solution. The solution must remain in the ear canal for five minutes. The head is then tilted to the other side to allow the solution to run out and the procedure is repeated in the other ear. The duration of the solution in the ear canal is critical to the success of the prophylaxis. The dive supervisor should observe and time ear prophylaxis when diving is taking place in waters where external otitis is known to be a problem.

This programme completely eliminated otitis externa which had occurred in 30–70% of divers. Application of other preventive efforts was ineffective. The acetic acid-aluminium acetate prophylaxis has had no adverse effects since its introduction five years ago.

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¹ Raymond, L W, *Lancet*, 1975, 1, 807.
² Thalmann, E D, *A Prophylactic Program for the Prevention of Otitis Externa in Saturation Divers*. Navy Experimental Diving Unit Report 10-74. Washington, DC, USNEDU, 1974.

HLA-B12 as a genetic marker for vesicoureteric reflux?

SIR,—Primary vesicoureteric reflux is a congenital defect due to lateral ureteric ectopia and may be inherited.¹ To date over 80 affected families have been reported,²