volved. Some patients may have spent time in an intensive treatment unit or in a medical ward before transfer, but 75% are discharged within 48 h of admission to the unit, approximately 15% to psychiatric hospitals.

Outpatient follow-up is now carried out in the unit, which has reduced the default rate to less than 5%, and over the past three years the relapse rate, defined as repeat of self-poisoning within 12 months, has fallen to less than 10%.

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¹ Barraclough, B, *et al*, *British Journal of Psychiatry*, 1974, **125**, 355.

Treatment of chlonorchiasis

SIR,—Your timely leading article on liver fluke infection (29 April, p 1091) draws attention to the problem of infection with *Clonorchis sinensis* in countries like Britain and Canada, which have appreciable numbers of Chinese immigrants, but makes no reference to a recent investigation in New York City in which a single stool examination from each of 150 Chinese-born residents showed that 26% were infected with this liver fluke.¹ Subsequent correspondence with the senior author of this report has revealed that no effective treatment is available for these patients in the USA.

In a letter in 1975² I reported that excellent results had been obtained at the Hospital for Tropical Diseases in London using a supply of hexachloroparaxyol (HPX) which I had obtained from a colleague in China (where the drug is freely available) and at the same time I drew attention to the fact that this drug was unobtainable in the West. Since then appeals to Hoechst Pharmaceuticals to remarket their product (Hetol) have failed and I have been equally unsuccessful in persuading a leading British pharmaceutical company to approve the manufacture of HPX, even though the company's research department found the drug to be non-toxic to laboratory animals. Since the publication of that letter I have corresponded with physicians in Britain, Canada, the United States, Switzerland, West Germany, and even Hong Kong, all complaining of inability of any alternative drug of value in treating clonorchiasis. At this very moment I know of a proved case in London urgently in need of treatment. Is there no pharmaceutical company in Britain willing to meet this challenge and is the NHS prepared to let this problem go by default?

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¹ Kammerer, W S, *et al*, *Tropical Doctor*, 1977, **7**, 105.
² Jopling, W H, *British Medical Journal*, 1975, **3**, 767.

Prognostic value of oculovestibular reflex

SIR,—In their interesting paper Dr M A Hanid and others (22 April, p 1029) emphasise the importance of observing oculovestibular responses (OVR) in patients in hepatic coma. They conclude that "the prognostic value of the OVR in fulminant hepatic failure was absolute." Our own coma study¹⁻³ to which they refer has now been extended to include 500 patients and our experience of the behaviour of OVRs in hepatic coma differs somewhat from that of Dr Hanid and his colleagues. Of 51 patients in hepatic coma, 19

fulfilled the criteria of fulminant hepatic coma. Of the whole group only four patients had absent OVRs at the time of initial assessment and one of these recovered from coma.

There are a number of possible reasons for our differing results. We accept an OVR as negative only if there is no response after injection of 100 ml of ice-cold water into the auditory canal. Secondly, the effect of drugs other than paralytic agents must be considered in evaluation of the OVR, particularly in patients with hepatic disease, in whom the metabolism of drugs may be retarded. However, the main discrepancy between our results must depend on our differing interpretations of the word prognosis.

Our observations on OVRs were made with a view to evaluating this sign as a predictor of final outcome. Dr Hanid and his colleagues appear to have monitored the OVR in the course of the illness and they observed its disappearance as deterioration progressed. All dying patients at some stage will lose the OVR and we would submit that as an early predictor of outcome in hepatic coma these responses are of less value than they imply.

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¹ Caronna, J J, *et al*, *Transactions of the American Neurological Association*, 1975, **100**, 25.
² Levy, D, *et al*, In preparation.
³ Bates, D, *et al*, *Annals of Neurology*, 1977, **2**, 211.

SIR,—Dr M A Hanid and others in their recent article (22 April, p 1029) stress what they call the absolute prognostic value of the oculovestibular reflex in fulminant hepatic failure. By this they imply that the absence of any movement of the eyes in response to irrigation of either ear with iced water implies an inevitably fatal outcome. They go on to generalise their argument by quoting a similar totally adverse prognostic implication in head injuries¹ and in medical (including other metabolic) causes of coma.² In a recent study³ of coma in childhood Seshia *et al* came to an identical conclusion: of 75 children, all four who had absent ocular motility using ice caloric testing died irrespective of the presence of other signs of brainstem function.

Any exceptions to "absolute" rules must be recognised, especially when the question is one of life or death. We therefore briefly report a single case¹ of very severe diabetic coma in which total neurological recovery followed earlier complete absence of oculovestibular reflexes.

A 12½-year-old girl with a seven-day history of thirst was found unconscious in bed on the morning of 2 November 1975 and admitted to the intensive therapy unit of this hospital. She was in shock with unrecordable blood pressure, temperature 39.0°C, in grade IV coma with no responses except sluggish pupil light reactions, but had shallow respirations with a fruity odour. After rapid infusion of isotonic saline the peripheral circulation improved, blood pressure came within normal limits, and core temperature fell to normal. At this stage the diagnosis of hyperosmolar diabetic coma had been confirmed (blood glucose 180 µmol/l (3240 mg/100 ml), plasma osmolality 430 mmol/kg, pH 7.02) and the neurological examination was repeated in full.⁵ All neurological reactivity was absent apart from some pupil light reaction, intact oculocardiac reflex and spontaneous regular respiration. In particular, irrigation of either ear

with large quantities of iced water induced no ocular movements and doll's eye responses were absent. On this basis she was regarded as having a pure metabolic coma,⁶ and treated with 6 U of soluble insulin intravenously and intramuscularly and then 4 U intramuscularly hourly. Within 3 h she gained some tonic ocular deviation on ice caloric testing, and she was obeying commands by 48 h. Extensive dependent ischaemic skin lesions which may have reflected her extreme hyperglycaemia¹ healed with scarring, but she made a complete neurological and psychological recovery (Wechsler testing on 23 February 1976: verbal scale 118, performance 111, full scale IQ 115.)

Your readers will be well aware that poisoning can abolish all neurological reactivity. It is vital to remember that severe brainstem dysfunction may also occur in metabolic coma and that it should be entirely reversible if structural damage from brain swelling can be prevented by meticulous therapy.

It is a pleasure to thank Dr Oman Craig for permission to report this case.

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¹ Poulsen, J, and Zilstorff, K, *Acta Neurologica Scandinavica*, 1972, **48**, 282.
² Caronna, J J, *et al*, *Transactions of the American Neurological Association*, 1975, **100**, 25.
³ Seshia, S S, Seshia, M M K, and Sachdeva, R K, *Developmental Medicine and Child Neurology*, 1977, **19**, 614.
⁴ Craig, O, *Childhood Diabetes and Its Management*, p 221 and fig 32. London, Butterworth, 1977.
⁵ Plum, F, and Posner, J B, *The Diagnosis of Stupor and Coma*, 2nd edn. Philadelphia, Davis.

Confidentiality of medical records

SIR,—In their paper on self-poisoning in Nottingham (22 April, p 1032) Dr D R Blake and Professor J R A Mitchell state that "the casualty department staff keep their records on a triplicate form, the third copy of which is processed by the Trent Regional Health Authority in Sheffield for research purposes."

This quotation prompts the question whether the second and third copies include the patient's names and addresses. If they do, other questions arise: (1) Who keeps the second copies? (2) Are the patients warned (for instance, by a large notice in the casualty department) that information about their visit to that department will be sent to the regional health authority? (3) Does this procedure happen in other regions? (4) If so, should I, as an employee of the North-west Thames Region, make sure that I take my overdose in another region, lest my employer regard me as a mental health risk? And since three of the Thames regions have their offices in the same building, will I even be safe in, for example, the North-east Thames Region?

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Price of survival in childhood leukaemia

SIR,—We were interested in your leading article (11 February, p 321) and the letter from Dr P G F Swift and others (15 April, p 986) on the price of survival in childhood leukaemia. The initial endocrine study by our group¹ showed impaired growth hormone (GH) responses to hypoglycaemia in leukaemic children with acute lymphoblastic leukaemia (ALL) several years after they had received cranial irradiation (DXT) with a dose of 2500