patients with barbiturate poisoning. They found that in both grades III and IV coma overdose with the two groups of drugs caused an equal degree of hypoxia, mainly through ventilation-perfusion imbalance. In a second study, of 113 deaths from tricyclic antidepressants occurring in hospital in 1976,² only 42 patients were described as breathing normally and less than half of those with obvious respiratory depression and hypoxia were artificially ventilated. Although the majority of patients in this latter series were already severely affected on arrival in hospital, it seems likely that the mortality could have been reduced by more aggressive treatment of hypoxia (whether caused by respiratory depression, ventilation-perfusion imbalance, or repeated convulsions) and by the avoidance of negatively inotropic drugs, which would be expected to aggravate rather than improve tricyclic-antidepressant-induced cardiotoxicity.

The child described by Dr Cronin and his colleagues was given a saline emetic, the dangers of which have previously been discussed in your columns.³ I assume that this patient was not in fact hypernatraemic on admission to hospital.

Unlike the patient described in this case report, the majority of children admitted to hospital with tricyclic antidepressant poisoning have taken medication prescribed for other members of their family. In a study of 489 patients with tricyclic antidepressant poisoning, which included 92 children under 5, it was found that only 12.5% of those in this age group had taken their own medication, compared with 50% in the age range 6-15. Packing antidepressants in child-proof containers may well reduce poisoning in toddlers. However, there are other equally dangerous medications prescribed for children and it is perhaps unjustified to single out the tricyclic compounds as requiring warning labels describing their "lethal properties." Indeed, such warnings might encourage some people to take them.

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¹ Sutherland, G R, Park, J, and Proudfoot, A T, *Clinical Toxicology*, 1977, **11**, 403.

² Crome, P, and Newman, B, *Journal of the Royal Society of Medicine*, in press.

³ *British Medical Journal*, 1977, **2**, 977.

⁴ Crome, P, and Newman, B, *Postgraduate Medical Journal*, in press.

"Therapeutic" salicylate poisoning in children

SIR,—The dangers of imipramine poisoning in children and the role of doctors in prevention has been rightly stressed (17 March, pp 705 and 722). The fall in the number of deaths in children due to salicylate poisoning was noted. Death and severe illness due to salicylates still occur, however, and as this drug is available without prescription the following cases are of interest.

Case 1—This 5-month-old child developed symptoms of a viral upper respiratory infection seven days prior to admission and was given an unknown number of junior aspirin, but none in the 24 hours prior to admission. He developed tachypnoea the day before admission and a few hours before admission was seen at a nearby casualty department but not admitted. On admission to our unit he was febrile (temperature 39.9°C) and his heart rate was 200/min and respiratory rate 60/min. The chest was clear to auscultation. Initial

investigations showed a marked metabolic acidemia (pH 7.18, PaCO_2 3.59 kPa (27 mm Hg), PaO_2 7.98 kPa (60 mm Hg), bicarbonate 10 mmol (mEq)/l, and base excess -17). The serum salicylate was 3.3 mmol/l (45.5 mg/dl). He was treated with intravenous fluids and bicarbonate and made a full recovery.

Case 2—This 11-month-old boy received 16 junior aspirin tablets (1200 mg) in the 24 hours prior to admission because of irritability attributed to teething. He became tachypnoeic 12 hours prior to admission. On admission he was severely dehydrated; his respiratory rate was 54/min and heart rate 160/min. His chest was clear to auscultation. Blood gases were: pH 7.11, PaCO_2 2.79 kPa (21 mm Hg), PaO_2 7.32 kPa (55 mm Hg), bicarbonate 7 mmol(mEq)/l, and base excess -22. Serum salicylate was 5.46 mmol/l (75.4 mg/dl). He was treated with intravenous fluids and bicarbonate and he also made a full recovery. He had marked haematuria and a prolonged prothrombin time (patient 53.2 seconds, control 13.5 seconds).

Both children had near lethal poisoning.¹ The dangers of accidental therapeutic poisoning with aspirin have been emphasised,² but there were delays in recognising the correct diagnosis in case 1. Both children received the drug for trivial reasons, and in both cases the mothers had bought and used the drug as a harmless symptomatic measure.

Salicylates are freely available in pharmacies and supermarkets. Childproof wrapping will not prevent such poisoning as is reported here. Packets of junior aspirin contain advice to parents to use aspirin in children less than 1 year old on medical advice. Doses are given for other children. There is no mention of the hazards. Aspirin has a limited place in paediatric practice and has no place in trivial illness.³ If this potentially dangerous drug is to remain freely available to the public, then clear warning of its dangers should be printed on the packets. It would be preferable to limit its availability.

A copy of this letter has been sent to the Commission for Review of Medicines.

Dr W R McAinsh kindly allowed me to publish details of case 1. Mrs Muriel Agnew gave helpful information and stimulated reporting of these cases.

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¹ Done, A K, *Pediatrics*, 1960, **26**, 800.

² Craig, J O, Ferguson, I C, and Syme, J, *British Medical Journal*, 1966, **1**, 757.

³ Hutchison, J H, *Practical Paediatric Problems*, p 619. London, Lloyd-Luke (Medical Books) Ltd, 1975.

Chronic subdural haematoma

SIR,—In your leading article (17 February, p 433) entitled "Chronic subdural haematoma" you state that radioisotope imaging cannot be relied on as a diagnostic procedure—a view not taken by most neurosurgeons.

Information on the capabilities of radioisotope imaging compared with computerised axial tomography (CAT) in the diagnosis of chronic subdural haematoma is available in several reports.¹⁻³ If these studies are pooled the following results are obtained: in 35 patients in whom the diagnosis was made at operation, CAT had a preoperative diagnostic accuracy of 94% and radioisotope imaging of 91%.

Thus there is no doubt in these studies that the two modalities are comparable in accuracy. But if we take in to consideration the facts that with CAT the patient needs to be more co-

operative, receives a larger dose of radiation, and is at greater risk from injected contrast material, then radioisotope imaging becomes a much more attractive diagnostic modality.

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¹ Alavi, A, et al, *Journal of Nuclear Medicine*, 1977, **18**, 64.

² Alderson, P O, et al, *Neurology (Minneapolis)*, 1976, **26**, 803.

³ Christie, J H, et al, *American Journal of Roentgenology*, 1976, **127**, 171.

* * Dr Helliwell is quite correct in pointing out the value of radioisotope imaging in the diagnosis of chronic subdural haematoma and perhaps we should have indicated that it has a value. Although he points to the comparability of results with CAT scanning, in some of the papers he refers to there is no comment about the age of the lesions and the reliability of the investigations, whereas an earlier paper¹ drew attention to the difficulty in diagnosis by isotope scanning in the haematomas of a younger age. Certainly if isotope scanning is available it is a reasonable investigation and may lower the chances of overlooking this remediable condition but, if the presence of midline shift demonstrated by CT scan is taken into account, isotope scanning cannot be relied on in the same way to exclude the diagnosis. Unfortunately the appearances on isotope scanning after infarction may mimic closely those of chronic subdural haematomas, particularly when the calcified pineal is not visible on the skull radiographs. —Ed, *BMJ*.

¹ Cowan, R J, et al, *Journal of Neurosurgery*, 1970, **32**, 30.

Red eyes and hypercalcaemia

SIR,—I read with interest the report of the demonstration of two cases at the Royal College of Physicians of London (17 March, p 727). The second case, that of a 66-year-old white woman admitted with visual deterioration, aches and pains in the legs, and a left homonymous hemianopia, I found particularly fascinating. It was stated in the history that she had had excessive thirst and constipation for some time, and nine weeks before admission had developed red, watering eyes. She was subsequently found to be hypercalcaemic, and the red eyes and the hypercalcaemia were not explained by the postmortem diagnosis of Creutzfeldt-Jakob disease.

Dr Michael Smith and myself reported a case in February 1976 of recurring red eyes due to a seasonal hypercalcaemia.¹ Red eyes associated with hypercalcaemia are almost certainly due to metastatic crystallisation of calcium and phosphate and hydroxyapatite in the conjunctiva.² I feel the "conjunctivitis" referred to by Professor W B Matthews in the discussion of the case was due to the hypercalcaemia and not in fact to Creutzfeldt-Jakob disease or an infective process.

It was not stated how long the symptoms of thirst and constipation had been present, but it would appear that the lady had been hypercalcaemic for some time. Was the rise in serum calcium from 3.1 mmol/l (12.5 mg/100 ml) to 4.6 mmol/l (18.3 mg/100 ml) associated with increased exposure to ultraviolet light? Is it possible that the histological findings at necropsy were the effect of hypercalcaemia on neuronal tissue? It would certainly seem