downward herniation of the cerebellar tonsils.⁵ The imaging studies of our patient disclosed the laterocerebellar subarachnoid cyst, which may have contributed to forming the tonsillar descent.

The outcomes of the surgery, usually craniectomy with upper cervical laminectomy, have been gratifying, especially provided no other signs of brain stem compromise were present.³ Thus, early recognition and intervention for this deformity are crucial in ensuring a favourable neurological outcome.

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MATTERS ARISING

Head injury

We read with dismay the statement by Teasdale¹ in his review of head injury that "CT should be performed if a skull fracture is present". This is supported by the un referenced statement that "The finding of a skull fracture raises the risk of intracranial injury by more than 200-fold". In a comprehensive study of the role of skull radiography in head injury by Masters *et al*² 71 of 21 300 (0.3%) patients with normal skull radiographs had intracranial injury compared with 67 of 758 (8.8%) with skull fractures. This implies that the presence of a skull fracture raises the risk of intracranial injury by a factor of 26, but even then over 90% of those with a skull fracture after head injury will have a normal CT. To recommend that all these patients have CT will not only pro-

duce a low yield of positive results but also represents a substantial waste of resources. Additionally, in absolute terms more cases of intracranial injury occur in the group without skull fracture. Clearly, using the skull radiograph as an indication for CT is misguided.

When should CT be performed? Masters *et al*² reviewed 7032 patients with head injury categorised by the history and physical findings as being at low, medium, or high risk of intracranial injury. There were no cases of intracranial injury in the 5252 low risk patients, though 12 had skull fractures. All the 36 cases of intracranial injury occurred in the 1780 moderate and high risk patients, of which only 20 had skull fractures. It follows that the clinical findings and neurological state are the deciding factors in proceeding to CT. The principal indications for CT are persistent and substantial impairment of consciousness (Glasgow coma scale score of 12 or less) or focal neurological deficit.³ The skull radiograph should not be a consideration in this decision. Such an approach is also in accordance with the guidelines of the Royal College of Radiologists.⁴ It should be noted that these arguments relate to the indications for CT after head injury. They also relate to the indications for skull radiography after head injury, providing compelling evidence that skull radiography should be reserved for suspected penetrating injury or depressed fracture, otherwise if the clinical state merits imaging then the appropriate test is CT. However, this remains controversial and is tangential to our primary concern.

The recommendation by Teasdale that all patients with skull fracture should have CT is erroneous and is an example of the common trap of treating the radiograph and not the patient.

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Teasdale replies:

I am surprised to find Coakley, Owen, and Finlay resurrecting a debate of the past decade and apparently unaware of data published in the *British Medical Journal* in 1990.¹ This is in the references in the review, although not specifically quoted at this

point. In brief, this study—which was distinctive in being comprehensive—showed that in the adult patient who arrives at hospital conscious after a head injury—the group in question—the finding of a skull fracture increases the risk of an intracranial haematoma requiring operation by 230-fold in those without a history of amnesia and by 387-fold in patients recovered from post-traumatic amnesia. Also, the problems of extrapolation from the study of Masters *et al*² were pointed out soon after its publication.^{3,4}

Clinicians responsible for the care of patients with head injury certainly do not "treat" radiographs but use them selectively, in a risk based approach to management that minimises potentially avoidable mortality and morbidity.

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NOTICE

Announcement from the British Neuropsychiatry Association: 1996 summer meeting

The 1996 Summer meeting will be held on 14-16 July at Robinson College, Cambridge. It will include topics on neurodevelopment, language, and the presentation of short scientific papers and single case videos by members. The Association's AGM will be held on 16 July.

For further details of these meetings please contact: Sue Garratt, Administrative Assistant, BNPA, 17 Clocktower Mews, London N1 7BB. Telephone/Fax: 0171 226 5949.

For details of membership of the BNPA, which is open to medical practitioners in psychiatry, neurology, and related clinical neurosciences, please contact: Dr Jonathan Bird, Secretary BNPA, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol, BS16 1QT. Telephone: 01179 701212 ext 2925/2929 or Sue Garratt at the address given above.