

produced a fall towards the side of the lesion. Although the inferior cerebellar peduncle involves the vestibulocerebellar pathways, there was no spontaneous nystagmus and a caloric test gave normal nystagmus responses,⁴ as in our patient.

Thus experimental data support the hypothesis that our patient's lateropulsion originated in a lesion of the inferior and possibly superior cerebellar peduncles, in accordance with the topography of the lesion on brain MRI.

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Progressive dysphagia due to adult Chiari I malformation mimicking amyotrophic lateral sclerosis

Chiari I malformation, characterised by caudal descent of the cerebellar tonsils, has been shown to cause progressive dysphagia, usually associated with other apparent signs and symptoms of dysfunction of lower cranial nerves, medulla, and cerebellum. Without the associated hindbrain malfunctions, however, this deformity still needs to be listed as a possible cause of neurogenic dysphagia in consideration of its readiness to be diagnosed on sagittal views of MRI and its excellent reversibility on decompression surgery. There are two reports^{1,2} of dysphagia as a sole manifestation of adult Chiari I malformation, in both of which the diagnosis was long delayed. Presented here is a patient diagnosed as possibly having amyotrophic lateral sclerosis, who was subsequently found to have Chiari I malformation as a cause of progressive dysphagia.

A 63 year old woman had an unremarkable medical history until 1990 when she started having some difficulties in swallowing liquid, but was able to eat solid food. In 1991 she developed aspiration pneumonia, which was confirmed by an otherwise unremarkable barium swallow test. Over the next three years she was admitted to hospital four times for recurrent aspiration with increasing dysphagia. In October 1994 she was

referred by a neurologist to our hospital for terminal care of her "amyotrophic lateral sclerosis." By the time of admission, she had nasal regurgitation on every liquid intake and became unable to swallow even solid foods. She required a nasogastric tube for feeding.

Physical examination showed an emaciated woman with a body weight of 30.5 kg and height of 142 cm. No physical anomalies such as a short neck and low hairline were noted. Neck flexion was rather restricted and neck extension, although full, was associated with a dull pain in the occipital area. No papilloedema was noted. The gag reflex was bilaterally absent with moderate palatal hypoaesthesia. Her voice was slightly nasal with hypomobility of the soft palate, but neither hoarseness nor dysarthria was noted. Her tongue did not show atrophy or fasciculation. External ocular movements were full and there was no nystagmus. She had diffuse muscle weakness and general hyperreflexia with indifferent plantar response. There was slight impairment of coordination in the upper and lower limbs. She had mild glove and stocking type dysaesthesia with slightly decreased sensation on all modalities. Romberg's test was equivocal. She had slight difficulties in walking straight and standing on one foot.

Investigations showed an unremarkable urinalysis, complete blood count, serum chemistry, and arterial blood gas analysis. Spirometry showed a percentage of the predicted value of vital capacity of 57% and a forced expiratory volume in one second of 73%. Fiberoptic laryngoscopy was essentially unremarkable except for a somewhat weak laryngeal elevation. Examination of CSF was normal with no evidence of a spinal block. Electromyography and brainstem auditory evoked responses were unremarkable. Somatosensory evoked potentials suggested bilateral peripheral neuropathy. Motor and sensory nerve conduction studies showed slightly decreased velocity in all limbs. Skull radiographs were unremarkable, without basilar impression or platybasia. The coronal views of MRI were notable for a laterocerebellar subarachnoid cyst in the left posterior fossa mildly compressing the cerebellum (fig 1), and the sagittal views showed extension of cerebellar tonsils below the foramen magnum to the C2 level (fig 2). No hydrocephalus or spinal cavity was noted in the imaging studies.

She underwent suboccipital craniectomy with C1 decompression laminectomy in January 1995. One month later, she reported nearly complete resolution of dysphagia, nasal voice, and dysaesthesia of the hands. The gag reflex became weakly positive, the soft palate moved upwards well, and the palatal sensation returned. Unchanged were general hyperreflexia with indifferent plantar response and slightly impaired coordination of the extremities.

This woman was first diagnosed as having amyotrophic lateral sclerosis based on the findings of progressing severe bulbar palsy, general hyperreflexia, and diffuse muscle weakness that turned out to have resulted from malnutrition. Other findings such as impairment of coordination and instability of walking were so subtle that they were attributed to muscle weakness. It was unusual for amyotrophic lateral sclerosis, however, that the patient had no apparent sign of dysfunctions of the lower cranial nerves, especially of the tongue, despite the presence of striking dysphagia. This led us

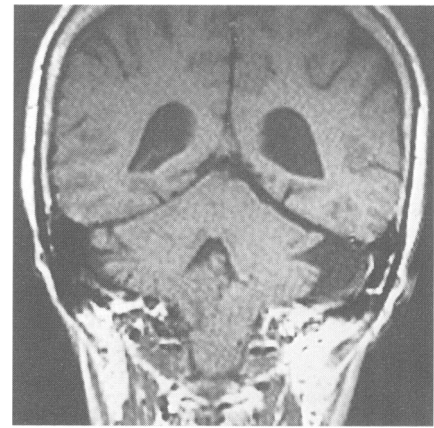


Figure 1 Coronal section through the fourth ventricle: MR T1 weighted imaging (TR = 500 ms, TE = 15 ms) shows a left laterocerebellar subarachnoid cyst mildly compressing the cerebellar hemisphere.



Figure 2 Midline sagittal MR T1 weighted imaging (TR = 500 ms, TE = 15 ms) shows descent of the cerebellar tonsils to the C2 vertebral level.

to investigate the patient with MRI, which showed caudal displacement of the cerebellum.

The suggested mechanisms of dysphagia in Chiari I malformation have been stretch injury to the lower cranial nerves caused by caudal displacement of the medulla, or dysfunction of the brain stem, especially the swallowing centre itself, from compression. Pollack *et al* examined patients with dysphagia due to Chiari I malformation by cineoesophagograms, and found widespread impairment of all phases of swallowing, which was consistent with lesions involving the medullary swallowing centre.³ The uncommon manifestation of our patient—namely, prominent dysphagia without apparent involvements of other lower cranial nerves—may also be explained by rather selective impairment of a swallowing centre in the medulla.

The aetiology of adult Chiari I malformation remains obscure. Although traditionally viewed as a congenital anomaly, there is much evidence that this malformation may be an acquired deformity. Cerebellar descent occurred after repeated lumbar punctures or spinal subarachnoid shunting, which supported the hypothesis that the CSF pressure difference between the spinal and cranial compartments causes tonsillar herniation.⁴ Others suggested that a mismatch between the volume of the posterior fossa and its tissue contents may produce

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.