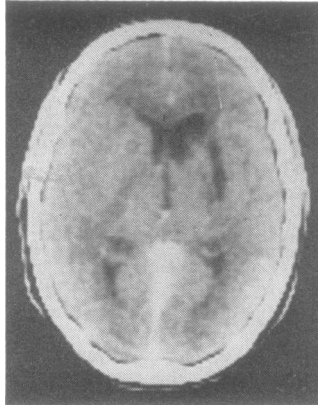


Hemi-dystonia due to focal basal ganglia lesion after head injury and improved by stereotaxic thalamotomy

Sir: A case of post-traumatic hemidystonia was recently reported in your journal by Brett *et al.*,¹ and the points of interest were the delayed onset of dystonia and the finding of a lesion in the basal ganglia on CT scan.

We wish to report a case of post-traumatic hemidystonia, occurring the day following a head injury, in whom a similar lesion was seen on the CT scan, and the patient's involuntary movements were improved following thalamotomy. The patient, a 24-year-old Iraqi, gave no family history. At the age of eight years, he was in a road traffic accident, and may have lost consciousness for a very short period; he suffered lacerations around the mouth. The following day the involuntary movements of the left side of the body were first noted, and these became worse so that by the time he was adolescent, he required two walking sticks because of sudden myoclonic jerks of the left leg, and when sitting down he had to place his left arm behind his back and lean on it to stop it shooting up into the air. He was employed as a clerk. On examination, he was of average intelligence and showed dystonic posturing in his left upper limb, and between the abnormal movements there was quite good grip, although not as strong as the right. The more flagrant abnormal movements, however, came from the shoulder joint and were a mixture of torsion dystonia and hemiballismus. There was mild left sided oro-facial dystonia, characterised by grimacing movements on that side. When walking, myoclonic jerks would cause the left leg to shoot upwards and throw him off balance unless he used a walking stick. The foot was usually held everted owing to distal dystonia and although there was some shortening of the left peronei, the foot could be brought to a neutral position. Hyperkeratosis had developed under the medial part of the left sole. There was dystonic posturing in the toes. A CT scan (fig) showed a slight dilatation of the anterior half of the right lateral ventricle, and there was a linear lucency extending from the head of the right caudate nucleus, crossing the anterior limb of the internal capsule into the lentiform nucleus; in the case of Brett *et al* the lucent area was in the putamen, and there was no hemiatrophy.



CT Scan of the patient

A stereotaxic thalamotomy was performed, using micro-electrode recording for location of the sensory relay nuclei. A 10 mm lesion was made to their anterior limit at 13 mm from the midline, and a second lesion was placed 6 mm more anteriorly and 3 mm under the first. There was an immediate improvement in the involuntary movements so that the flinging movements from the left shoulder were enormously reduced in amplitude and the patient could now walk without a stick and no longer needed to keep the left arm behind his back when sitting. The dystonic posturing of the left hand was also reduced and he could now use the hand more in everyday activities.

The mechanism of production of the basal ganglion abnormality in these two cases is speculative. Their initial injury was not severe, but both occurred as a result of road traffic accidents, and it is possible that they sustained concomitant neck injury. We are reporting elsewhere an instance of tremor developing after head injury with a similar low attenuation lesion in the cerebral white matter attributable, in that case, to embolism from an angiographically demonstrated traumatic carotid dissection.

Maki *et al*² report four similar infarcts on CT scans in children who developed hemiparesis or hemidystonia within minutes or hours of a minor head injury. These authors suggest that the mechanism is rotational, from neck twisting, when the perforating branches (lenticulostriate) of one middle cerebral artery have been dislocated, on the side opposite to which the head or face has been struck. It is possible that more cases of

hemidystonia due to similar lesions, will now be found by CT scan. In view of the resistance of the condition to medication, thalamotomy, even in later childhood, should perhaps be considered in order to permit greater integration of the affected limbs into daily living.

We wish to thank Dr Peter Harvey for referring the patient.

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References

- ¹ Brett EM, Sheehy MP, Marsden CD, *Neurol Neurosurg Psychiatry*, 1981;44: 8461.
- ² Maki Y, Akimoto H, Enomoto T. Injuries of basal ganglia following trauma in children. *Child's Brain*, 1980;7:113-23.

CORRECTION

Delayed auditory brainstem responses in diabetes mellitus by MW Donald, CE Bird, JS Lawson, FJJ Letemendia, TN Monga, DHC Surridge, P Varette-Cerre, DL Williams, DML Williams, and DL Wilson.

It is regretted that the authors' proof corrections did not appear in the published version of this paper (*Journal of Neurology, Neurosurgery and Psychiatry* 1981;44:641-4). On page 642, second paragraph, the sixth sentence should read "Amplifier bandwidth was 150-3000 Hz."

In the table, non-diabetic, right ear latency for wave V should read "5.61 ± 0.49" as in the text.

The first author in reference 6 should read "Letemendia FJJ".