Matters arising

Contralateral gaze deviation after frontal lobe haemorrhage

Sir: Dr Sharpe and the colleagues, in their short report (Sharpe JA, Bondar RL, Fletcher WA. Contralateral gaze deviation after frontal lobe haemorrhage. J Neurol Neurosurg Psychiatry 1985;48:86-88.), discussed the mechanism of contralateral gaze deviation. The discussion was mainly based on the feature observed on CT scan, which was postulated to be responsible for the patient's contralateral gaze deviation.

Should the intracerebral haematoma be caused by hypertensive disease or a rupture of an arteriovenous malformation, it will yield localising value, and the discussion might be meaningful. In cases with head trauma, however, the neurological presentations could be resulted from various regions of the damaged brain, mostly being inappropriate for the assessment of precise localisation; even using CT, brain damaged secondary to lesions isodense to brain parenchyma cannot be identified. Moreover, judging from being stuporous in the acute stage, it is not plausible that this patient suffered only from the isolated haematoma in the frontal lobe; the coexistence of other brain damage is highly possible. From these reasons, it appears inappropriate to discuss the mechanism of contralateral gaze deviation on the basis of CT scan findings in this head injured patient.

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References

Sharpe et al reply

Of course focal lesions identified by CT scanning do not guarantee absence of other lesions caused by head injury. As pointed out in our report¹ unrecognised coup or contracoup injury to the ipsilateral hemisphere might account for the gaze deviation despite the absence of corresponding clinical EEG or CT signs. Although the initial stupor in our patient was a manifestation of a diffuse concussive injury, it lasted two days while the ocular deviation persisted for a week and the patient was mobile. Stupor also accompanies contralateral gaze deviation associated with hypertensive cerebral haemorrhage; pressure with distortion of remote brain regions may contribute to the "wrong side" deviation just as it causes the stupor, even though the anatomical extent of haemorrhage does not extend below the rostral midbrain.² In addition, remote effects of acute cerebral lesions on brainstem ocular motor function probably contribute to gaze disorders in a manner analogous to the diachisis of spinal shock.³ Our observation that frontal lobe haemorrhage may be associated with contralateral gaze deviation is supported by a reported case of hypertensive haemorrhage into the parietal and frontal lobes, above the thalamus.⁴ The smooth eye movement imbalance that we identified contralateral to a cerebral haematoma is a proposed mechanism of the "wrong side" gaze deviation.⁵ Although localisation may be less reliable after head injury, we consider that quantitative assessment of ocular motor behaviour is appropriate for assessing mechanisms of gaze deviation after head injury, pending such quantitative study in patients with contralateral gaze deviation associated with hypertensive cerebral haemorrhage.

Notice

The third International Spinal Cord Monitoring Symposium will be held 21-25 October, 1986 in Annapolis, Maryland, USA. Information may be obtained from Thomas B. Ducker, MD, 100 Cathedral Street, Annapolis, Maryland 21401, USA.

Correction. In the paper "Simulated paraplegia: an occasional problem for the neurosurgeon" (J Neurol Neurosurg Psychiatry 1985;48:826–831) thanks were expressed to the late Dr Sheilagh Davies. Dr Davies is in active medical practice and the word "late" should have been applied to Mr Bernard Fairbairn who died in 1985.
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*J Neurol Neurosurg Psychiatry* 1985 48: 1310
doi: 10.1136/jnnp.48.12.1310

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