

Ocular skew deviation in hepatic coma
 SIR,—Ocular skew deviation has been reported in many types of structural defects of the posterior fossa. A recent extensive review of patients with skew deviation states that no cases have been seen in association with metabolic coma or stupor.¹ Recently a patient was seen in hepatic coma, who had concomitant ocular skew deviation which resolved with clearing of the encephalopathy.

A 68-year-old man with a long history of ethanol abuse was evaluated for progressive obtundation. On initial examination, the patient was unresponsive to verbal command but responded appropriately to painful stimulation of all extremities. Neurological examination otherwise was unremarkable. Laboratory studies on admission showed a slight elevation of glutamine in the CSF. An electroencephalogram showed changes compatible with a severe metabolic encephalopathy. On the third hospital day, the patient was found to be entirely unresponsive to verbal or painful stimuli. The right eye was maintained in primary position but the left eye was moderately hypertrophic as compared to the right. Oculocephalic and oculovestibular reflexes were intact both horizontally and vertically. Pupils

were 3 mm diameter, equal, round, and reactive. The rest of the neurological examination remained unremarkable. The patient was treated with lactulose and sedative medication was discontinued. Within two days, he responded to pain and the ocular skew deviation was no longer present. Five days after initiation of therapy, the patient was alert and responded to verbal commands with full ocular movements. At that time, he specifically denied any prior history of vertical strabismus or symptomatology suggestive of a posterior circulation cerebral vascular accident.

Transient ocular motor disturbances have been observed in patients with hepatic encephalopathy (usually associated with structural lesion of the brainstem) such as: ocular bobbing,² dysconjugate gaze³ and absent horizontal responsiveness to oculovestibular testing.⁴ This appears to be the first report of a patient with ocular skew deviation in hepatic coma. Transient focal neurological deficits such as those described above and others are an infrequent but well recognised complications of metabolic disorders. They are usually seen with structural lesions, are presumed to occur on the basis of selective vulnerability to metabolic insult of regions of

the central nervous system previously overtly or sub-clinically compromised. Skew deviation is thought to result from acute damage to areas in the brainstem responsible for vertical control of the oculomotor system. The widespread vestibular ocular interconnections could provide a neuroanatomic substrate for the poorly localisable system of vertical ocular motor control and may well have been selectively compromised in the patient described.

References

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