Letters

Downbeat nystagmus—a false localising sign due to communicating hydrocephalus

SIR,—Downbeat nystagmus is an uncommon form of vertical nystagmus in which the rapid phase is in a downward direction. It is present when looking straight ahead and increased by looking down. It is usually described as giving presumptive evidence for a lesion in the lower brain stem, upper cervical cord or cerebellum.¹ Amongst the clinical conditions described as responsible are platybasia with Arnold Chiari malformation, Klippel-Feil syndrome, deformities of the cervical spine at C5–6 level, alcoholic cerebellar degeneration, multiple sclerosis and brain stem infarction.¹ Other associated conditions are chronic meningitis with basal arachnoiditis, sphenoid wing meningioma, aneurysm of the supraclinoid portion of the carotid syphon,² morphine poisoning³ and it has also been described in eight patients with no definite diagnosis.¹ With many of these conditions the patients may also have had internal hydrocephalus, but the nystagmus was thought to be due to the primary lesion. Its mechanism remains unknown although Zee *et al*⁴ using a computer model based on clinical data from three patients have postulated that “it occurs due to defective transmission of down velocity information to the neural integrator responsible for smooth eye movements”. In the patient to be described it seems that internal hydrocephalus had a direct part to play in provoking downbeat nystagmus.

MB, a 57-year-old lady, was admitted in August 1979 with a subarachnoid bleed. Five weeks and one week before

admission she had developed severe generalised headache associated with vomiting. On admission she was normotensive, drowsy, and speaking incoherently, with neck stiffness, bilateral papilloedema and an extensor left plantar response. Over the next week she became progressively unresponsive. Lumbar puncture showed uniformly blood stained CSF. Other investigations included a normal blood count and clotting profile; there was unequivocal biochemical evidence of inappropriate antidiuretic hormone (ADH) secretion (serum sodium 110 mmol/l, serum osmolality 210 and urine osmolality 490 mosm/kg). A CT scan showed hydrocephalus with enlargement of the entire ventricular system without evidence of any fresh subarachnoid blood or intracerebral abnormality. She was treated with fluid restriction and the serum sodium rose to normal in the first week. With the rise of sodium, her level of consciousness improved, and she began to speak. Though still confused, her eye movements could be examined. She was found to have a coarse downbeat nystagmus, without horizontal nystagmus or diplopia and the range of eye movements appeared full. Repeat CT scan after two weeks showed increased hydrocephalus. Bilateral carotid angiograms showed marked spasm of the right middle cerebral artery, and a lobulated aneurysm 1.5 cm in diameter on the anterior communicating artery. Because of the persistent confusion and increasing ventricular size a ventriculoperitoneal shunt (Unishunt 5–9 cm H₂O pressure) was inserted. By the next day she was talking rationally and the nystagmus had resolved completely. The

aneurysm was clipped about a week later. A check CT scan showed normal appearances and she has remained well since.

We believe that the most satisfactory interpretation of this patient's illness is that the downbeat nystagmus was in some way caused by hydrocephalus. If correct, this has two implications for other patients with both downbeat nystagmus and hydrocephalus. First, for their clinical care it may not be necessary to seek any other structural abnormality. Secondly, if a local structural lesion is present, downbeat nystagmus may be due to associated hydrocephalus and not to the local lesion.

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References

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