Short report

Ocular bobbing in encephalitis

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SUMMARY Abnormal eye movements described as ocular bobbing are most often encountered in the setting of pontine vascular disease. This report deals with the onset and course of ocular bobbing in a young woman who had encephalitis confirmed by biopsy. Unusual eye movements developed during progressive deterioration of brain and brain stem function which led to coma. The eye movements occurred spontaneously and could also be triggered by cutaneous stimulation of the face, head, extremities, and auditory canals. The patient recovered fully over a period of several months.

Ocular bobbing is an unusual disturbance of eye movement. The eyes repeatedly move briskly downward from the horizontal position only to drift slowly back to the horizontal position. Ocular bobbing occurs most commonly in patients with pontine haemorrhage, although it may occur in various other clinical settings. We report clinical and pathological data about a young woman with encephalitis confirmed by biopsy who developed ocular bobbing.

Case report

A 19-year-old woman was admitted to the University of Rochester Medical Center because of fever, a stiff neck, and deterioration in the level of consciousness. Two days before admission she had an exudative pharyngitis treated with penicillin and later cephalexin. She developed strange behaviour and a high fever one day before admission, and was difficult to arouse on the morning of admission. She was unresponsive to verbal stimuli. Her pulse was 110/minute, temperature 40°C, and her neck was rigid. Cheyne-Stokes respirations alternated with periods of regular rapid breathing. She exhibited random elevation and extension movements of her arms, and withdrew her extremities to avoid painful stimuli. The fundi were normal, pupils were equal and reactive, and oculocephalic responses were full; there was no facial weakness. Paratonia was evident in all

extremities, and the tendon jerks were symmetrical. An EEG showed mild bilateral occipital slowing. Intermittent polyrhythmic slow waves were present in the temporal regions. CT scan and radionuclide brain scan were normal. Lumbar puncture showed a total CSF protein of 1.06 g/l, CSF glucose of 3.2 mmol/l and a lymphocytic pleocytosis of 60 mononuclear white cells/mm³.

Her level of responsiveness declined during the second day in hospital. Muscle stretch reflexes were diminished in her lower limbs. Brain stem reflexes persisted. A repeat EEG showed increased generalised slowing. A right temporal craniotomy and temporal lobe biopsy were performed. The brain was noted to be oedematous and bulged at the operative site. A course of adenine arabinoside was initiated.

The brain biopsy (figure) contained several foci of pleomorphic microglia and macrophages forming microglial nodules. Some blood vessels had perivascular lymphocytic cuffing. No inclusion bodies were found. No perivascular demyelination was evident. The biopsy was thought to be consistent with viral meningoencephalitis.

During the third day her limbs became flaccid and she required respiratory assistance due to long periods of apnoea. Pupillary light responses, oculocephalic responses, and corneal reflexes remained intact. She had abnormal eye movements on the fifth day in hospital. On oculocephalic testing she had full adduction but absent abduction bilaterally. She developed spontaneous conjugate rapid down-gaze with a slow drift back to the horizontal meridian with a "bobbing quality." For the next six days she continued to show ocular bobbing. Upon painful stimuli or passive movements of her limbs or neck, bobbing eye movements were precipitated. After the stimulus, the eyes

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Figure Brain biopsy:
(a) Numerous scattered
pleomorphic microglia (arrows).
(b) Perivenular cuffing by
lymphocytes. H and E $\times 340$.

moved briskly and conjugately downward, where they remained for one to 10 seconds, followed by a slow drift back to the original position. This was repetitive, but seemed to tire over a period of one to three minutes. Bobbing could be again induced by sensory stimuli. Cold water caloric irrigation of the external canals caused unusually vigorous bobbing which persisted for several minutes. No spontaneous or induced abduction of either eye was noted. At no time did the patient lose corneal or light reflexes.

On the tenth day in hospital she began to breathe spontaneously and exhibited lip movements. On the thirteenth day she was noted to have some visual fixation when presented with visual stimuli and exhibited 50% of full abduction bilaterally when stimulated with the doll's head manoeuvre. Two and a half weeks after admission she was alert and able to comprehend simple sentences. Volitional lateral eye movements were full although she had some horizontal lateral gaze nystagmus. In the weeks that followed she has made steady improvement, gradually regained normal use of her limbs, and continence, and is doing well in college. An EEG five months after the illness was normal. No specific infectious agent was incriminated by culture or serologic tests.

In summary, she developed a rapidly progressive meningoencephalomyelitis. Brain biopsy specimen showed changes consistent with viral encephalitis. She exhibited dramatic stimulus-sensitive ocular bobbing at the peak of her illness which lasted for one week. Her course was one of progressive improvement eventuating in full recovery.

Discussion

C Miller Fisher described ocular bobbing in 1959¹ and gave a more detailed account in 1964.² Two patients reported had pontine infarcts and the third a pontine haemorrhage. In these patients

lateral eye movements were either severely impaired or absent. Daroff and Waldman³ reported ocular bobbing in a two-year-old child who had a pontine glioma. In this patient the eye movements differed from the initial description in that downward excursions were often more evident in one eye than the other and the eyes would occasionally remain in a downward position for 10 seconds before drifting upward. As in our patient ice water irrigation of the ear canals increased the frequency and amplitude of these eye movements. Hameroff *et al*⁴ reported four additional cases of ocular bobbing associated with cerebellar haemorrhage, pontine haemorrhage, brain stem contusion, and basilar artery thrombosis. They cautioned that ocular bobbing does not necessarily imply a poor or fatal outcome, and mentioned a patient who exhibited ocular bobbing during recovery following cardiac arrest. The potential for recovery despite ocular bobbing has subsequently been emphasised.⁵ Some authors concluded from early reports that typical ocular bobbing strongly suggests pontine haemorrhage,⁶ while subsequent reports have emphasised the variety of clinical settings in which ocular bobbing may be observed.^{7,8} For example, ocular bobbing has been described with bromide intoxication,⁹ in a patient with hepatic encephalopathy,¹⁰ and in association with multiple sclerosis.¹¹ Nair *et al*¹² reported transient ocular bobbing in a 12-year-old girl who was diagnosed clinically as having brain stem encephalitis; however no tissue was examined neuropathologically. Our case emphasises the possibility for recovery despite the presence of ocular bobbing as well as the variety of settings in which ocular bobbing may occur.

The precise mechanism for ocular bobbing remains uncertain. Because the lesions most commonly associated with ocular bobbing are located bilaterally in the paramedian pontine tegmentum, and because stimulation of the more laterally placed superior vestibular nucleus can result in vertical eye movement,¹³ spontaneous or induced activity of vestibulo-oculomotor pathways may mediate ocular bobbing.^{3 14}

Ocular bobbing occurring in encephalitis is rare. Our case represents the second described in the literature and is the first with pathological verification of encephalitis. Recognition of an association between ocular bobbing and encephalitis is important because of differences in treatment and prognosis between pontine vascular disease and sporadic encephalitis.

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