Short report

The clinical spectrum of ocular bobbing and ocular dipping

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SUMMARY  The term “ocular bobbing” defines a distinctive class of abnormal spontaneous vertical eye movements which occur in a variety of clinicopathological settings. Four cardinal forms, which correspond to the predicted permutations of the two characteristic clinical variables, initial vertical excursion and phasic velocity, have now been described. Reverse ocular dipping, with directional reversal and phasic inversion from typical ocular bobbing, is the last link in this functional tetrad and is newly presented. The four pathological forms share several basic phenomenological features but exhibit clinical and aetiological diversity and significant differences in prognosis. An analysis of the clinical spectrum of disorders subsumed under the general heading of “ocular bobbing” is presented.

Since the seminal description of ocular bobbing by Fisher in 1961,¹ this distinctive class of abnormal spontaneous vertical eye movements has been shown to encompass a clinicopathological spectrum of disturbances associated with diverse clinical settings, disease states and phenomenological variations (table). Four dissociable forms have now been observed¹—⁴ and correspond to the predicted permutations obtained by combining the cardinal vertical excursions seen (up or down) with the initial phasic velocity (fast or slow). Reverse ocular dipping, which exhibits reversal of direction and inversion of phase when compared with the original description of ocular bobbing, is the final link of the clinical tetrad and is newly described.

Case report

A 42 year old man with a history of intravenous drug abuse, acquired immunodeficiency syndrome (AIDS) and persistent cryptococcal meningitis was admitted to the hospital for placement of an Omaya reservoir and intrathecal amphotericin. This therapeutic regimen resulted in modest clinical improvement coincident with a drop in CSF cryptococcal antigen titres. At this time, his mental state remained significantly impaired. He could respond to two-step lateralised motor commands and answer questions using simple phrases. A right homonymous hemianopsia, moderate right-sided facial, limb weakness and hyperactive deep tendon reflexes were present. No abnormal brainstem or cerebellar signs were seen. He had two brief seizures during the course of his illness, one generalised and the other right focal motor. These were treated with phenytoin (300 mg daily). Phenytoin serum levels were consistently in the lower therapeutic range. The interictal EEG showed diffuse background slowing; the CT scan, diffuse cerebral atrophy with focal hypodense areas in the left parieto-occipital and right paravermian regions. CSF pressure at the time of Omaya reservoir placement was normal.

During the period of modest clinical improvement, abnormal eye movements were first noted. These consisted of gradual conjugate upward movement of the eyes over 2–4 seconds so that the pupils were completely covered by the upper lids. The globes then stayed in this position for 2–10 seconds followed by a rapid downward movement to midposition. The cycle was repeated at irregular intervals of 5–15 seconds. These ocular movements were dampened by voluntary effort and exacerbated by noxious stimuli. Eye movements were normal as assessed on bedside clinical examination. He had intact horizontal and vertical saccadic and pursuit movements and optokinetic nystagmus in both the horizontal and vertical planes, with absence of roving eye movements or lid retraction. Vertical and horizontal oculocephalic and caloric responses were also preserved. Pupils were midposition and equal and minimally reactive to light and accommodation. The patient was awake and responsive throughout these ocular movements and showed
Table Characteristic features of ocular bobbing and ocular dipping

<table>
<thead>
<tr>
<th>Ocular Bobbing</th>
<th>Initial vertical excursion (Direction/Phase)</th>
<th>Clinicopathological associations</th>
<th>Distinguishing clinical features</th>
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</thead>
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<tr>
<td>Typical</td>
<td>Down/Fast</td>
<td>Pontine locus (haemorrhage, tumour, infarct)</td>
<td>Absence of horizontal conjugate gaze</td>
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<td></td>
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<td>Extra-axial mass</td>
<td>Calories may accentuate</td>
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<td></td>
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<td>Encephalitis</td>
<td>Impaired arousal</td>
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<td></td>
<td></td>
<td>Metabolic/toxic encephalopathy</td>
<td>Prognosis poor with pontine pathology</td>
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<tr>
<td></td>
<td></td>
<td>Cerebellar haematoma</td>
<td>Presence of horizontal conjugate gaze</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Metabolic encephalopathy</td>
<td>Prognosis variable</td>
</tr>
<tr>
<td>Atypical</td>
<td>Down/Fast</td>
<td></td>
<td>Associated with:</td>
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<td></td>
<td>Convergence</td>
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<td>Reverse Ocular Bobbing</td>
<td>Up/Fast</td>
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<td>Phase papillary constriction</td>
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<td>Ocular Dipping (Inverse Bobbing)</td>
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<td>Caloric induction</td>
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<tr>
<td>Reverse Ocular Dipping</td>
<td>Up/Slow</td>
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<td>Locked-in state</td>
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no adventitious movements or electroencephalographic concomitants.

Discussion

Ocular bobbing and related disorders comprise a distinctive class of abnormal spontaneous vertical eye movements, usually seen in comatose states, that can be distinguished from ocular myoclonus, myorhythmia, nystagmus and opsoclonus by several features: irregular rate, biphasic velocity, tonic inter-phase interval, extent of vertical excursions, absence of associated adventitious movements and clinical accompaniments. Ocular bobbing consists of intermittent, often conjugate, fast downward movement of the eyes followed, after a brief tonic interval, by a slower return to primary position. It has been associated with intrinsic pontine pathology, particularly haemorrhage, tumours or infarction.1 5–16 In these patients, the prognosis is often poor, and although some individuals do recover,12 severe residual functional deficits are usually apparent.9 Ocular bobbing may also occur in the setting of extra-axial posterior fossa masses,7 17 18 diffuse encephalitis19 and toxic and metabolic encephalopathies.9 20 21

The incidence of bobbing is much lower in association with these clinical entities but prognosis for meaningful survival may be better, especially in the last named category.22 In most cases, voluntary conjugate horizontal eye movements are absent and this feature was first used to define “typical” forms. It was initially postulated that ocular bobbing represented a disturbance in the pathways mediating downward gaze.23 However, the subsequent demonstration of impaired voluntary upward gaze in this disorder15 reinforced Fisher’s original suggestion that the bobbing may reflect the residual movements of patients who have severe limitations of their horizontal and vertical eye movements.1 5 The vertical excursions are usually conjugate but may infrequently be dysconjugate and occupy the full vertical range or only a fraction of it.5 6 The use of cold caloric can be found to increase the amplitude and frequency of the movements or to have no demonstrable effect.5 6 In some cases, the bobbing has been found to exist when cold caloric can still induce conjugate horizontal movements and ceases with disease progression.5 The presence of normal pupillary reactivity and respiratory efforts, among other functions, in severely affected patients suggests that major midbrain and medullary centres are intact.5 There is also a monococular form of bobbing which coexists with a contralateral oculomotor nerve palsy.9

Several atypical forms of ocular bobbing have also been described. These have been associated with obstructive hydrocephalus, cerebellar haematoma or metabolic encephalopathy and usually display intact spontaneous and reflex horizontal movements.5 9 Other varieties occur with convergence movements (“V” bobbing), phasic pupillary constriction, cold caloric induction or in concert with a “locked-in” state.5 9 Especially in the latter example, downward eye excursions have been shown to be greater than in typical cases.5 9

Reverse ocular bobbing, in which the eyes initially jerk upward with a fast component and after a brief delay slowly return to midposition, has been found to be a non-localising sign in coma. Several examples
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have been seen in patients deeply comatose from metabolic encephalopathy. A slow conjugate downward movement of the eyes, followed by a rapid return to midposition, has been termed ocular dipping (or inverse ocular bobbing) and has been seen following anoxic coma or after prolonged status epilepticus. The generalised electroencephalographic slowing seen with the latter cases suggests that cortical depression may be a necessary concomitant. In addition, with anoxic encephalopathy, metabolic suppression or scattered cortical infarcts have been associated with incomplete lenticular nuclear damage and evocation of the dipping with passive eye movements. Roving conjugate or dysconjugate horizontal eye movements are prominent findings and normal spontaneous and elicited reflex upgaze are present. No pontine dysfunction is evident and normal recovery is possible. The suggestion that ocular dipping may be caused primarily by diffuse dysfunction rather than a single structural locus is supported by the absence of brainstem abnormalities in two necropsy cases and by the tendency of the eye findings to abate as patients regain consciousness.

The present case report documents a fourth form of abnormal eye movements, termed reverse ocular dipping, which consists of a slow upward deviation of the eyes, a brief tonic phase and then a rapid return to midposition. The patient was awake and partially responsive in contrast to all reported cases of ocular dipping and most cases with ocular bobbing which were associated with coma. There was an absence of roving eye movements which were present in all reported examples of ocular dipping, and there were intact oculocephalic and caloric responses, which are absent in "typical" ocular bobbing. The fact that reverse ocular dipping is associated with advanced metabolic or viral encephalopathy and the absence of clinical or neuroimaging signs of brainstem involvement also implicates diffuse dysfunction as a causative mechanism in this condition. However, the different clinical settings in which these two forms of ocular dipping occur suggest that they may be nosologically distinct. The clinical tetrad of disordered vertical eye movements subsumed under the general heading of ocular bobbing will serve as an important resource both for our future clinical understanding of altered states of consciousness and for basic research on the physiology of vertical eye movements in humans.

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References