

Ocular bobbing¹

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Ocular bobbing is an unusual neurological sign observed in gravely ill, stuporous or comatose patients with extensive disease of the pons. In a setting of absent spontaneous and reflex horizontal eye movements, there occur abrupt downward jerks of both eyes, followed by a slower return to mid-position. The movements may occur at regular intervals or may be arrhythmic and unpredictable. The sign was first briefly mentioned by Fisher (1961) and more recently fully described by the same author (Fisher, 1964). His three cases verified pathologically were those of adults with pontine infarction or haemorrhage. We have recently observed ocular bobbing in a child with a pontine neoplasm.

CASE REPORT

The patient was a Negro girl aged 2 years 9 months when first admitted to the Grace-New Haven Community Hospital. Her development had been normal and no significant prior illnesses had been noted. On the morning of admission, she was noted to fall, shake her limbs, and stare into space in a glassy-eyed fashion. Immediately thereafter, left-sided weakness was noted and she was brought to the hospital. Family history contributed nothing.

On initial examination, the child was lethargic but would withdraw vigorously from painful stimulation and could be induced to answer simple questions. Vital signs and general physical examination were normal. Neurological examination revealed a left lower facial weakness, a mild spastic left hemiparesis and hyperreflexia, and bilateral Babinski signs. Fundoscopic examination was normal, and remained so throughout the entire hospital course.

Initial blood count, urine analysis, urea nitrogen, electrolytes, blood sugar, calcium and phosphorous determinations were normal. No evidence for a bleeding diathesis was elicited. Skull radiographs and anechoencephalogram were normal. Lumbar puncture revealed a uniformly pink fluid under normal pressure. Red blood cell count was 11,000, there was one lymphocyte and protein was 23 mg. per 100 ml. Subsequent lumbar punctures showed a rise in protein to a maximum of 196 mg. per 100 ml.

The hospital course was marked by a brief period of mental clearing, followed by renewed obtundation and the onset of focal, left-sided seizures. On the third hospital

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day, opisthotonos was noted for the first time. Four-vessel angiographic studies were carried out and were normal. Treatment with hypothermia and anticonvulsants was instituted, but stupor, opisthotonos, and intermittent focal seizures persisted. On the ninth hospital day, it was first noted that the patient's spontaneous and oculocephalic reflex eye movements were restricted to the vertical plane, and three days later only downward movements could be elicited.

The spontaneous eye movements consisted of abrupt downward jerks. Slower return to mid-position usually occurred promptly, although up to 10 seconds might elapse before the return. Either eye might make a larger excursion than its mate. No definite time cycle was noted, nor was there any associated palatal or skeletal myoclonic movement. Ice water caloric stimulation of either labyrinth produced a definite increase in the frequency and amplitude of the movements. The pupils remained small but reactive throughout. At no time were any horizontal or upward movements noted or elicited.

On the 23rd hospital day a ventriculogram showed marked posterior displacement of the fourth ventricle. Exploratory craniotomy was carried out with drainage of a cyst in the roof of the fourth ventricle. Post-operatively, there was transient improvement in vital signs and level of consciousness, but no change in the ocular phenomena. The patient died suddenly on the 37th hospital day.

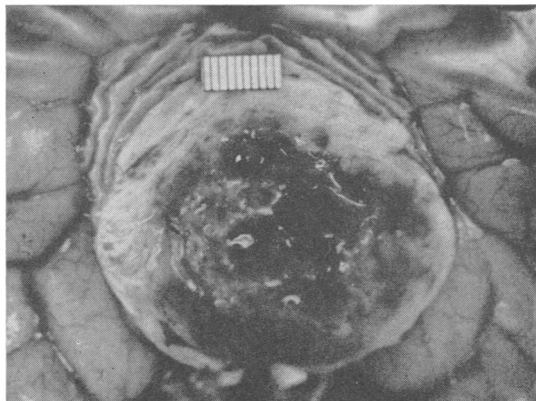


FIG. 1. Section of mid-pons demonstrating massive area of haemorrhage and necrosis. Microscopical examination revealed a glioblastoma multiforme.

At post-mortem examination, pulmonary congestion and atelectasis, necrotizing oesophagitis, and haemorrhagic cystitis were found. The brain contained a large haemorrhagic tumour mass almost completely replacing the pons (Fig. 1), and extending from the caudal mid-brain area superiorly to the pontomedullary junction inferiorly. Microscopically, this was a glioblastoma multiforme. No lesions were found in the cerebral hemispheres or main vascular trunks of the brain.

DISCUSSION

The eye movements in our case differed somewhat from those described by Fisher (1964). In his cases, the movements were always conjugate and were of limited excursion with an immediate return to mid-position. They occurred at regular intervals and were not influenced by ice water caloric stimulation. In our patient, the downward excursions were sometimes greater in one eye, and would occasionally remain in the deviated position for about 10 seconds before returning. The extent of the movement was greater, in that full downward range was achieved. There was no regularity of time intervals between jerks, and ice water caloric stimulation definitely increased their frequency and amplitude. Despite these differences, the basic phenomenon appears to be the same.

The irregularity, occasional dysconjugate nature, persistence of the deviated downward position, and lack of synchronous associated movements of mid-line structures distinguish this phenomenon from ocular myoclonus. The initial jerk followed by the slow return is the reverse sequence of the movement in nystagmus. A different descriptive term is needed, and Fisher's designation of 'ocular bobbing' seems appropriate.

The anatomical pathways for vertical eye movements are not as well understood as those for the horizontal plane. A frame of reference for the former requires a brief review of the latter. Conjugate horizontal gaze to either side is controlled by the contralateral cerebral cortex. Clinical evidence seems to implicate area 8 of Brodmann as a 'centre' for voluntary horizontal gaze (Cogan, 1956; Adler, 1959). The subcortical pathway to the brain-stem is unclear, but may involve the anterior limb of the internal capsule. It passes caudally in the paramedian reticular formation of the brain-stem ventral to the median longitudinal fasciculus, and decussates at the level of the trochlear nucleus (Bender and Shanzer, 1964). In the mid-pons, the tract has connexions with the ipsilateral abducens nucleus and the contralateral median longitudinal fasciculus, ascending in the latter to the medial rectus portion of the oculomotor nucleus. The presence of a synaptic 'centre' in the pons is improbable. If one

exists, it would appear to be in the vestibular nucleus complex (Spiegel, 1933).

There are no known cerebral centres for vertical *i.e.*, upward or downward, gaze. Whereas the oculomotor pathways for horizontal movements are unilateral, there is strong experimental evidence that mediation is bilateral for vertical movements (Bender, 1960; Bender and Shanzer, 1964). True vertical movements occur only occasionally from unilateral stimulation of the monkey cortex (Wagman, 1964). Similarly, in the monkey brain-stem, only rarely does unilateral stimulation or destruction affect vertical movements (Bender and Shanzer, 1964). However, bilateral cerebral and brain-stem stimulation characteristically produces vertical eye movements, and bilateral paramedian brain-stem lesions, as reported by the same authors, result in gross defects in these movements.

In man also, paralysis of vertical gaze is generally secondary to bilateral lesions. However there are pathologically verified cases in which unilateral brain-stem lesions were responsible (Környey, 1969; Molnár, 1959; Hatcher, 1965).

The specific role of the midbrain tectum in vertical eye movements is denied by some (Spiller, 1909; Denny-Brown, 1962; Bender and Shanzer, 1964; Pasik and Pasik, 1964; Spiegel, 1964) and maintained by others (Cogan, 1956; Adler, 1959; Cogan, 1962a and 1964b; Myers, 1964).

The vertical movement pathway ultimately terminates in the midbrain nuclei of the oculomotor and trochlear cranial nerves. Experimental and clinical evidence indicates that the tract descends beyond the midbrain to a lower brain-stem level, and then re-ascends to these nuclei (Bender and Shanzer, 1964; Smith, David, and Klintworth, 1964; Spiegel, 1964). Lesions strictly limited to the medulla can produce vertical nystagmus (O'Brien and Bender, 1945). Cogan (1956) feels that there is a medullary influence on vertical movements, located in a 'vestibular reflex centre'.

While in monkeys simultaneous bilateral cold caloric stimulation produces pure vertical eye movements (Bender and Shanzer, 1964), this is not apparently true for normal man (Norton, 1964). In this connexion, it is of interest that downward jerks similar to ocular bobbing have been described after unilateral caloric stimulation in a comatose man with multiple traumatic intracerebral haemorrhages (Fisher, 1964).

The separation of upward and downward vectors in vertical eye movements is difficult. Usually, both are involved in experimental brain-stem lesions. In small pre-tectal lesions in monkeys there is paralysis of upward gaze only, but if the lesions are enlarged, downward paralysis also results (Bender and Shanzer,

1964). In man, midbrain lesions disturbing vertical gaze predominantly involve upward movements (Duke-Elder, 1949; Cogan, 1956).

In purely downward eye movements, the medulla may have particular importance. Bender (1960) has stated that in man there is indirect evidence that caudal brain-stem lesions induce defects in the downward plane. Cogan (1964c) feels that vertical nystagmus limited to downward gaze is characteristic of caudal medullary lesions.

In our patient and in those of Fisher (1964), the extensive pontine destruction resulted in paralysis of voluntary and reflex horizontal and presumably upward eye movements. The medulla was essentially spared in all of the cases. Considering the previously mentioned data, this would suggest that ocular bobbing originates from the portion of the vestibular nuclear complex within the medulla.

Experiments conducted by Spiegel (1933) seem pertinent in further delineating the mechanism of ocular bobbing. He found that unilateral stimulation of the frontal or occipital lobes in animals produced horizontal, but only occasionally vertical, eye movements. However, after bilateral injury to the vestibular nuclei, cortical stimulation resulted in vertical movements in almost 50% of attempts. The presence of focal seizures in our patient suggests some degree of cortical irritability which may have played a role. However, it should be noted that none of Fisher's cases had seizures, so it is apparent that cortical discharges are not essential for the production of ocular bobbing. Vestibular influence is established by the fact that, in our patient, caloric testing exaggerated the bobbing movements.

Fisher (1964) postulates that ocular bobbing represents 'normal roving movements of the eyes in which the lateral component is in abeyance leaving the vertical excursion in evidence'. He also states that the neural discharge originates in the midbrain. We believe that the unique pattern of this phenomenon suggests something other than 'normal roving movements'. Further, on the basis of the previous discussion it seems reasonable to consider ocular bobbing as representing medullary neural activity, which may be triggered by cortical and/or vestibular stimulation though it is not invariably dependent on either of them.

SUMMARY

Ocular bobbing is an unusual eye movement disorder seen in patients with extensive pontine disease. The phenomenon consists of spontaneous downward jerks of the eyes in a clinical setting of total paralysis of eye movement in the other planes.

A case of ocular bobbing in a child with a pontine glioma is presented and the oculomotor gaze pathways are reviewed. It is concluded that ocular bobbing probably represents neural activity from an intact medulla.

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