Ocular bobbing with superior cerebellar artery aneurysm

Case report

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A teen-age girl became comatose after the sudden onset of headache. Initial angiography did not reveal the site of bleeding. The subsequent onset of ocular bobbing directed attention to the region of the pons. Repeated angiography showed an aneurysm of the superior cerebellar artery. At surgery, the fundus of the aneurysm was adherent to the pons and there was a small hematoma within the pons. Ocular bobbing is rare, but is most commonly seen in association with destructive lesions of the pontine tegmentum, and is a useful localizing sign.

KEY WORDS: ocular bobbing · superior cerebellar artery · aneurysm · pontine hemorrhage

Ocular bobbing is a rare disorder of eye movement characterized by abrupt, spontaneous, conjugate downward deviation of the eyes with slow return to mid-position. This phenomenon is most commonly seen with destructive lesions of the pontine tegmentum. The development of ocular bobbing in the following case was a useful sign in the localization of her neurological lesion and in the selection of appropriate diagnostic studies.

Case Report

This 13-year-old girl was in good health until the morning of admission, when she suddenly cried out and complained of a buzzing sensation in her right ear. She thought an insect had entered her ear. Three to 5 minutes later she complained of a very severe headache and lapsed into coma. She vomited several times before arriving at the hospital.

Examination. At the time of initial examination, the patient responded to pain by moving her extremities. The neck was supple. The pupils were 2 mm in diameter and equal. There was no Babinski sign or clonus. The cranial nerves were intact. On general examination, her blood pressure was 114/78, with a pulse of 124. The temperature was 37.5° and her respiratory rate was 32. A lumbar puncture showed spinal fluid that was grossly bloody in all tubes. The opening pressure was 210 mm H₂O. The patient was treated with anti-convulsants and epsilon-aminocaproic acid.

Late in the afternoon of the day of admission, the patient showed progressive lethargy. Her gaze was dysconjugate, and there was minimal movement of the eyes with head rotation. She had a right hemiparesis. She was treated with mannitol. The following morning she was more responsive and had some spontaneous movements of all ex-
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Fig. 1. Angiogram, anteroposterior (left) and Towne (right) projections, showing the superior cerebellar artery aneurysm (arrows). The insets are magnified views of the area of the aneurysm.

tremities. A left sixth nerve paresis was present.

A left carotid and a right retrograde brachial angiogram were done. The basilar artery and both posterior cerebral and superior cerebellar arteries were filled with contrast material, but no aneurysm was demonstrated. Treatment with epsilon-aminocaproic acid was continued. Her condition remained the same.

On the third day of her illness, she was noted to have spontaneous, conjugate downward deviation of the eyes at a rate of four to six per minute. Oculocephalic reflexes were absent. Caloric stimulation tests were not done. This finding was felt to represent ocular bobbing, and believed to localize the lesion to the brain stem. Repeat angiography showed a left superior cerebellar artery aneurysm (Fig. 1). The fundus pointed medially and inferiorly and appeared to enter the pons.

Operation. On the 11th day following subarachnoid hemorrhage, a craniotomy with a subtemporal approach to the aneurysm was performed. The tentorium was incised, exposing a small hematoma that indicated the site of the aneurysm. The fundus was adherent to the pons. During mobilization of the vessels, a small amount of hematoma extruded from the substance of the pons. The aneurysm was trapped.

Postoperative Course. The postoperative course was complicated by non-absorptive hydrocephalus requiring a ventriculoperitoneal shunt. Her improvement was very slow and she continued to be markedly obtunded. Six months after the subarachnoid hemorrhage, she began to respond to her environment and over the next 2 months became alert with appropriate thought processes. As she tried to do more things, it was apparent that she had a marked ataxia involving both the extremities and the trunk. Her eye movements were dysconjugate and her hemiplegia was gradually improving.

Discussion

Multiple abnormalities of eye movement are seen in lesions of the midbrain and pons and are useful in the localization of lesions in this area. These include paralysis of conjugate gaze to the side of the lesion, vertical and horizontal nystagmus, skew deviation, paralysis of bilateral horizontal gaze, paralysis of vertical gaze, and many others. Fisher coined the term ocular bobbing to describe spontaneous, abrupt conjugate downward movement of the eyes followed by a slow return to the primary position. These movements occur two to 12 times per minute and are seen in association with bilateral paralysis of horizontal gaze. Oculo-
Cephalic responses are absent horizontally and no lateral movement can be elicited by caloric stimulation although at times caloric stimulation may increase the rate of bobbing. The quick downward movement of the globe followed by the slower return to the primary position resembles the movement of the upper eyelid during winking.

Ocular bobbing has been seen most often in association with pontine hemorrhage, but has also been seen with pontine infarction, glioma, or trauma. Cerebellar hemorrhage can give rise to ocular bobbing although it most commonly is associated with "atypical" bobbing. Atypical bobbing is differentiated from the typical form by the preservation of horizontal eye movements. Atypical bobbing has been seen with hydrocephalus, head trauma, posterior communicating aneurysm, and metabolic encephalopathy and is, therefore, less specific in its localizing value. The pathophysiological mechanism underlying ocular bobbing is unknown. It has been proposed that it represents the remnant of roving eye movements in the presence of paralysis of horizontal gaze or the manifestation of "medullary neural activity."

References

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