Specificity of “Peering at the Tip of the Nose” for a Diagnosis of Thalamic Hemorrhage

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Background: Tonic inward and downward deviation of the eyes ("peering at the tip of the nose") is regarded as a unique feature of thalamic hemorrhage, but the mechanisms of this ocular finding remain obscure.

Objectives: To describe 4 patients who showed tonic inward and downward deviation of the eyes from brainstem or thalamic lesions and to discuss the possible mechanisms involved.

Design: Case report.

Setting: Secondary and tertiary referral hospitals.

Results: One patient developed alternating esotropia with downward ocular deviation from thalamic hemorrhage compressing the midbrain. Two patients showed multiple infarctions in the territory of the posterior circulation with or without the involvement of the thalamus. Another patient had lateral pontine hemorrhage extending up to the midbrain tegmentum. Ocular bobbing preceded or accompanied tonic ocular deviation in 3 patients.

Conclusions: Tonic inward and downward deviation of the eyes may develop in thalamic or brainstem lesions. Irritation or destruction of the neural structures involved in the vergence and vertical gaze may cause this ocular sign in mesodiencephalic lesions. Skew deviation and esotropia from abduction deficit may be involved in some patients. Ocular bobbing and tonic downward deviation may share a common pathogenesis.

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REPORT OF CASES

PATIENT 1

A 65-year-old man developed sudden left hemiplegia and dysarthria. Blood pressure on admission was 180/90 mm Hg. He had normal pupils and full ocular motility. He showed dysarthria, left facial weakness, and left hemiplegia. Other findings of the general medical and neurological examinations were normal. An initial computed tomographic scan of the head showed right thalamic hemorrhage with extension into the lateral and third ventricles. Three days later, he became unconscious. Both eyes showed downward deviation with additional esotropia of the left eye. The right pupil was 2.0 mm and the left was 2.5 mm, both reactive to light. His head and neck were turned to the right. Intermittent right-beating nystagmus was noted. Both eyes were fixed on doll’s eye maneuvers, horizontally and vertically. Follow-up computed tomography showed increased right thalamic and intraventricular hemorrhage, compressing the midbrain (Figure 1B). The next day, in contrast to the previous findings, the right eye became deviated inward and downward. The left eye also showed a mild downward deviation (Figure 1A). Three weeks later, the patient could respond to commands and showed no tonic ocular deviation. The only abnormal ocular motor finding was a gaze palsy to the left.
PATIENT 2

A 66-year-old man with a history of diabetes mellitus and hypertension was found in an unconscious state. He had experienced a stroke 2 years previously and had been treated with warfarin sodium. Her blood pressure was 190/100 mm Hg. The pupils were equal at 2.0 mm and reactive to light. Both eyes were fixed on horizontal and vertical doll’s eye maneuvers. The left eye was hypotropic. She showed intermittent bobbing eye motion, which was more prominent or purely monocular in the left eye. A CT scan revealed pontine tegmental hemorrhage mainly on the left side, which extended up to the midbrain (Figure 2B). Three weeks later, the left eye showed tonic downward and inward deviation (Figure 2A). Occasional bobbing of the left eye remained. She also showed intermittent abducting nystagmus of the right eye on attempting rightward gaze.

PATIENT 3

A 54-year-old man with hypertension presented with a sudden loss of consciousness. On arrival at our hospital, his blood pressure was 190/100 mm Hg. Neurological examination showed a comatose state and marked extensor rigidity of the arms and legs. The pupils were equal at 1.0 mm and reactive to light. The corneal responses were absent. The eyes did not move with doll’s eye maneuvers or caloric stimuli. The left eye showed monocular bobbing. Diffusion-weighted magnetic resonance imaging of the brain showed multiple infarcts in the territory of the posterior circulation and in lateral pontine tegmental and thalamic hemorrhages.

In autopsied patients with thalamic hemorrhage and this ocular sign, the hematoma was usually found to have extended into or to have compressed the midbrain. The mesodiencephalic junction contains neural structures that are involved in vertical gaze and vergence. Damage to this area gives rise to characteristic neuro-ophthalmologic findings.

The descending cortical pathways for convergence pass through the paramedian thalamus and inhibit the contralateral premotor vergence neurons in the midbrain, which in turn project to the medial rectus subnucleus of the oculomotor nuclei on the same side. Patients may develop unilateral or bilateral esotropia with abduction deficit due to lesions in the thalamus or mesodiencephalic junction by injuring this descending inhibitory pathway for convergence or by directly irritating the convergence neurons.

Patient 1 showed alternating esotropia resulting from thalamic hemorrhage compressing the midbrain. The initial contralateral esotropia may be explained by an injury to the ipsilesional descending pathway for convergence before decussation (Figure 5A, step 1). The subsequent ipsilesional esotropia may be caused by an

**COMMENT**

Tonic inward and downward deviation of the eyes (peering at the tip of the nose) is considered a unique feature of thalamic hemorrhage, although little is known about the underlying mechanism. We observed this ocular finding in diffuse ischemic infarcts in the territory of the posterior circulation and in lateral pontine tegmental and thalamic hemorrhages.
injury to the ipsilesional descending convergence pathway after decussation (Figure 5A, step 2) or by irritation of the ipsilesional convergence neurons (Figure 5A, step 3) due to further extension of the hematoma.

The mesodiencephalic junction contains the rostral interstitial nucleus of the medial longitudinal fasciculus, the interstitial nucleus of Cajal, the mesencephalic reticular formation, and the posterior commissure, all of which are involved in premotor control of vertical eye movements (Figure 5A).10 Forced downward gaze is common in the lesions affecting this area and presumably represents an imbalance in the vertical gaze plane.4 The vertical dissociation of the eyes may originate from skew deviation, which is an element of ocular tilt reaction. Ocular tilt reaction, which consists of head tilt, ocular torsion, and skew deviation, is observed after damage to the vestibular pathways that subserve eye-head coordination in the roll plane. This pathway runs from the labyrinths via ip-
silateral pontomedullary vestibular nuclei crossing to the contralateral interstitial nucleus of Cajal (Figure 5B). Damage to this pathway before decussation in the lower brainstem would cause skew deviation with the hypotropic ipsilesional eye, whereas a lesion in the upper brainstem after decussation gives rise to skew deviation with the hypotropic contralesional eye (Figure 5B, step 1), as in our patients 3 and 4. The accompanying esotropia in the hypotropic eye may be from concurrent damage to the descending pathway for convergence (Figure 5B, step 2) or to the abducens fascicle (Figure 5B, step 3).

In patient 2, pontine hemorrhage, predominantly on the left side, extended up to the midbrain tegmentum. Previous reports on lateral tegmental pontine hemor-

rhages have described patients with tonic downward and inward ocular deviation in the ipsilesional eye. Irritation of the mesencephalic downgaze and convergence centers due to rostral extension of the hematoma may give rise to this ocular sign. In our patient, the pupils were equal and the light reflex was preserved. The rostral interstitial nucleus of the medial longitudinal fasciculus lies dorsomedial to the red nucleus and rostral to the oculomotor nucleus. The vergence neurons also lie 1 to 2 mm dorsolateral to the oculomotor nucleus. These considerations, although hardly conclusive, argue against the direct irritation of the downgaze and convergence neurons without involving the oculomotor nuclei as the principal cause of this ocular finding. In monkeys, burst neu-
rons with upward or downward on-directions are intermingled in the rostral interstitial nucleus of the medial longitudinal fasciculus in about equal proportions.14-16 Axons mediating upward saccades may exit both rostral interstitial nuclei of the medial longitudinal fasciculus dorsally, then decussate in the posterior commissure.17,18 Burst neurons with upward on-directions project bilaterally to oculomotor nucleus neurons, whereas neurons with downward on-directions project ipsilaterally to the motoneurons of the oculomotor and trochlear nuclei without decussation.15,16 The downward deviation of the ipsilesional eye may have been due to the irritation of this descending fiber subserving downgaze (Figure 5C, step 1). Damage to the abducens fascicle by pontine hematoma may also give rise to the inward deviation of the ipsilesional eye (Figure 5C, step 2).

In 3 of our patients (patients 2, 3, and 4), ocular bobbing preceded or accompanied tonic inward and downward deviation of the eye. Previous reports of lateral tectal pontine hemorrhages described a patient who had

Figure 4. Patient 4. A, The left eye shows intermittent tonic inward and downward deviation. B-D, Axial T2-weighted magnetic resonance imaging demonstrates multiple infarcts in the bilateral cerebellum, pons, and midbrain.
this ocular deviation with ipsilateral ocular bobbing.\textsuperscript{1,2} Ocular bobbing refers to fast downward jerks of both eyes followed by a slow drift to the midposition. The downward jerks may be disjunctive or purely monocular. Patients with ocular bobbing also had abnormal upward voluntary eye movements.\textsuperscript{19} Although the mechanisms of ocular bobbing and tonic downward deviations are not precisely known, they may share a common pathogenesis of tonic or phasic imbalance in the system controlling vertical eye motion. Instability or changes of the imbalance in this system during early phases may explain the co-occurrence or transition of ocular findings observed in our patients.

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REFERENCES


