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

Kwang-Dong Choi, MD; Dae Soo Jung, MD, PhD; Ji Soo Kim, MD, PhD

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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Clinical Profiles of the Patients

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Side of Ocular Deviation*</th>
<th>Other Ocular Findings*</th>
<th>Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/65</td>
<td>L → R</td>
<td>R beating nystagmus</td>
<td>R thalamic hemorrhage</td>
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<tr>
<td></td>
<td></td>
<td>Complete gaze palsy</td>
<td></td>
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<td></td>
<td></td>
<td>initially</td>
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<td></td>
<td></td>
<td>L gaze palsy 3 wk later</td>
<td></td>
</tr>
<tr>
<td>2/F/66</td>
<td>L</td>
<td>Complete gaze palsy</td>
<td>L pontine tegmental hemorrhage</td>
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<td></td>
<td></td>
<td>Ocular bobbing (L, L&gt;R)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>L internuclear ophthalmoplegia</td>
<td></td>
</tr>
<tr>
<td>3/M/54</td>
<td>L</td>
<td>Complete gaze palsy</td>
<td>Multiple brainstem, cerebellar, and thalamic infarctions</td>
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<td></td>
<td></td>
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<tr>
<td>4/F/73</td>
<td>L</td>
<td>Complete gaze palsy</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Ocular bobbing (L, L&gt;R)</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Ocular myoclonus</td>
<td></td>
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</tbody>
</table>

Abbreviations: F, female; L, left; M, male; R, right.
*The arrow indicates the change of the eye showing ocular deviation: greater than sign, asymmetry of the ocular bobbing.

PATIENT 2

A 66-year-old man with a history of diabetes mellitus and hypertension was found in an unconscious state. She 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 hematomata 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 neuroophthalmologic findings.

The descending cortical pathways for convergence pass through the parameter thalamus and inhibit the contralateral premotor vergence neurons in the midbrain, which in turn project to the medial rectus nucleus of the oculomotor nuclei on the same side. Patients may develop unilateral or bilateral esotropia with abduction deficit (pseudoabducent palsy) 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 contralesional 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
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). Forced downward gaze is common in the lesions affecting this area and presumably represents an imbalance in the vertical gaze plane. 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.\textsuperscript{2,3} Irritation of the mesencephalic downgaze and convergence centers due to rostral extension of the hematoma may give rise to this ocular sign.\textsuperscript{3} 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.\textsuperscript{11} The vergence neurons also lie 1 to 2 mm dorsolateral to the oculomotor nucleus.\textsuperscript{12,13} 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-

Figure 3. Patient 3. A, The left eye shows tonic downward and inward deviation. B-D, Diffusion-weighted magnetic resonance imaging reveals multiple infarcts in the pons, right side of the midbrain, right thalamus, and both occipital lobes.
rons with upward or downward on-directions are intermingled in the rostral interstitial nucleus of the medial longitudinal fasciculus in about equal proportions.\textsuperscript{14-16} Axons mediating upward saccades may exit both rostral interstitial nuclei of the medial longitudinal fasciculus dorsally, then decussate in the posterior commissure.\textsuperscript{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.\textsuperscript{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 tegmental 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. 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. 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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Corresponding author and reprints: Ji Soo Kim, MD, PhD, Department of Neurology, College of Medicine, Seoul National University, Seoul National University Bundang Hospital, 300 Gumi-dong, Bundang-gu, Seongnam-si, Gyeonggi-do, 463-707, Korea (e-mail: jisookim@smu.ac.kr).

REFERENCES


