Unilateral Saccadic Pursuit in Patients With Sensory Stroke
Sign of a Pontine Tegmentum Lesion

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Background and Purpose—Pure hemisensory syndrome can be caused by small strokes occurring in a number of regions, including the thalamus and pons. Differentiation of the pontine sensory syndrome from the thalamic sensory syndrome has generally been made on the basis of distribution of sensory loss and involvement of specific sensory modalities but not without uncertainties and difficulties. Because the pontine tegmentum plays a pivotal role in generating horizontal eye movement, we attempted to discriminate these 2 syndromes by analyzing horizontal eye movements in stroke patients with pure hemisensory syndrome.

Methods—Horizontal saccade, pursuit, vestibulo-ocular reflex (VOR), and VOR cancellation (VORC) were evaluated using electro-oculography in 6 patients with hemisensory syndromes, 3 due to pontine stroke and 3 due to thalamic stroke, and all were verified by MRI or CT. In addition, somatosensory evoked potentials (SEPs) were recorded.

Results—Smooth pursuit and VOR directed toward the side of the lesion were impaired unilaterally in patients with pontine sensory stroke, whereas those 2 movements were intact bilaterally in patients with thalamic sensory stroke. Saccade and VOR were preserved in all patients. SEPs were normal in all patients with pontine and thalamic sensory strokes. No difference was found in the pattern of sensory disturbance between the 2 types of stroke patients.

Conclusions—Ipsilateral impairment of the smooth pursuit system may be a sign of a pontine lesion in patients with hemisensory stroke. (Stroke. 1998;29:2377-2380.)

Key Words: eye movements ▪ pons ▪ sensory stroke ▪ thalamus

Pure sensory stroke is defined as the presence of mostly hemisensory symptoms without other major neurological signs.1 Although thalamic stroke was the first described and is the most frequent cause of this syndrome,1–3 nonthalamic strokes involving the brain stem, internal capsule, or cerebral cortex also have been reported to produce this syndrome.3 Small pontine tegmentum lesions have been reported to be a major cause of pure sensory stroke.4-6 Differentiation of the pontine sensory syndrome from the thalamic syndrome was made on the basis of the distribution of sensory loss and involvement of specific modalities of sensation, but was not without difficulties and uncertainties.

The dorsal pons contains most brain stem centers for the horizontal saccades and smooth pursuit eye movements. Premotor commands for the horizontal saccades are generated by burst neurons within the ipsilateral paramedian pontine reticular formation (PPRF).7 The burst neurons project directly to the ipsilateral abducens nucleus to contact abducens motoneurons and internuclear neurons that project up the contralateral medial longitudinal fasciculus to contact the medial rectus subgroup of the contralateral oculomotor nucleus.7 This pathway mediates the horizontal saccades. Smooth pursuit eye movements are thought to be relayed by a double decussation pathway in the brain stem.8 The dorsolateral pontine nucleus (DLPN) in the basal pons is considered to be the major gateway for ipsilateral smooth pursuit–related signals to the cerebellum. The DLPN receives afferences from ipsilateral cortical areas such as the middle temporal and medial superior temporal areas9 and projects mainly to the contralateral flocculus/paraflocculus.10-14 The contralateral medial vestibular nucleus receives afferent projections from the contralateral flocculus/paraflocculus and subsequently projects to the ipsilateral abducens nucleus (the second decussation).8,15 Although other parallel pathways such as projections from the DLPN to the bilateral vermis have been postulated to exist, smooth pursuit eye movements are considered to be controlled mainly by this DLPN-flocculus/paraflocculus-vestibular pathway in the brain stem.8,15 Characterization of the horizontal eye movements was therefore conducted in an attempt to differentiate a pontine sensory syndrome from a thalamic one.

Subjects and Methods
We examined 3 patients with pontine sensory stroke (2 infarcts and 1 hemorrhage) and 3 patients with thalamic sensory stroke (2 infarcts...
and 1 hemorrhage) between June 1995 and March 1998. All 6 patients were examined by one of us (K.J.) and then underwent CT scan, MRI, or both within 7 days after onset. Horizontal eye movements were recorded with an alternating current electro-oculography (time constant, 16 seconds) also within 7 days after onset. Saccades and smooth pursuit were elicited with a light-emitting diode (LED) ramp located 100 cm in front of the subject. Horizontal visually-guided saccades were studied by instructing the patient to look as quickly as possible at a suddenly appearing 20° lateral target. Horizontal smooth pursuit was induced by asking the patient to track an LED moving according to a triangular velocity profile (amplitude, 40°; velocity, 16°/s). The gain (defined as the ratio of eye velocity to target velocity) in smooth pursuit was calculated.7,13 We qualitatively evaluated vestibulo-ocular reflex (VOR) and VOR cancellation (VORC; the ability to suppress VOR) on the basis of normal latencies on both sides.

### Results

The MRI/CT scan findings and clinical features of all 6 patients are summarized in the Table.

#### Neuroradiological Examinations

Results of the imaging studies are shown in the Table and in Figure 1. Three patients had small lesions involving the unilateral dorsal pons, and the other 3 had small lesions in the thalamus. The pontine lesions seemed to include the medial lemniscus within the paramedian dorsal pons, at least partially, and the thalamic lesions were located in the posterolateral part of the thalamus, very likely including the ventral posterolateral nucleus. The imaging findings in our patients were consistent with findings reported previously in sensory stroke patients.3,6

#### Sensory Patterns and Topography

Although all 6 patients had paresthesia as a main complaint, examination revealed objective sensory abnormalities in only 1 patient with pontine sensory stroke (patient 2) and in 2 patients with thalamic sensory stroke (patients 5 and 6). The pontine stroke patient and 1 thalamic stroke patient (patient 5) showed mildly decreased medial lemniscal sensation (tactile, vibration, and/or position). The other thalamic stroke patient (patient 6) showed mildly decreased spinthalamic sensation (pinprick and/or temperature) and medial lemniscal sensation; lemniscal sensory deficit was dominant. None of the patients had painful paresthesia like that observed in so-called thalamic pain syndrome.

Two pontine stroke patients (patients 2 and 3) and 1 thalamic stroke patient (patient 6) had sensory symptoms limited to the perioral-hand area (cheiro-oral syndrome). The other 3 had hemisensory symptoms involving the arm, leg, trunk, and face. None of the patients had any bilateral sensory symptoms. Besides the hemisensory symptoms, patient 6 experienced transient dysarthria and right-hand clumsiness, which disappeared 3 days after onset. In all pontine and thalamic sensory stroke patients, SEPs were elicited with normal latencies on both sides.

### Eye Movements

Eye movements were conjugate, and there was no spontaneous or gaze-evoked nystagmus in any patient. Horizontal saccades and VOR were normal in all pontine and thalamic stroke patients. Peak velocities of horizontal saccades were within normal ranges in all patients (see the Table).7 In pontine sensory stroke patients, smooth pursuit eye movements directed toward the side of the lesion (ipsilateral smooth pursuit) were impaired unilaterally and interrupted by catch-up saccades (Figure 2). This was obvious on direct observation without electro-oculographic monitoring. In contrast, thalamic sensory stroke patients demonstrated almost

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#### Table: Clinical and Neuro-Ophthalmological Features of the Patients With Sensory Stroke

<table>
<thead>
<tr>
<th>No./Sex/Age</th>
<th>Lesion</th>
<th>Sensory Syndrome</th>
<th>Saccade Velocity (°/s)</th>
<th>Smooth Pursuit Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Site/Nature</td>
<td>Paresthesia</td>
<td>Modality of Sensory Loss</td>
<td>Topography</td>
</tr>
<tr>
<td>1/M/56</td>
<td>L-P/I</td>
<td>+</td>
<td>N</td>
<td>Hemi</td>
</tr>
<tr>
<td>2/M/48</td>
<td>R-P/I</td>
<td>+</td>
<td>ML</td>
<td>C-O</td>
</tr>
<tr>
<td>3/F/64</td>
<td>L-P/H</td>
<td>+</td>
<td>N</td>
<td>C-O</td>
</tr>
<tr>
<td>4/M/59</td>
<td>R-T/I</td>
<td>+</td>
<td>N</td>
<td>Hemi</td>
</tr>
<tr>
<td>5/M/59</td>
<td>R-T/H</td>
<td>+</td>
<td>ML</td>
<td>Hemi</td>
</tr>
<tr>
<td>6/F/53</td>
<td>L-T/H</td>
<td>+</td>
<td>ML&gt;ST</td>
<td>C-O</td>
</tr>
</tbody>
</table>

L-P indicates left pontine tegmentum; R-P, right pontine tegmentum; R-T, right thalamus; L-T, left thalamus; I, infarction; H, hemorrhage; +, presence of paresthesia; N, normal objective sensation; ML, medial lemniscal sensory loss; ST, spinthalamic sensory loss; Hemi, hemisensory deficit; and C-O, cheiro-oral syndrome.
normal smooth pursuit bilaterally (Figure 2). These findings were confirmed by the gain in horizontal smooth pursuit, which showed marked right-left asymmetry only in the pontine sensory stroke patients (Table). Ipsilateral VORC (the cancellation of VOR elicited by rotation in the direction of the lesion) was similarly impaired in the pontine sensory stroke patients, whereas VORC was preserved in the thalamic sensory stroke patients (Figure 2).

Discussion

The differentiation between sensory stroke syndromes of pontine or thalamic origin has been based on the distribution of sensory loss and involvement of particular modalities of sensation. In pontine sensory stroke patients, the sensory loss involves mainly medial lemniscal sensation and is frequently distributed bilaterally in the facial area, whereas in thalamic sensory stroke patients it usually involves all sensory modalities and is unilaterally distributed on the face as well as the arm, leg, and trunk. However, there are reports of selective hemisensory loss of medial lemniscal sensation with a thalamic lesion and hemisensory deficits of all sensory modalities with a pontine lesion. In the present study, no difference was observed in the patterns of sensory disturbance between the 2 groups, underscoring the difficulty in clinical differentiation between pontine and thalamic sensory stroke based only on the pattern of sensory deficits.

SEPs are considered to depend on the integrity of the dorsal column–medial lemniscal sensory pathway. Because a majority of pontine sensory stroke patients show paramedian dorsal pontine lesions involving the medial lemniscus, SEPs are thought to have diagnostic value especially in patients with pontine sensory stroke. In our study, however, SEPs were normal in all pontine and thalamic stroke patients. Robinson et al also reported that SEPs are consistently normal in pure sensory stroke. Therefore, SEPs may not be a helpful diagnostic test in pure sensory stroke.

The present study revealed that smooth pursuit directed toward the side of the lesion was impaired ipsilaterally in all 3 patients with pontine sensory stroke, while both saccades and VOR were spared. The simultaneous impairment of ipsilateral VORC was consistent with this selective impairment of smooth pursuit, because VORC and smooth pursuit are considered to share a common pathway. Pontine tegmentum lesions in our patients may not have involved the PPRF, abducens nucleus, vestibular efferents to abducens, or medial longitudinal fasciculus, accounting for the normal saccades and VOR.

Unilateral lesions of the pontine tegmentum have reportedly caused deficits of ipsilateral or contralateral smooth pursuit eye movements. Defective contralateral smooth pursuit with pontine tegmentum lesions may be associated with several possible mechanisms: (1) disruption of afferent projections to the flocculus/paraflocculus after their decussation; (2) ipsilateral central vestibular damage; and (3) disruption of the ipsilateral vestibular fibers to the abducens nucleus before their decussation. As for ipsilateral smooth pursuit impairment by the pontine tegmentum lesion as seen in our pontine sensory stroke patients, 4 mechanisms have been postulated: (1) involvement of corticopontine pathways; (2) disruption of the pontocerebellar mossy fibers to the flocculus/paraflocculus before their decussation across the midline; (3) impairment of inhibitory projections from the flocculus/paraflocculus to the vestibular nucleus; and (4) damage to “pursuit neurons,” which lie inferior to the abducens nucleus, outside the PPRF, and which are modulated in response to ipsilateral smooth pursuit eye movements.

A rostral lesion in the pons can destroy the corticopontine pathways that mediate the smooth pursuit, but this is unlikely in our patients because, as verified by MRI/CT, our patients’ lesions did not involve the corticopontine pathway that descends through the cerebral peduncle and passes through the ventrolateral pons to terminate pontine nuclei in the pontine base. Johnston et al suggested that not only contralateral but also ipsilateral pursuit deficits with pontine tegmentum lesions can be caused by damage to the pontocerebellar pathways. However, a recent study by Glickstein et al revealed that most pontocerebellar mossy fibers decussated in the pontine base and joined the contralateral middle cerebellar peduncle to enter the flocculus/paraflocculus, suggesting that a pontine tegmentum lesion is unlikely to affect the pontocerebellar fibers before their decussation. One patient who had a lesion adjacent to the fourth ventricle was reported to demonstrate low-velocity ipsilateral smooth pursuit, which may be related to a disruption of the inhibitory projections from the flocculus/paraflocculus on the vestibular nucleus as they course in the angular bundle of Löwy. Pontine lesions in our patients with sensory stroke seem to lie more medially than the angular bundle of Löwy. Unilateral pontine tegmentum damage that abolishes ipsilateral saccades can also paralyze ipsilateral smooth pursuit. This may be related to “pursuit neurons” located in the vicinity of the abducens nucleus. Although the existence of “pursuit neurons” is still controversial, damage to such neurons may have led to the defective ipsilateral pursuit in our pontine sensory stroke patients.

In conclusion, although the precise mechanism of the smooth pursuit deficits is unclear, ipsilateral impairment of the smooth pursuit system may be a sign highly suggestive of a pontine tegmentum lesion in patients with sensory stroke.
References

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