ROLES OF HEAD, GAZE, AND SPATIAL ORIENTATION IN THE PRODUCTION OF OSCILLOPSIA

Masahiro Takahashi, MD, Yukihiro Okada, MD, Akira Saito, MD, Yasuhiro Takei, MD, Ikuko Tomizawa, MD, Keiko Uyama, MD, Izumi Takeuti, MD, and Jin Kanzaki, MD

Department of Otolaryngology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo 160, Japan

Reprint address: Masahiro Takahashi, M.D., Department of Otolaryngology, Keio University School of Medicine, 35 Shinanomachi, Shinjuku-ku, Tokyo, 160 Japan

**Abstract** — To clarify the factors causing oscillopsia, we investigated head movement, gaze stability, and perception under various situations. High-frequency head movements, whether they were horizontal rotations or passively induced vertical oscillations, produced blurred vision and gaze fluctuations in patients with labyrinthine loss. However, this sensation differed from the oscillopsia perceived during walking, as it did not involve a sensation of oscillation of the surrounding space or a loss of body balance. Although patients with labyrinthine loss showed large irregular head perturbations during stepping, the resultant retinal velocity slips seemed too small to explain oscillopsia. Walking while wearing horizontal reversing prisms produced loss of spatial orientation, dysequilibrium, and instability of vision in normal subjects, which resembled the symptoms found in patients with oscillopsia. The present study suggests that oscillopsia represents a perceptual inability to detect spatial orientation during head or body movements rather than a mere blurring of vision caused by deficient compensation.

**Keywords** — oscillopsia; locomotion; head stability; gaze; spatial orientation.

**Introduction**

Oscillopsia, which is an illusion of movement of the seen world, has been reported to occur in various kinds of vestibular disorders of both peripheral and central origins (1). Although it has been believed that oscillopsia due to labyrinthine dysfunction is produced by a retinal velocity slip, its severity does not necessarily parallel the magnitude of gaze fluctuation (2-4). The discrepancy was typically found in a patient with congenital inner ear anomalies who manifested no complaint of oscillopsia (5). In addition, the reason why oscillopsia is most commonly perceived during upright locomotions is still to be clarified. These facts indicate that there must be some additional factors other than deficient compensation for the appearance of oscillopsia. Our recent studies of locomotion under reversed vision have shown that failure to detect spatial orientation plays a crucial role in evoking dysequilibrium and motion sickness (6).

In this paper, to clarify the mechanism underlying oscillopsia, we investigated the effects of head perturbation, gaze instability (retinal velocity slip) and upright locomotion on visual perception, body balance, and spatial orientation.

**Methods**

The test conditions in the present study consisted of the following 6 different situations, which have been separately reported: 1) active sinusoidal head rotation on the horizontal plane with an amplitude of ±20 deg at a rate of 0.33, 0.67 and 1.0 Hz (7); 2) passive pendular rotations of the whole body by a motor-driven chair with an amplitude of ±20
deg at a rate of 0.2, 0.33, 0.67, and 0.85 Hz (8); 3) voluntary high-frequency head oscillation in the horizontal plane at frequencies higher than 1 Hz (9); 4) stepping and running on the spot at a rate of 1.0 Hz and 1.5 Hz, respectively (10); 5) passive, vertical oscillation of the standing whole body by an electrically driven platform which was vertically oscillated with a displacement of 1 to 10 cm at a rate of 1, 2, and 3 Hz (11); 6) walking in a park while wearing horizontal or vertical reversing goggles (Dove prisms) (6). Subjects were examined while sitting on a chair in test conditions 1 through 3, and they were asked to gaze at a target on the wall 150 cm away in test conditions 1 through 5.

Except for the examination in a free field (condition 6), head movements were recorded by a gyrosensor which accurately recorded head movements up to 6 Hz. Horizontal eye movements (and vertical movements in test conditions 4 and 5) were recorded by a DC electro-oculograph. These were electrically added to the head movements to obtain gaze recordings. During the stepping and running tests and the vertical platform oscillation tests, linear head movements were recorded by stroboscopic pictures of 10-cm-long fluorescent bars fixed to the front and side of the subject's head. The pictures were taken at the rate of 10 frames a second. Recording was started after the subjects became adapted to the dark (about 10 to 15 minutes later) while gazing at a small red lamp in a semi-dark room. In the active and passive rotation tests, we calculated the ratio of eye velocity to the corresponding head velocity (gain). In the tests of high-frequency head shaking, stepping and running, and vertical whole-body oscillation, we obtained the maximal frequency and velocity of head movement. During the stepping and running tests, forward-backward, right-left, and vertical displacements of the head were measured on the stroboscopic pictures.

During the tests with reversing goggles, subjects were asked to walk in a neighboring park. The locomotion tasks included walking straight, turning in a square, proceeding to a stationary target, and walking on a path. Under the vertical reversal of vision, they were further asked to shake their heads vertically while walking, and to go up and down stairs. Perceptual and locomotional disorders and symptoms of motion sickness were carefully noted.

In all test conditions, perceptual abnormalities of vision and balance were judged by the subject's descriptions of blurred vision and uneasiness or loss of balance. Furthermore, we asked the subjects whether they felt shaking of the ground or the surrounding space. Table 1 summarizes the characteristics of head movements in the test conditions.

### Table 1. Characteristics of Head Rotations or Perturbations under the Present Test Conditions

<table>
<thead>
<tr>
<th>Nature</th>
<th>Movement</th>
<th>Plane</th>
<th>Frequency</th>
<th>Amplitude</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active head rotation</td>
<td>Active</td>
<td>Rotation</td>
<td>Horizontal</td>
<td>0.33-1.0 Hz</td>
</tr>
<tr>
<td>Passive rotation</td>
<td>Passive</td>
<td>Rotation</td>
<td>Horizontal</td>
<td>0.2-0.85 Hz</td>
</tr>
<tr>
<td>High-frequency shaking</td>
<td>Active</td>
<td>Rotation</td>
<td>Horizontal</td>
<td>1.0-5.0 Hz</td>
</tr>
<tr>
<td>Stepping</td>
<td>Active</td>
<td>Rotation</td>
<td>3-dimensional</td>
<td>1.0-2.0 Hz</td>
</tr>
<tr>
<td>Running</td>
<td>Active</td>
<td>Rotation</td>
<td>3-dimensional</td>
<td>1.0-2.0 Hz</td>
</tr>
<tr>
<td>Linear</td>
<td></td>
<td></td>
<td>3-dimensional</td>
<td>1.5-3.0 Hz</td>
</tr>
<tr>
<td>Vertical oscillation</td>
<td>Passive</td>
<td>Rotation</td>
<td>Vertical</td>
<td>1.0-3.0 Hz</td>
</tr>
<tr>
<td>Reversed vision</td>
<td>Active</td>
<td>Random</td>
<td>3-dimensional</td>
<td>Random</td>
</tr>
</tbody>
</table>
Oscillopsia stepping and running, we examined normal adults (n = 35, 35, 27, and 25, respectively), patients with a unilateral loss of labyrinthine function in both early and compensated stages (n = 40, 40, 35, and 40, respectively), and patients with bilateral labyrinthine loss (n = 5, 6, 4, and 5, respectively). In the case of vertical whole-body oscillation, normal subjects (n = 10) and patients with bilateral labyrinthine loss (n = 5) were examined. Loss of labyrinthine function was diagnosed by an absence of response to ice water irrigation. The causes of unilateral labyrinthine loss included surgery for an acoustic neuroma, vestibular neuritis, labyrinthitis, temporal bone fracture, and unknown origin; causes of bilateral loss included streptomycin intoxication, meningitis, renal failure, syphilitic labyrinthitis, and congenital inner ear anomalies (a 28-year-old deaf worker and a 34-year-old deaf fisherman). Gait tests with reversing goggles were examined in normal subjects (n = 10) and in one of the patients with inner ear anomalies.

Results

Active and passive rotations in the horizontal plane. Although sinusoidal rotations at higher frequencies (0.85 Hz–1.0 Hz), both active and passive, did not influence gaze stabilization in normal subjects (Figure 1), they induced a blurring of vision and fluctuations of gaze due to deficient compensation in patients with bilateral labyrinthine loss (Figure 2). During sinusoidal rotations with amplitude A at frequency F, the maximal slip velocity corresponds to \( \pi AF(i - G) \) (G, gain value). The value at 0.85 Hz was 75 deg/sec in the early stage (gain, 0.4) and 25 to 38 deg/sec in the compensated stage (gain, 0.7 to 0.8). Although patients with inner ear anomalies presented similar findings during passive rotations (gain, 0.5 at 0.85 Hz), gaze disturbance was remarkably improved during active rotations (gain, 0.82 at 1.0 Hz)(Figure 2). In patients with unilateral labyrinthine loss, gaze disturbance and blurred vision appeared during rotations to the affected side in the early stage (Figure 1), and quickly recovered with time in most cases; the gain increased linearly on a logarithmic time scale after onset (Figure 3). Patients with bilateral lesions also showed an incomplete recovery of gaze disturbance, the pattern of which resembled that of the unilateral lesions, but with a delayed onset of recovery (Figure 3).

Voluntary head oscillations at high frequencies aggravated gaze disturbance and blurred vision in patients with bilateral lesions (Figure 2). They also revealed gaze disturbance due to deficient compensation in the compensated stages of unilateral lesions. Even normal subjects exhibited oscillations of the gaze recording and complained of blurred vision at frequencies higher than 3 Hz, during which the approximate maximal head velocity was 170 to 200 deg/sec. Patients with bilateral labyrinthine loss and patients with unilateral loss in the early stage found it difficult to achieve high-frequency head oscillations; the
mean peak frequency of head oscillation was 4.0 Hz in normal subjects, 2.3 Hz in patients with unilateral lesions, and 2.0 Hz in patients with bilateral lesions. However, they did not feel an imbalance of the head and body, or shaking of the ground during head oscillations.

**Vertical Whole-Body Oscillations and Upright Locomotion**

During walking and running, head perturbations are characterized by a vertical linear displacement, high-frequency oscillation, and repetitive movements. Stepping at 1 Hz produced horizontal head oscillations at 1 Hz and vertical oscillations at 2 Hz. Similarly, running at 1.5 Hz produced horizontal oscillations at 1.5 Hz and vertical oscillations at 3.0 Hz. In order to estimate the effect of vertical displacement on the head and gaze stabilities during locomotion, we reproduced vertical oscillations of various amplitudes and frequencies with the vertical oscillating platform. At oscillations of 3 Hz, which simulated running, the pitching head amplitude markedly increased as the displacement increased (Figure 4). The mean amplitude
amounted to 8 deg. or more at oscillations of 5 cm at 3 Hz. At that time, the approximate maximal head velocities of pitching motion and vertical displacement, which were calculated from the equation of $\pi AF(A, \text{amplitude}, F, \text{frequency})$ were 76.3 deg/sec and 47.1 cm/sec, respectively, during which not only patients with labyrinthine lesions, but also normal subjects, perceived blurred vision. However, this was not accompanied by a difficulty of balance or oscillation of the surrounding space. Patients described that it differed from oscillopsia upon walking.

During stepping and running tests, rotatory and linear moments to the head resulted in complicated head movements in space. As shown by the three-dimensional schemata indicating the magnitude of rotatory and linear head displacements for a second, the head stability in space was degraded in patients with acquired bilateral labyrinthine lesions (Figure 5). The mean values of horizontal and vertical head rotation amplitudes were $3.5$ ± $1.0$ deg. and $2.1$ ± $0.5$ deg., respectively ($1.7$ ± $1.0$ deg. and $0.3$ ± $0.5$ deg. in normals); the mean right-left linear displacement was $4.5$ ± $1.1$ cm ($1.5$ ± $0.5$ cm in normals). The subjects experienced some blurring of vision and uneasiness of balance. During running, they involuntarily suppressed vertical oscillations.
Locomotions under Horizontal or Vertical Reversed Vision

Horizontal or vertical reversing prisms induced mismatches between the vestibular and visual inputs, and produced image slips of the visual surroundings during upright locomotion. Walking while wearing horizontally reversing prisms soon evoked marked restriction of locomotion with shuffling and inability to walk in normal subjects. Once spatial orientation was confused, they felt as if the internal representation of the space was oscillating or turning even during standstill. Walking only a few steps resulted in marked oscillation of the surrounding space, shaking of the ground, and loss of balance. More than half of the subjects soon (at least within 20 minutes) developed motion sickness. Although the patient with congenital labyrinthine loss exhibited locomotor difficulties with deviations and turnings during the test, he complained of neither oscillation of the space in the brain nor loss of balance.

In contrast, vertical reversal of vision did not induce dysequilibrium, locomotional disturbance, or motion sickness. The subjects could repeat pitching and rolling head movements during the test without any discomfort. Whereas horizontal reversal produced loss of spatial orientation and an illusion of shaking of the ground, such sensations were not provoked during vertical reversal even during running or vertical motions of the head.

Discussion

Oscillopsia, being one of the most annoying symptoms in patients with bilateral labyrinthine loss, seems to be a key in clarifying the strategies that stabilize the head in space and the perceived image in the brain. In the present study, in order to evaluate the roles of head perturbation, retinal velocity slip, and locomotion in the production of oscillopsia, we investigated head and gaze stabilities, and perceptions under various internal and external conditions.

If oscillopsia simply reflects a retinal veloc-
Oscillopsia is typically perceived during walking rather than during horizontal head rotations (12), upright locomotions should provide the factors needed to provoke the sensation. We then took notice of the effect of vertical displacement on the head stability. Although the amplitude of pitching head motion was markedly increased by an increase in the frequency and amplitude of vertical oscillation, passively induced vertical oscillations simulating stepping or running did not produce oscillopsialike sensations in patients with labyrinthine lesions.

In patients with bilateral labyrinthine loss, stepping resulted in large, irregular and often linear head perturbations, which significantly differed from those observed in normal subjects. However, the amplitude of angular oscillation was at most 5.5 deg. (mean ± 2 SD) in the horizontal plane and 3.1 deg. in the vertical plane (11); the amplitude of linear displacement was 6.7 cm (mean ± 2 SD) in the right–left direction. If the head oscillations are nearly sinusoidal, the resultant maximal head velocities are 17.3 to 19.5 deg/sec and 22.3 cm/sec. Even considering the magnification of the perturbations during real walking (13,14), these values may be much smaller than the values obtained during voluntary head movements (15) or vertical whole-body oscillations. Thus, it seems probable that oscillopsia reflects a failure to reproduce the surrounding space in the brain, rather than a magnification of resultant head perturbations or retinal velocity slips. We suspected that upright locomotion without labyrinthine functions induced a breakdown in the strategy of the higher CNS to ascertain spatial orientation, and that the loss of spatial orientation evoked a sensation of oscillating space and disabled the control of body balance. To examine the hypothesis, reversed vision may be a good model, since it artificially produces retinal velocity slips and worsens spatial orientation in normal subjects during upright locomotion.

Horizontal reversal of vision induced visual instability, dysequilibrium, and restriction of head movement. It also produced locomotion ataxia and motion sickness (6,16,17). However, vertical reversal did not induce such phenomena, although retinal image slip occurred similarly in both situations (6). These results suggest that not only stability of the surrounding space, but also an ability to control balance were remarkably affected by the internal representation of the space in the brain. Right–left reversal of vision acted on the perception and locomotion as if it were an acute bilateral loss of labyrinthine function, despite different internal and external situations. Thus, it seems highly probable that a failure to detect spatial orientation may underlie the oscillopsialike sensation, loss of balance, and locomotion ataxia in both situations. As regards the reason why vertical reversal of vision did not deprive the subjects of spatial orientation, we must consider two possibilities: first, that gravity has a stronger influence on vertical orientation than vision (6), and secondly, that vertical orientation is not as indispensable to upright locomotion as horizontal orientation.

We infer that spatial orientation plays the most important role in stabilizing the perceived image of the surrounding space and in controlling head, body, and gaze. Once spatial orientation is confused or lost by erroneous or deficient inputs, disorders in the feedforward regulation may result in gaze disturbance, dysequilibrium, and locomotion ataxia, and, at the same time, failure to identify the self in space may be felt as oscillopsia. The errors of ocular movement and locomotion in the early
stages of unilateral labyrinthine lesions (gaze disturbance and deviations) may reflect asymmetry or distortion of spatial orientations. The state of spatial orientation was affected not only by internal and external situation (for example, labyrinthine loss or reversed vision), but also by actions that imperiled the accurate self-space relationship. Thus, a quick head turn while standing possibly induces loss of balance and oscillopsia in patients with labyrinthine loss, whereas the same action while sitting produced blurred vision alone.

Oscillopsia usually recovers in parallel with an improvement of body balance even in patients with bilateral loss of vestibular function (18,19). The recovery may be accomplished by acquiring a new strategy to reproduce the internal representation of space by other cues that realize the feedforward regulation of the head, gaze, and body. This inference is supported by the finding of large but regular head perturbations, gaze stability, and lack of oscillopsia in the patient with congenital inner ear anomalies.

REFERENCES