Costimulation of the Horizontal Semicircular Canal during Skull Vibrations in Superior Canal Dehiscence Syndrome

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Key Words
Superior canal dehiscence syndrome · Vestibuloocular reflex · Nystagmus · Vertigo · Vibration

Abstract
A sound- and pressure-induced vestibuloocular reflex (VOR) has been described as vertical and torsional in superior canal dehiscence (SCD), and the rotational axes of induced VOR have been assumed to fit with the axis of the affected superior semicircular canal (SC). However, it has been difficult to characterize the pattern of vibration-induced VOR (ViVOR). We aimed to characterize the pattern of ViVOR by comparing the intensity and the axis of VIVOR with several clinical parameters of SCD. Ten symptomatic SCD patients were recruited. SCD size and location were measured on a reformatted image in the plane of the SC. Unilateral vibratory stimulation (100 Hz) was applied to the mastoid surface. ViVOR were recorded using 3D videooculography. The median 3D velocity of ViVOR was measured and the 3D vector trajectory plotted for reference against the axes of the human semicircular canals. A correlation between the magnitude of ViVOR and the size of SCD was evaluated. We also compared the location of SCD with the vertical-to-torsional component ratio of the ViVOR. ViVOR were present in 7 patients; 6 patients showed a substantial horizontal component in the excitatory direction in addition to strong torsional and weak vertical components. The computed rotational axes of ViVOR were located mostly between the axes of the ipsilateral SC and horizontal canal (HC) with a variable deviation to the axis of the ipsilateral posterior canal (PC). The magnitude of ViVOR was not related to the size of the SCD. The vertical-to-torsional component ratio of ViVOR tended to decline as the dehiscence was closer to the common crus. In SCD, mastoid vibration may stimulate the affected-side HC and PC as well as the SC. SCD can be suspected when excitatory horizontal torsional ViVOR direct to the side of the auditory symptoms.

Introduction
Superior canal dehiscence (SCD) causes cochleoves-tibular hyperresponsiveness to various stimuli due to the presence of a third labyrinthine window in the superior semicircular canal (SC). Patients with SCD syndrome present with auditory and vestibular symptoms including autophony, hyperacusis, oscillopsia and vertigo induced by loud sounds or pressure [Aw et al., 2006; Brantberg et al., 2006; Minor et al., 1998].

Simple pressure stimulation of the middle ear or an increase in intracranial pressure can evoke a typical eye movement in SCD patients [Minor, 2005; Minor et al., 1998; Robinson, 1963]. A sound- and pressure-induced
vestibuloocular reflex (VOR) in patients with SCD has been described as vertical and torsional, and the rotational axes of induced VOR, computed by vector analysis, have been assumed to fit with the axis of the affected SC. In patients with left SCD, sound or pressure stimulates the left SC and the left superior rectus and right inferior oblique muscle, consequentially. This results in an upward and counterclockwise eye movement. Similarly, in right SCD, an upward and counterclockwise VOR can be induced by stimulation of the right SC.

Skull vibration stimulates whole vestibular end organs in both ears and horizontal torsional nystagmus may develop in the presence of vestibular asymmetry [Hamann and Schuster, 1999; Karlberg et al., 2003; Lackner and Graybiel, 1974; Michel et al., 2001]. The occurrence of nystagmus induced by skull vibration was reported to range from 72 to 100% in unilateral peripheral vestibulopathy [Hong et al., 2007; Koo et al., 2011; Ohki et al., 2011; Ohki et al., 2003]. It usually beats to the healthier side during vibration and is assumed to be the result of asymmetry in the excitatory functional activity of the peripheral vestibular system [Dumas et al., 2004; Koo et al., 2011; Ohki et al., 2003].

Vibration-induced VOR (ViVOR) testing is a simple and readily available method for the evaluation of vestibulopathy. It has also been tested in patients with SCD [Aw et al., 2011; Manzari et al., 2008; White et al., 2007]. Previous studies using vibration stimuli demonstrated relatively stronger downbeat torsional nystagmus with a few or absent horizontal components in most cases, which is apparently different from the nystagmus in unilateral vestibular loss patients [Aw et al., 2011; Manzari et al., 2008; White et al., 2007]. Skull vibration, however, also unpredictably induced either excitatory or inhibitory nystagmus in some SCD patients [Manzari et al., 2008].

We observed that sound-induced VOR were predominantly torsional and upward, with relatively rare horizontal components in SCD patients. Mastoid vibration, on the other hand, using a commercially available hand-held vibrator, induced substantial horizontal VOR that were in the excitatory direction and had a smaller vertical component. In our previous case report, describing a symptomatic case of SCD with superior petrosal sinus, tone-burst stimulation to the left, affected ear evoked mainly clockwise torsional nystagmus with little vertical eye movement, while mastoid vibration induced considerable left-beating nystagmus with a counterclockwise torsional component [Koo et al., 2010]. We suggested that the cause of atypical induced VOR by tone-burst stimulation in that patient was the location of the SC, which was closer to the common crus rather than at the top of the SC, facing the middle fossa dura. That is, both the SC and posterior semicircular canal (PC) were activated at the same time, and this resulted in the torsional components summing while the vertical components largely canceled each other out. However, the horizontal component was not clearly explained.

In this study, we recorded ViVOR in patients with SCD syndrome and analyzed patterns and the axes of eye movements to evaluate the pattern of the ViVOR in SCD patients. We also sought to determine whether ViVOR were correlated with the size and location of SCD.

**Subjects and Methods**

**Patients**

We retrospectively recruited 10 patients with symptomatic SCD who were diagnosed as having SCD syndrome at Seoul National University Bundang Hospital from March 2005 to March 2012. The diagnosis of SCD syndrome was based on: (1) symptoms related to increased cochleovestibular responsiveness, presenting with autophony and hyperacusis, the Tullio phenomenon, the Hennebert sign and disequilibrium; (2) visible SCD on high-resolution temporal-bone computed tomography (HR-TBCT); (3) an intact stapedial reflex; (4) a lower threshold in cervical vestibular evoked myogenic potential (cVEMP), compared with the healthy side, by 15 dB or more; and (5) exclusion of central pathologies, using a neurological and radiological examination and brain MRI. The patients consisted of 6 males and 4 females with ages ranging from 37 to 50 years; 9 patients had unilateral SCD (3 right, 6 left) and 1 had bilateral SCD. This study was approved by the institutional review board of Seoul National University Bundang Hospital (IRB No. B-1210-174-104). It was conducted according to the tenets of the Declaration of Helsinki.

**Clinical Evaluation**

We reviewed cochleovestibular symptoms including the Tullio phenomenon, the Hennebert sign, disequilibrium and auditory symptoms, such as autophony, hyperacusis and ear fullness, at presentation. All patients underwent audiovestibular evaluations at the initial visit. Nystagmus was observed on a video monitor without fixation, using video Frenzel goggles (Easy Eye; SLMED, Seoul, Korea). Spontaneous and gaze-evoked nystagmus was assessed. Head-shaking nystagmus was assessed using a passive head-shaking maneuver [Choi et al., 2007b]. Bedside head impulse tests were performed manually [Halmagyi and Curthoys, 1998]. Sound- and pressure-induced nystagmus was also evaluated.

**Audiometry**

Hearing levels were determined by pure-tone audiometry and speech audiometry. The bone conduction threshold and the air-bone gap at low frequencies (250 and 500 Hz) were evaluated. Tympanometry and stapedial reflex assessments were also performed.

**Cervical Vestibular Evoked Myogenic Potentials**

VEMP were recorded with surface electrodes on the ipsilateral sternocleidomastoid muscle during contralateral neck rotation in a supine position. Alternating tone bursts (500 Hz; rate: 2.1/s; rise-
Vibration-Induced VOR in SCD Syndrome

Fig. 1. Measurement of the size and location of SCD. Coronal (a) and reconstructed oblique image by HR-TBCT in the plane of the SC (b). a A bony dehiscence of the left superior canal facing middle cranial fossa dura is indicated (arrow). b SCD size (black solid line) and a location (black dotted line, distance from the canal side of SC ampulla to the proximal dehiscence site) were measured on the reformatted image.

Radiological Evaluation

HR-TBCT was performed using 64-channel and 256-channel multidetector row CT scanner (Brilliance 64 and iCT 256, Philips Healthcare, Cleveland, Ohio, USA). Helical scans were performed with a tube voltage 120 kVP and 149 mA with detector collimations of 40 × 0.625 mm at 64 channel, and 20 × 0.625 mm at 256 channel. Axial and coronal images were reconstructed with 0.7-mm slice thickness and 0.7-increment, respectively. The images were displayed on an INFINITT PACS system (version 3.0.9.1BN9; INFINITT Healthcare, Seoul, Korea) and 3D multiplanar reconstruction was subsequently used to obtain an oblique coronal reformatted image parallel to the SC (fig. 1). The length of the arc of the dehiscence (SCD size) was measured on a reformatted image in the plane of the SC. To estimate the proximity of the dehiscence to the common crus, the length of the arc of the dehiscence was added up to the length from the SC ampulla to the proximal dehiscence site (fig. 1b).

Analysis of Sound- and Vibration-Induced VOR

The VOR was recorded in a seated position in a dark room. For evaluation of the sound-induced VOR, 500-Hz short tone bursts (stimulation rate: 3.1/s; rise-fall time: 2 ms; plateau time: 3 ms; intensity: 93 dB nHL) were generated digitally, using a Madsen Aurical PC-based audiometer (GN Otometrics, Taastrup, Denmark) and delivered through an E-A-R 3A insert earphone (10 Ω; Kim, 2012; Lee et al., 2009). Plotted rotation axes were compared with the semicircular canal axes.

The axes orthogonal to the average human semicircular canal planes [della Santina et al., 2005] were defined as the semicircular canal axes. Because the nystagmus was from inhibition of the semicircular canals, the rotation axis of the nystagmus was inverted [Choi et al., 2007a, b; Lee et al., 2009]. The mean rotation axis for each nystagmus was analyzed offline and plotted in 3D [Kim and Kim, 2012; Lee et al., 2009]. Plotted rotation axes were compared with the semicircular canal axes.

The eye velocity (x, y, z) of each component (T, V, H) of VOR was characterized by magnitude and direction. Thus, the rotation axis of each VOR can be plotted in 3D using this eye velocity information [Aw et al., 2005; Choi et al., 2007b], and if the intensity of the right-side and left-side stimulation was different, the bigger elicited VIVOR was used for the analysis. Then, the rotation axis of each nystagmus was analyzed offline and plotted in 3D [Kim and Kim, 2012; Lee et al., 2009]. Plotted rotation axes were compared with the semicircular canal axes.

Nystagmus was recorded using 3D videoculography at a sampling rate of 60 Hz (SensoMotoric Instruments, Teltow, Germany). We selected the most consistent trial of the recorded data among the multiple trials and the induced VOR in the selected trial were averaged. Rightward, upward and clockwise directions from the patient’s view were positive for horizontal (H), vertical (V) and torsional (T) eye rotations. The digitized eye position data were analyzed using the MATLAB software [Choi et al., 2007b]. The eye velocity (x, y, z) of each component (T, V, H) in the induced VOR was calculated individually from 3D eye position data. The magnitude of the eye velocity of the induced VOR was calculated as

$$\sqrt{x^2 + y^2 + z^2}.$$

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Statistical Analyses

Spearman’s correlation was used to evaluate the correlation between the magnitude of the VIVOR and the size of the SCD. It was also used to compare the distance between the ampulla of the SC and the dehiscence site and a vertical-to-torsional component ratio of the VIVOR. The Mann-Whitney U test was used to compare differences in eye rotation axis between vibration and sound stimulation. Statistical analyses were performed using the SPSS software (version 18.0 for Windows). The criterion for statistical significance was set at p < 0.05.
Results

Clinical Assessment

All patients presented with vestibular and cochlear symptoms and signs, including disequilibrium, vertigo induced by loud sound or pressure, autophony and hyperacusis. All patients showed an air-bone gap at 250 Hz in pure-tone audiometry. Nine of the total of 11 ears (81.8%) presented with a lowered bone conduction threshold below 0 dB. VEMP thresholds for the affected ears ranged from 13 to 68 dB, and they were lower by 15–65 dB, compared with the healthy side, in unilateral SCD. VEMP thresholds for the 1 bilateral SCD case were 68 dB on right side and 58 dB on left side. Demographics and individual clinical presentations are summarized in Table 1.

Radiological Evaluation

The mean (±SD) size of the SCD in the reformatted plane of the SC was 4.0 (±1.3) mm, on average (range: 1.7–6.4 mm). The SCD was located at the arcuate eminence in 9 patients (10 sides) and at the level of the superor petrosal sinus in 1 patient. The mean length from the SC ampulla to the proximal dehiscence site was 3.6 mm (table 2).

<table>
<thead>
<tr>
<th>Case No.</th>
<th>ViVOR (assuming left SCD)</th>
<th>V-T ratio</th>
<th>SC ampulla to dehiscence site, mm</th>
<th>SCD size, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2 R (0.2)</td>
<td></td>
<td>5.2</td>
<td>4.7</td>
</tr>
<tr>
<td>2</td>
<td>16 R (1.8)</td>
<td></td>
<td>3.1</td>
<td>4.7</td>
</tr>
<tr>
<td>3</td>
<td>3.7 R (2.3)</td>
<td>4.3 U (1.1)</td>
<td>0.4</td>
<td>4.2</td>
</tr>
<tr>
<td>4</td>
<td>1.2 R (0.3)</td>
<td>1.6 U (0.4)</td>
<td>–</td>
<td>4.1</td>
</tr>
<tr>
<td>5</td>
<td>–4.4 R (1.6)</td>
<td>4.5 U (0.9)</td>
<td>3.8 CW (1.8)</td>
<td>4.0</td>
</tr>
<tr>
<td>6</td>
<td>–</td>
<td>–</td>
<td>6.8</td>
<td>3.7</td>
</tr>
<tr>
<td>7</td>
<td>23.4 R (3.6)</td>
<td>25.5 U (2.6)</td>
<td>26.4 CW (4.9)</td>
<td>3.5</td>
</tr>
<tr>
<td>8</td>
<td>9.4 R (1.8)</td>
<td>–2.6 U (1.3)</td>
<td>17.1 CW (5.6)</td>
<td>2.1</td>
</tr>
<tr>
<td>9</td>
<td>3.8 R (1.1)</td>
<td>3.1 U (1.4)</td>
<td>7.6 CW (2.1)</td>
<td>1.7</td>
</tr>
<tr>
<td>10</td>
<td>6.5 R (1.1)</td>
<td>1.8 U (0.9)</td>
<td>4.9 CW (1.4)</td>
<td>6.4/4.7</td>
</tr>
<tr>
<td>Mean VOR</td>
<td>8.3 R (9.1)</td>
<td>5.2 U (9.3)</td>
<td>11.3 CW (7.9)</td>
<td></td>
</tr>
</tbody>
</table>

Values in parentheses denote SD. R = Rightward; U = upward; CW = clockwise; V-T ratio = ratio of vertical-to-torsional component.

Table 1. Demographics and clinical assessment of 10 patients with SCD syndrome

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex/age, years</th>
<th>Imaging</th>
<th>Clinical vestibular and cochlear assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>SCD side</td>
<td>SCD size, mm</td>
</tr>
<tr>
<td>1</td>
<td>F/37</td>
<td>L</td>
<td>4.7</td>
</tr>
<tr>
<td>2</td>
<td>M/42</td>
<td>R</td>
<td>4.7</td>
</tr>
<tr>
<td>3</td>
<td>F/50</td>
<td>R</td>
<td>4.2</td>
</tr>
<tr>
<td>4</td>
<td>M/45</td>
<td>L</td>
<td>4.1</td>
</tr>
<tr>
<td>5</td>
<td>F/37</td>
<td>L</td>
<td>4.0</td>
</tr>
<tr>
<td>6</td>
<td>M/38</td>
<td>R</td>
<td>3.7</td>
</tr>
<tr>
<td>7</td>
<td>F/48</td>
<td>L</td>
<td>3.5</td>
</tr>
<tr>
<td>8</td>
<td>F/47</td>
<td>L</td>
<td>2.1</td>
</tr>
<tr>
<td>9</td>
<td>M/41</td>
<td>L</td>
<td>1.7</td>
</tr>
<tr>
<td>10</td>
<td>M/45</td>
<td>R/L</td>
<td>6.4/4.7</td>
</tr>
</tbody>
</table>

NR = No response.

Table 2. ViVOR in SCD patients

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Vibration-Induced VOR

ViVOR were present in 9 of the 10 patients. Among them, 2 patients showed SPV of each component smaller than 3°/s and were excluded from the analysis. The ViVOR was in the excitatory direction in 6 patients, and there was a relatively stronger torsional component with a variably weaker upward vertical component (table 2). The median 3D velocity of the vertical, horizontal and torsional position was plotted for reference against the axes of the human semicircular canals [della Santina et al., 2005]. The rotational axis of each ViVOR was not aligned to the axis of the affected-side SC, while that of tone-burst-induced VOR was aligned closer to the SC (fig. 2). Most were located between the axes of the horizontal canal (HC) and the SC. The mean rotational axis in all 7 patients who presented with ViVOR was also plotted between the axes of the HC and the SC, with the exception of case No. 1 (fig. 3, black line).

Case No. 3 (green line) had an SCD closer to the common crus and showed smaller inverted vertical components with an increased torsional component. Sound-induced VOR were presented in 3 of 9 unilateral SCD patients (cases No. 5, 7 and 8), while ViVOR were seen in 7 patients. The mean eye rotation axis (x, y, z) was 0.86/0.35/0.05 in sound-induced VOR and 0.70/0.30/0.34 in ViVOR by comparison with the normalized vector. There was a significant difference in the horizontal vector (z; p = 0.036), but no difference in the vertical (y) or torsional (x) vector. The ViVOR eye rotation axis showed a 20.7-degree vector rotation from those of sound-induced VOR toward the z-axis. In the 1 case of bilateral SCD, the torsional, vertical and horizontal components of the ViVOR were 6.5°/s rightward, 1.8°/s upward and 4.9°/s clockwise, respectively (table 2). Theoretically, vertical and torsional components would be weaker by cancelling each ear out, and the verti-

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**Fig. 2.** 3D videooculographic recording and rotation axis analysis of tone burst and ViVOR in patient No. 5 having left SCD. Note that tone-burst stimulations induced subtle horizontal VOR (a), while mastoid vibration induced substantial rightward ViVOR (b). The rotation axes of the 500-Hz tone burst are aligned closer to the left superior canal, while those of vibration lie between the left SC and HC (c). a Tone burst (500 Hz, 3.1/s). b Mastoid vibration (100 Hz). c Rotation axis (green: 500 Hz tone burst; red: vibration). LS = Left SC; LH = left HC; LP = left PC.
cal components would be stronger by adding up to the stimulation of both superior canals at the same time. Thus, it seemed as if it was a left unilateral SCD, although the measured dehiscence size was smaller in the left SC.

**Size of SCD**

The magnitude of the ViVOR was not correlated with the size of the SCD in 6 patients who presented with pathological ViVOR, except in the bilateral case (p = 0.957; fig. 4). There was no significant correlation of the magnitude of ViVOR with size in any plane.

**Location of SCD**

The vertical-to-torsional component ratio of ViVOR showed a tendency toward decline as the distance from the ampulla to the distal site of the dehiscence (length from SC ampulla to proximal dehiscence site + SCD size) increased. However, this was statistically nonsignificant (p = 0.085; fig. 5).

**Fig. 3.** Rotational axes of the ViVOR of 7 patients with SCD (different colors), assuming the lesion is on the left side (a). The axes of the semicircular canal in blue were calculated from the data by della Santina et al. [2005]. The axis of each ViVOR lies between axes of the HC and the SC (b), with the exception of 1 case (red line). The mean rotational axis (black line) was not aligned with the axis of the lesioned SC and was between the HC and SC. LS = left SC; LH = left horizontal semicircular canal; LP = left PC.

**Fig. 4.** Relationship between magnitude of ViVOR and size of SCD (p = 0.957). The magnitude of the ViVOR and SCD size is shown in each case.
Discussion

A diagnosis of SCD may be suspected in patients presenting with typical symptoms such as the Tullio phenomenon, pressure-induced vertigo and autophony with ipsilateral conductive hearing loss. To confirm the diagnosis of SCD, it is important to demonstrate not only anatomical dehiscence of the SC on HR-TBCT, but also hyperresponsiveness of labyrinthine receptors using certain laboratory tests (essentially, VEMP with a threshold analysis [Brantberg et al., 1999; Halmagyi et al., 2005] and typical nystagmus induced by sound and pressure). VEMP demonstrate a peculiar response with increased amplitude and a lowered threshold. Pressure stimulation of the middle ear or an increase in intracranial pressure can evoke a typical vertical and torsional VOR [Minor, 2005; Robinson, 1963]. However, typical sound- or pressure-induced vertigo may not be present and the induced nystagmus may be too subtle to detect in most SCD patients.

Mastoid vibration provides valuable bedside information by lateralizing the lesion side in patients with unilateral peripheral vestibular loss [Hamann and Schuster, 1999; Karlberg et al., 2003; Koo et al., 2011; Lackner and Graybiel, 1974]. This was also true in SCD patients in our study and previous reports. Aw et al. [2011] also advocated the efficacy of ViVOR for identifying unilateral or bilateral vertical semicircular canal dehiscence. The ViVOR was consistent with excitation of the affected-side SC and is evoked in ossicular chain dysfunction, even when air-conducted click VOR was absent or markedly reduced. In our series, 7 of the 10 patients showed substantial ViVOR. However, their rotation axes were not aligned with that of the affected SC stimulation. Rather, they clustered between the axes of the HC and SC, except in 1 patient (patient No. 5; red line), because they were accompanied by substantial horizontal components (fig. 3), reflecting costimulation of the HC during mastoid vibration in SCD patients. The axes of sound- or pressure-induced VOR nearly matched the axis of the affected SC. This indicates that the HC is stimulated simultaneously with the SC with vibration on the mastoid. The presence of horizontal components during vibration using a hand-held vibrator was also described in previous studies, although the intensities were variable. White et al. [2007] reported that 2 of 4 unilateral SCD patients, who were confirmed by anatomical and functional studies, showed strong horizontal nystagmus – 8°/s and 20°/s, respectively – beating to the SCD side during vibration stimulation, while mastoid vibration unpredictably induced either excitatory or inhibitory horizontal nystagmus [Manzari et al., 2008] or the SPV of the horizontal component of the nystagmus was negligible [Aw et al., 2011]. We also observed inhibitory horizontal nystagmus in 1 patient and negligible in 2 of the 10 subjects. Vestibular and neck muscle spindle afferents could be variably stimulated by mastoid vibration, depending on the type of vibrator and the characteristics of the vibration stimuli (such as frequency, size of contact area, pressure over the contact area, battery-powered or AC-driven). Mastoid or skull vibration also induces variable amounts of eye movement, although mostly weak nystagmus, even in healthy subjects without SCD, depending on the area of vibration and type of vibrator [Aw et al., 2011; Park et
If the vibration stimulation in SCD were sufficiently strong to overcome naturally occurring VIVOR, the direction would be more consistent, as in our series, so long as there is no other accompanying vestibulopathy. The lowered bone conduction threshold in SCD patients may be explained by increased oscillating compression and rarefaction of inner ear fluid in the presence of the third window when applying a bone vibrator over the mastoid tip. Similarly, skull vibration may also increase inner ear fluid motion in the semicircular canals and the fluid motion in the SC can be greater than that in the HC or PC because the third window is located in the SC. However, it seems that the rotation axes of VIVOR showed excitation of the affected-side SC plus HC in our study. Costimulation of the HC in our study may have been due to our use of a different skull vibrator from those in previous studies in terms of the size of the contact surface, its vibrating frequency and, especially, its vibrating power, although it could not be precisely controlled. The direction of the horizontal component of VIVOR would be excitatory because the excitatory response is stronger than the inhibitory response in the presence of identical cupular deflection by vibration-induced fluid oscillation through the cupula (Ewald’s second law) [Balogh et al., 1977]. The vibrator used in this study was a commercially available and readily accessible device, which is appropriate for office examinations with Frenzel goggles. The nystagmus was sufficiently strong to be visible under Frenzel goggles, while the excursion of the eyeball observed in the study by Aw et al. [2011], by bone oscillator, was at best 0.2°, which could not be recorded without a magnetic search coil system. Thus, the methods in our study can be considered more practical, providing adjuvant information to assist diagnosis of SCD syndrome; that is, the lesion side can be suspected when fast components of VIVOR direct to the side of autophony and low-frequency conductive hearing loss.

The direction of the vertical component of VIVOR distinguishes posterior canal dehiscence from SCD: it is downward in posterior canal dehiscence and upward in SCD [Aw et al., 2011]. Our cases showed mainly upward VOR, as expected. However, the relative amount of the vertical-to-torsional component was variable, and we assumed the possibility of variable costimulation of the PC as well by the vibration stimuli, depending on the proximity of the dehiscence to the common crus. As the length of the arc from the SC ampulla to the distal dehiscence site (common crus side) increased – that is, the distal site of the dehiscence came closer to the common crus – the vertical-to-torsional component ratio of the VIVOR showed a tendency toward decrease, though not statistically significantly so. In case No. 8, the VIVOR showed a small inverted vertical component with an increased torsional component, compared with other patients, and the rotation axis was between the SC and PC. This resulted from costimulation of the PC in addition to the SC, considering that the location of the dehiscence by the superior petrous sinus was closer to the common crus than to the superior canal ampulla in this patient. Simultaneous stimulation of the left SC and PC would generate rather pure torsional eye movements because the vertical components would cancel each other out [Koo et al., 2010]. One case with a 4.7-mm SCD (case No. 2) showed no vertical component in the VIVOR. Although the distance of the SC ampulla to the proximal dehiscence site was only 3 mm, the other end of the dehiscence (distal dehiscence site) came closer to the common crus. This, in addition to the large size of the dehiscence, may have caused costimulation of the PC, resulting in no vertical component. There is also the possibility of missing a very thin bony labyrinth that would be hard to detect given the current resolution of HR-TBCT, so the size and the location of the dehiscence on TBCT may differ from the actual dehiscence. For this reason, we may roughly anticipate the location and extent of the SCD by estimating the vertical-to-torsional component ratio of the VIVOR.

There was no relationship between magnitude of VIVOR and size of SCD. Pfammatter et al. [2010] divided 27 patients based on their symptoms and signs and evaluated the association between the symptoms/signs and the size of the SCD. They reported that the patient group with a larger-size SCD (≥2.5 mm) presented predominantly with cochleovestibular symptoms and/or signs, lower VEMP thresholds and objective vestibular findings. We had only 2 cases with smaller sizes (<2.5 mm); however, they also presented with both cochleovestibular symptoms and signs, and definite lower VEMP thresholds. No relationship between size of SCD and other clinical factors was demonstrated.

Conclusions

Mastoid vibration induced nystagmus in patients with SCD. In view of our findings, it is apparent that the rotation axes of VIVOR showed an excitation of the affected-side SC in addition to the HC, with variable costimulation of the PC. If patients who have unilateral ear fullness and autophony with unexplained dizziness and the Tullio phenomenon, the diagnosis of SCD syndrome in that ear
is strongly suggested when fast components of ViVOR direct to the side of the ear symptoms, because the fast component of ViVOR usually directs to the contralateral side (paralytic nystagmus) in unilateral vestibulopathy. The magnitude of the pathologic VOR did not correlate with the size of the dehiscence. The vertical-to-torsional component ratio of ViVOR seems to vary depending on the proximity of the dehiscence to the common crus. The ViVOR is an easy, tolerable and useful auxiliary method for the clinical diagnosis of SCD.

References


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