Extent of Lesions in Idiopathic Sudden Hearing Loss With Vertigo

Study Using Click and Galvanic Vestibular Evoked Myogenic Potentials

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Objective: To clarify the extent of the vestibular lesions in idiopathic sudden hearing loss with vertigo using vestibular evoked myogenic potentials (VEMPs) in response to click (click-VEMP) and galvanic (galvanic-VEMP) stimulation, as well as caloric tests.

Design: Retrospective study.

Setting: University hospital.

Patients: We enrolled 22 patients with idiopathic sudden hearing loss with vertigo in this study. All patients underwent audiometry and click-VEMP and caloric tests. Eight patients underwent a galvanic-VEMP test.

Results: Among the 22 patients, 17 (77%) showed an absence of click-VEMPs on the affected side. In response to caloric testing, 10 patients (45%) showed a decreased response on the affected side. All 8 patients who underwent galvanic-VEMP testing showed normal responses. Most patients with decreased caloric responses (9 [90%] of 10 patients) showed an absence of click-VEMPs, whereas 9 (53%) of the 17 patients who showed abnormal click-VEMPs showed decreased caloric responses. Initial hearing level and hearing outcome significantly correlated with abnormalities on the vestibular test results.

Conclusions: The lesion site of vestibular disorders in idiopathic sudden hearing loss with vertigo appeared to be within the labyrinth on the basis of galvanic-VEMP findings. Results of the click-VEMP and caloric tests suggested that the saccule could be involved more frequently than the semicircular canals. The combined use of click-VEMP and caloric tests is useful for evaluating vestibular functions in idiopathic sudden hearing loss with vertigo because the extent of vestibular abnormalities correlated well with hearing outcome.


DIOPATHIC SUDDEN HEARING LOSS (ISHL) is commonly defined as a severe sensorineural hearing loss of sudden onset and unknown etiology. The annual incidence of ISHL is estimated at approximately 10 cases/100 000 population. Although a number of different pathologic processes might result in sudden hearing loss, it is widely believed that viral infection, vascular obstruction, and cochlear membrane breaks account for most ISHL cases. Of these, a viral cause is thought to be the most common.

About 30% to 40% of patients with ISHL (hereafter referred to as ISHL patients) have accompanying vertigo. It has been reported that vertigo appears more frequently in association with profound hearing loss and that hearing recovery is worse in patients with than without vertigo. Vestibular functions in ISHL patients have been evaluated by means of caloric testing in several studies. About 40% of the ISHL patients with accompanying vertigo showed reduced caloric responses in the affected ear. However, the lesion site causing vestibular symptoms in ISHL other than the lateral semicircular canal remains unknown.

Vestibular evoked myogenic potentials (VEMPs) stimulated with clicks (click-VEMPs) on the sternocleidomastoid muscle have been used as a clinical test of the vestibular system. Clinical and neurophysiological studies have suggested that these are generated by activation of saccular afferents. Click-VEMPs have been used as a clinical test of the saccular afferents, whereas the caloric test has been used as a clinical test of lateral semicircular canal afferents. Com-
bined use of the click-VEMP and caloric tests has facilitated more precise examination of the function of the vestibular apparatus. Furthermore, VEMPs evoked by short-duration galvanic stimulation (galvanic-VEMPs) have been reported to be useful for differentiating labyrinthine lesions from nerve lesions in patients with an absence of click-VEMPs.13-15

We studied the lesion site causing vestibular symptoms in ISHL with vertigo using click-VEMP, galvanic-VEMP, and caloric tests.

METHODS

Twenty-two patients (age range, 21-74 years; median age, 54 years) who were referred to our dizziness clinic and diagnosed as having ISHL with vertigo between January 1, 1999, and December 31, 2003, were enrolled in this study. All patients were seen within 10 days of the onset of hearing loss. The diagnostic criteria for ISHL with vertigo included a more than 30-dB sensorineural hearing loss occurring in at least 3 contiguous frequencies in less than 3 days,16 a single attack of rotatory vertigo occurring almost simultaneously with the onset of hearing loss, and no other neurological signs. Those patients who had multiple attacks of vertigo were excluded from the study to rule out Meniere’s disease or migraine. Those patients who had nonvestibular dizziness such as orthostatic hypotension were also excluded from the study. The durations of the vertigo attacks of all patients ranged from 30 minutes to 5 days (median, 24 hours). Twenty-one (95%) of all patients had associated nausea, and 13 (59%) had associated vomiting. A detailed medical history was obtained in all patients, who also underwent a battery of tests, including physical examination, neurological examination, pure-tone audiometry, blood examination, plain radiographic examinations (mastoid and internal auditory meatus), electronystagmography, caloric test, and click-VEMP test. Eight patients also had received a galvanic-VEMP test. When no cause of the sudden hearing loss and vertigo could be found, the disorder was considered.

All patients were treated with steroids, cyanoacibalin (vitamin B12), and adenosine triphosphate. Steroid treatment consisted of hydrocortisone sodium succinate (500 mg/d, with the dose decreased every other day by 200 mg) for 14 patients and prednisone (30 mg/d, with the dose decreased every 3 days by 10 mg) for 8 patients.

Audiograms were categorized as high- or low-tone hearing loss, flat type, and profound hearing loss. The group with high-tone hearing loss was defined as those patients with an average loss of 4 to 8 kHz, surpassing the average of 0.25 to 0.5 kHz by 30 dB or more. The low-tone hearing loss group demonstrated an average loss of 0.25 to 0.5 kHz, surpassing the average of 4 to 8 kHz by 30 dB or more. The flat-type group consisted of patients with a difference between the worst and best hearing levels of 20 dB or less among 6 frequencies of 0.25, 0.5, 1, 2, 4, and 8 kHz. In the group with profound hearing loss, at least 2 frequencies produced results that were off the scale, and the difference between the hearing level and the maximum sound level generated by the audiometer was within 10 dB at all 6 frequencies.

Follow-up audiograms were obtained several weeks after the treatment in each case. Recovery of hearing in the affected ear was evaluated using the unaffected ear as the baseline for the affected ear. Pure-tone average was calculated as the average threshold at 500, 1000, and 2000 Hz. Complete recovery was defined as recovery of hearing to within 10 dB of the unaffected ear. Partial recovery was defined as recovery of hearing within 50% or more of the average pure-tone score of the unaffected ear. No recovery was defined as less than 50% recovery of hearing.

The click-VEMP, galvanic-VEMP, and caloric tests were performed within 1 month of the onset of hearing loss. The methods for recording click-VEMPs and galvanic-VEMPs are described elsewhere.11,17 Briefly, surface electromyographic activity was recorded in a supine patient from symmetrical sites over the upper half of each sternocleidomastoid muscle, with a reference electrode on the lateral end of the upper sternum. During recording, the patients were instructed to continuously contract the sternocleidomastoid muscle. Electromyographic activities were monitored on a display during recording to maintain muscle activity at a constant level in each patient. The electromygographic signal from the stimulated side was amplified and bandpass filtered (20-2000 Hz). To record click-VEMPs, rarefaction clicks (0.1 milliseconds, 95-dB normal hearing level) were presented through a headphone (type DR-531; Elega Acous Co Ltd, Tokyo, Japan). To record galvanic-VEMPs, a 3-mA (1-millisecond) galvanic stimulation was presented through the electrodes on the mastoid (cathode) and the forehead (anode). The stimulation rate was 5 Hz, and the analysis time was 50 milliseconds. Responses to 100 stimuli were averaged twice.

We analyzed the amplitude of the first positive-negative peak (p13-n23) for click-VEMPs and p1.3g-n23g for galvanic-VEMPs ipsilateral to the stimulated ear and the latencies of the peaks.9 The average of 2 runs was taken for the amplitude and latencies.

For the evaluation of amplitude, the percentage of VEMP asymmetry was calculated as 100[(Au−Aa)/(Aa+Au)], where Au is the amplitude of the p13-n23 and the p13g-n23g on the unaffected side and Aa is the amplitude of the p13-n23 and the p13g-n23g on the affected side. The mean±SD VEMP asymmetry of healthy control subjects at our laboratory was 12.5±8.7 for click-VEMP and 17.8±10.5 for galvanic-VEMP. Hence, the upper limit of VEMP asymmetry was defined as 29.9 for click-VEMPs and 37.8 for galvanic-VEMPs. The mean±SD latencies of click-VEMPs were 11.8±0.86 milliseconds for p13 and 20.8±2.2 milliseconds for n23. The mean±SD latencies of galvanic-VEMPs were 10.9±1.0 milliseconds for p13g and 18.8±2.4 milliseconds for n23g. Hence, the upper limits of the latencies were defined as 13.5 milliseconds for p13, 25.2 milliseconds for n23, 12.9 milliseconds for p13g, and 23.6 milliseconds for n23g.14,17

Caloric nystagmus was recorded using an electronystagmograph. Canal paresis was calculated using the maximal slowphase eye velocity of caloric nystagmus. Canal paresis of greater than 20% was defined as abnormal.

For statistical analyses, nonparametric tests were used because of the small number of each group. Kruskal-Wallis analysis of variance on ranks was used for comparison among the groups, and Spearman rank correlation tests were used to examine possible correlation between variables and types of vestibular lesions. P values of less than .05 were considered significant.

RESULTS

Clinical characteristics of the patients are presented in the Table. Of 22 patients, 14 (64%) were men and 8 (36%) were women. The right ear was involved in 11 patients (50%), and the left ear was involved in 11 (50%). Among the 22 patients, 17 (77%) showed an absence of click-VEMPs on the affected side, whereas all patients showed normal responses on the unaffected side. On caloric testing, 10 patients (45%) had decreased ca-
The median values of initial pure-tone average on the affected sides were 43.3 dB in the C type, 70 dB in the C + O type, and 115 dB in the C + O + S type. We assumed the rank of severity concerning vestibular organ involvements as C type, followed by C + O type, with C + O + S type as the most severe, and sought a correlation between variables and the type ranking. There was significant correlation between the initial pure-tone average and the type ranking (Spearman rank correlation test, r = 0.51; P = .02).

The types of audiograms of the 22 patients were as follows: high-tone hearing loss in 12 (55%), profound hearing loss in 6 (27%), flat-type audiogram in 3 (14%), and low-tone hearing loss in 1 (5%). All 4 patients in the C–type group showed high-tone hearing loss, whereas 4 (50%) of the 8 patients in the C + O–type group and 4 (44%) of 9 patients in the C + O + S–type group showed high-tone hearing loss. The percentage of profound hearing loss was largest in the C + O + S–type group (5/9 patients [56%] compared with 0/3 patients in the C–type group and 1/8 patients [13%] in the C + O–type group).

The hearing outcomes of each group are shown in Figure 4. Among the 22 patients, 4 (18%) showed complete recovery. Four patients (18%) showed partial recovery. The remaining 14 patients (64%) showed no recovery. When complete recovery and partial recovery were summed, the recovery rate was 36%. The recovery rate of each group was as follows: 3 (75%) of 4 patients in the C–type group, 3 (38%) of 8 patients in the C + O–type group, and 11 (11%) of 9 patients in the C + O + S–type group. There was significant correlation between the recovery rate and the type ranking (Spearman rank correlation test, r = -0.49; P = .03).

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Side</th>
<th>Initial PTA</th>
<th>Type of Audiogram</th>
<th>Canal Paresis</th>
<th>Click-VEMP</th>
<th>Galvanic-VEMP*</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/30</td>
<td>R</td>
<td>66.7</td>
<td>High-tone HL</td>
<td>–</td>
<td>Normal</td>
<td>Normal</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>2/M/60</td>
<td>L</td>
<td>43.3</td>
<td>High-tone HL</td>
<td>–</td>
<td>Normal</td>
<td>Normal</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>3/F/47</td>
<td>L</td>
<td>43.3</td>
<td>High-tone HL</td>
<td>–</td>
<td>Normal</td>
<td>Partial recovery</td>
<td></td>
</tr>
<tr>
<td>4/M/30</td>
<td>R</td>
<td>43.3</td>
<td>High-tone HL</td>
<td>–</td>
<td>Normal</td>
<td>No change</td>
<td></td>
</tr>
<tr>
<td>5/F/59</td>
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<td>+</td>
<td>Normal</td>
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<td></td>
</tr>
<tr>
<td>6/M/61</td>
<td>L</td>
<td>81.7</td>
<td>High-tone HL</td>
<td>–</td>
<td>Absent</td>
<td>Normal</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>7/M/37</td>
<td>L</td>
<td>51.7</td>
<td>Low-tone HL</td>
<td>–</td>
<td>Absent</td>
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<td></td>
</tr>
<tr>
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<td>L</td>
<td>65.0</td>
<td>High-tone HL</td>
<td>–</td>
<td>Absent</td>
<td>Complete recovery</td>
<td></td>
</tr>
<tr>
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<td>103.3</td>
<td>Flat-type HL</td>
<td>–</td>
<td>Absent</td>
<td>No change</td>
<td></td>
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<tr>
<td>10/M/22</td>
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<td>High-tone HL</td>
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<td>Absent</td>
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<tr>
<td>11/F/29</td>
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<td>Partial recovery</td>
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<tr>
<td>12/F/58</td>
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<td>75.0</td>
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<td>–</td>
<td>Absent</td>
<td>No change</td>
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<tr>
<td>13/F/63</td>
<td>R</td>
<td>111.7</td>
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<tr>
<td>14/M/67</td>
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<tr>
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<tr>
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<td>High-tone HL</td>
<td>+</td>
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<td>No change</td>
<td></td>
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<tr>
<td>19/M/29</td>
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<td>31.7</td>
<td>High-tone HL</td>
<td>+</td>
<td>Absent</td>
<td>Normal</td>
<td>No change</td>
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<tr>
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<td>103.3</td>
<td>Profound HL</td>
<td>+</td>
<td>Absent</td>
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<td></td>
</tr>
<tr>
<td>21/M/63</td>
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<td>Profound HL</td>
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<td>Absent</td>
<td>Normal</td>
<td>No change</td>
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<td>65.0</td>
<td>High-tone HL</td>
<td>+</td>
<td>Absent</td>
<td>Partial recovery</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: Click-VEMP, vestibular evoked myogenic potentials (VEMPs) evoked by clicks; galvanic-VEMP, VEMPs evoked by galvanic stimulation; HL, hearing loss; L, left; PTA, pure-tone average; R, right; +, present; –, absent.

*Galvanic-VEMP testing was performed in 8 patients.

KLoric responses (canal paresis, >20%) on the affected side. Four patients (18%) had normal caloric responses and normal VEMPs on the affected side. Eight of the 17 patients who showed an absence of click-VEMPs on the affected side underwent galvanic-VEMP testing. All 8 patients showed normal galvanic-VEMPs on the affected side. Most patients with decreased caloric responses (9 [90%] of 10 patients) did not show click-VEMPs on the affected side. However, 9 (53%) of the 17 patients who showed abnormal click-VEMPs showed decreased caloric responses on the affected side.

We classified patients with normal VEMPs and normal caloric responses as the cochlea (C) type; those with abnormal VEMPs but normal caloric responses as the cochlea and otolith (saccule) (C + O) type; those with normal VEMPs but abnormal caloric responses as the cochlea and (lateral) semicircular canal (C + S) type; and those with abnormal VEMPs and caloric responses as the cochlea, otolith (saccule), and (lateral) semicircular canal (C + O + S) type. Representative cases of the C + O and C + O + S types are shown in Figure 1 and Figure 2, respectively. Of the 22 patients, 4 (18%) were classified as the C type; 8 (36%), as the C + O type; and 9 (41%), as the C + O + S type. Only 1 patient (5%) was classified as the C + S type (Figure 3). The median age of the C–type group was 38.5 years; the C + O–type group, 49.5 years; the C + S–type group, 39 years; and the C + O + S–type group, 60 years. There was no significant difference in age among these groups (Kruskal-Wallis analysis of variance on ranks, P = .54). In the present study, the C + S type was not included in the statistical analysis because of the small number of this group (n = 1).
In the present study, we have examined vestibular functions of ISHL patients with vertigo using click-VEMP, galvanic-VEMP, and caloric testing. Click-VEMPs have been used as a clinical test of the vestibular system, especially of the saccule and inferior vestibular nerve region.9-11 Clinical studies have suggested that this response is of vestibular origin, especially of the saccule and inferior vestibular nerve region.9,11 Neurophysiological experiments in guinea pigs and cats have shown that primary vestibular afferents, especially saccular afferents, respond to relatively loud clicks.12,18 Combined use of VEMP and caloric testing, a clinical test of the lateral semicircular canal and superior vestibular nerve region, has facilitated more precise examination of the vestibular system. Abnormal VEMPs have been reported in various diseases such as vestibular neuritis,9 Ménière’s disease,19 and acoustic neuromas.10

Figure 1. A representative case of the cochlea and otolith (saccule) type of vestibular dysfunction (patient 6). A, Pure-tone audiogram showing high-tone hearing loss in the left ear (L). R indicates right ear. B, Vestibular evoked myogenic potentials evoked by clicks (click-VEMP) showing an absence of response on the left side. C, Vestibular evoked myogenic potentials evoked by galvanic stimulation (galvanic-VEMP) showing normal responses on both sides. Results of caloric tests demonstrated normal responses in both ears (canal paresis, 8.3% on the left side). The first positive-negative peaks (p13-n23 for click-VEMP and p13g-n23g for galvanic-VEMP) are shown. Downward arrow indicates off the scale.

Figure 2. A representative case of the cochlea, otolith (saccule), and (lateral) semicircular canal type of vestibular dysfunction (patient 14). A, Pure-tone audiogram showing profound hearing loss in the right ear (R). L indicates left ear. B, Vestibular evoked myogenic potentials evoked by clicks (click-VEMP) showing an absence of response on the right side. C, Vestibular evoked myogenic potentials evoked by galvanic stimulation (galvanic-VEMP) showing normal responses on both sides. Results of caloric tests showed no responses in the right ear (canal paresis, 100%). The first positive-negative peaks (p13-n23 for click-VEMP and p13g-n23g for galvanic-VEMP) are shown. Downward arrow indicates off the scale.
However, there have been no reports on click-VEMPs in ISHL with vertigo. In this study, approximately three fourths (77%) of the patients showed an absence of click-VEMPs on the affected side, whereas fewer than half (45%) of the patients showed decreased caloric responses, suggesting that the saccule and/or its afferents are involved in ISHL with vertigo more frequently than the lateral semicircular canal and its afferents.

It has recently been shown that galvanic stimulation also evokes myogenic responses on the sternocleidomastoid muscle. Watson and Colebatch found that these myogenic potentials disappeared after selective vestibular nerve section and suggested that galvanic stimulation would stimulate the most distal portion of the vestibular nerve, whereas clicks would act at the receptor level. Murofushi et al. have reported that combined use of click- and galvanic-VEMPs is useful in the differential diagnosis of labryinthine lesions from retrolabyrinthine lesions in patients with vestibular deficits. In the present study, all 8 patients who underwent galvanic-VEMP testing showed normal responses on the affected side, suggesting that the vestibular lesion of ISHL with vertigo lies within the labyrinth. This result is consistent with the fact that the lesion causing hearing loss is in the cochlea. Furthermore, the result of the present study is in contrast to that of vestibular neuritis, which is also considered to be caused by viral infection. Murofushi et al. examined 11 patients with vestibular neuritis using click-VEMP and galvanic-VEMP testing. In their study, 8 of the 11 patients showed absence of click-VEMPs and galvanic-VEMPs on the affected side, suggesting that the site of the lesion in vestibular neuritis was primarily within the vestibular nerve.

According to the results of the click-VEMP and caloric tests, we have divided the lesion sites in ISHL patients with vertigo into C, C + O, C + S, and C + O + S types. We referred to the results of VEMP and caloric testing as indicating the otolith and semicircular canal, respectively, although the functions of the utricle and the anterior and posterior semicircular canals remain unknown. Those patients who were classified into the C type might have dysfunction of the utricle and/or the anterior and posterior semicircular canals, although it is possible that the function of the saccule and/or lateral semicircular canal might have recovered before vestibular testing. In this study, most patients were classified into the C, C + O, or C + O + S types, whereas patients showing the C + S type were exceptional. This result could be explained by an anatomic distribution of damage at the cochlea extending to the semicircular canals through the otolith organs. Khetarpal investigated the histopathologic characteristics of the temporal bone in sudden deafness with and without vertigo and found no direct relationship between the presence of vertigo and damage to the vestibular apparatus. He hypothesized that vertigo in ISHL was caused by transmission of biochemical changes in inner ear fluid from the cochlea to the vestibular apparatus. Nakashima and Yanagita reported the highest incidence of vertigo in patients with high-frequency hearing loss (43%) and explained this finding by an anatomic factor, i.e., the cochlear basal turn is more proximal to the vestibular apparatus than the upper turn. High incidence of a high-frequency hearing loss was also observed in our study.

Viral infection has long been considered one of the most probable causes of ISHL. Viruses such as the herpes simplex virus, varicella-zoster virus, mumps, cytomegalovirus, rubella, and the enteroviruses have been suggested to play a causative role in the pathogenesis of ISHL. Microscopic studies of the temporal bones most commonly showed atrophy of the organ of Corti, tectorial membranes, and stria vascularis, which was consistent with viral infection. Although the role of a viral infection in the induction of ISHL is poorly understood, mechanisms involving biochemical alteration of the endolymph or the intravascular coagulopathy that affects the hair cell but allows reversible hearing loss have been suggested. It has been reported that hearing recovery in sudden deafness is worse in patients with vertigo than in those without vertigo. In our study, 36% of the patients with sudden deafness with vertigo showed hearing recovery. This recovery rate is compatible with those reported in...
other studies. In the present study, the hearing recovery rate was on the order of C type, followed by C + O type, and then C + O + S type, suggesting that hearing recovery worsened as more parts of the vestibular apparatus became involved. This result might be caused by differences in initial hearing levels because there was an inverse relationship between the initial pure-tone average and the extent of the vestibular lesion. It has been reported that hearing recovery was worse in patients with profound hearing loss in several studies, whereas Nakashima and Yanagita showed that hearing recovery was worse in patients with vertigo, even when the initial hearing loss was the same.

In conclusion, the lesion site of the vestibular disorders in ISHL with vertigo is within the labyrinth in terms of galvanic-VEMPs. Results of click-VEMP and caloric testing suggested that the saccule is involved more frequently than the semicircular canals. Combined use of click-VEMP and caloric testing is useful for evaluating vestibular functions in ISHL with vertigo because types of vestibular abnormalities correlated well with hearing outcome.

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