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ORIGINAL RESEARCH

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Differences in vestibulo-ocular reflexes between vestibular neuritis and labyrinthitis

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Abstract

Objective: To expand our understanding of the pathophysiological mechanisms underlying vestibular neuritis and labyrinthitis by identifying any difference in the vestibulo-ocular reflex for each semicircular canal.

Study Design: Retrospective analysis.

Setting: The Department of Otorhinolaryngology – Head and Neck Surgery, Chosun University Hospital, from January 2015 to December 2021.

Methods: We included 23 vestibular neuritis and 27 labyrinthitis patients who had been hospitalized. Pure-tone audiometry, a bithermal caloric test, and a video head-impulse test were performed within 5 days of symptom onset.

Results: In the vestibular neuritis group, mean vestibulo-ocular reflex gains were decreased to 0.51 in the ipsilesional horizontal canal and 0.55 in anterior canal, leading to marked asymmetry, whereas the gain of the ipsilesional posterior canal was relatively preserved at 0.85. In the labyrinthitis group, the mean vestibulo-ocular reflex gain was 0.72 in the ipsilesional horizontal canal, 0.73 in the ipsilesional anterior canal, and 0.55 in the ipsilesional posterior canal. We observed statistical differences in the vestibulo-ocular reflex gain and incidence of corrective saccades on the ipsilesional side in three semicircular canals between the groups ($p = .002$ for horizontal canal, $p = .003$ for anterior canal, and $p < .001$ for posterior canal). The receiver operating characteristic curve showed that pure-tone audiometry, ipsilesional posterior canal gain, and gain asymmetry of posterior canal were excellent parameters for distinguishing labyrinthitis from vestibular neuritis.

Conclusion: Vestibular neuritis and labyrinthitis patients have different degrees and patterns of video head-impulse test involvement in the three semicircular canals, suggesting that the two distinct disorders may have different etiologies.

KEYWORDS
head-impulse test, labyrinthitis, sudden hearing loss, vertigo, vestibular neuritis, vestibulo-ocular reflex

1 | INTRODUCTION

The spectrum of sudden loss of inner-ear function can be observed in clinical practice, with vestibular neuritis (VN) and sudden sensorineural hearing loss (SSNHL) serving as representative examples.[1] A state known as labyrinthitis may also be observed, which simultaneously exhibits features of both VN and SSNHL.[2] Acute vestibular syndrome (AVS) is characterized by the sudden onset of acute continuous vertigo, motion intolerance, gait instability, and spontaneous nystagmus lasting longer than 24 h.[3] Although VN is the most common cause of peripheral AVS, labyrinthitis—which shares the characteristics of VN—can be another possible disorder of AVS.[4,5] VN and labyrinthitis as a subtype of AVS are considered two distant disorders with different underlying pathophysiologic features because they can be easily differentiated clinically depending on the presence of sudden hearing deterioration.[2,5] In fact, labyrinthitis has been reported as SSNHL with vertigo in several studies, and its prognosis and audio-vestibular properties have been actively reported.[1,4–8] However, the subjective nature of vertigo and the heterogeneity of study populations causes variability in the reported results.[6–9] Moreover, few studies have explored the differences in vestibular function between SSNHL and vertigo, which can be considered labyrinthitis and VN,[5,10] and little is known about the differences and characteristics of the vestibulo-ocular reflex (VOR) between these two diseases.

The video head impulse test (vHIT) is a valuable tool for evaluating the VOR during relatively rapid head rotation and for quantitatively analyzing the function of each semicircular canal in the clinical setting.[11,12] For VN and labyrinthitis patients, a thorough functional assessment of labyrinthine activity is currently available using the vHIT, which can help shed more light on the pathophysiologic processes underlying these two disorders. Thus, this study primarily aimed to determine whether there was a difference in VOR gain and pattern involvement for each semicircular canal and to expand our understanding of the pathophysiological mechanism of these two disorders with strict diagnostic criteria. We also aimed to ascertain whether patterns of vHIT anomalies could be distinguished between labyrinthitis and VN, along with hearing loss.

2 | MATERIALS AND METHODS

2.1 | Participants

The data of 23 VN and 27 labyrinthitis patients were retrospectively reviewed at the Department of Otorhinolaryngology-Head and Neck Surgery, Chosun University Hospital, from January 2015 to December 2021. VN patients were included based on the presence of AVS symptoms with acute continuous vertigo, spontaneous horizontal-torsional nystagmus beating toward the healthy side, gait disturbance, nausea, or vomiting for <3 days after symptom onset. Labyrinthitis was diagnosed when the criteria for both SSNHL (presence of sudden sensorineural hearing loss ≥30 dB in hearing threshold for at least three consecutive frequencies)[13] and VN[2] were met concomitantly. Patients without spontaneous nystagmus or with suspected positional vertigo were excluded from the study. Patients with a history of audio-vestibular disorders, such as VN, SSNHL, Meniere's disease, or vestibular migraine, were excluded from further analysis. All enrolled patients were hospitalized, and pure-tone audiometry and vestibular function tests, including the bithermal caloric test and vHIT, were completed within 5 days of symptom onset. All enrolled patients were confirmed to have no stroke, hemorrhage, or cerebellopontine angle tumor via brain MRI. This study was approved by the Institutional Review Board of the Chosun University Hospital in Gwangju, Korea (IRB number CHOSUN 2022-08-011). Because this was a retrospective study, the requirement for informed consent was waived.

2.2 | Pure-tone and speech audiometry

Pure-tone audiometry (PTA) and speech discrimination were performed by an experienced audiologist. Average pure-tone thresholds of 500, 1000, 2000, and 4000 Hz were defined as PTA_4. The speech discrimination score was defined as the percentage of correctly repeated 50 monosyllabic Korean words at the sound intensity of the most comfortable hearing level. For both tests, the audiologist used noise masking in the unaffected ear, as needed. All tests were conducted using a Madsen Orbiter 922 audiometer® (Otometrics) in a soundproof room with double soundproof walls that satisfied the noise tolerance level (ISO 8253-1:2010) of headphones.

2.3 | Bithermal caloric test

Bithermal caloric test (SLVNG, SLMED, Seoul, Korea) was performed with the patient positioned in a chair reclined 30° to vertically orient the semicircular canals. Each external auditory canal was irrigated alternately with a constant flow of water at temperatures of 30°C and 44°C for a constant period (30 s). Induced nystagmus was recorded using electronystagmography in a dark room with the patient's eyes open. The maximum slow-phase eye velocity of nystagmus evoked by each ear was analyzed for unilateral canal weakness (CW) and directional preponderance (DP), as determined by Jongkees' formula after the degree of spontaneous nystagmus was automatically corrected. Abnormal CW was defined as >25% asymmetry based on the Jongkees formula.[14]

2.4 | Video head impulse test

The function of the three paired semicircular canals was evaluated using a three-dimensional vHIT (ICS Impulse, Otometrics, Taastrup, Denmark). Patients were positioned at a distance of 1 m from a target located at eye level. To ensure the reliability of the examination process, the goggles were fastened to the patient's head using an elastic band to minimize slippage. The technician manually performed >20 rotations (head rotation, 15°–20°; duration, 150–200 ms; peak

TABLE 1 Demographic and clinical data of the study population.

	VN ($n = 23$)	Labyrinthitis ($n = 27$)	p-value
Age	53.39 ± 14.74	57.56 ± 19.00	.397[a]
M:F	12:11	11:16	.419[b]
R:L	7:16	15:12	.075[b]
HTN	8 (34.8%)	12 (44.4%)	.487[b]
DM	2 (8.7%)	9 (33.3%)	.036[b]
CVD	2 (8.7%)	4 (14.8%)	.674[c]
Time to test	2.84 ± 2.32	3.61 ± 2.74	.263[d]

Abbreviations: CVD, cardiovascular disease; DM, diabetes mellitus; F, female; HTN, hypertension; M, male; VN, vestibular neuritis.
[a]Independent *t*-test.
[b]Chi-square test.
[c]Fisher's exact test.
[d]Mann-Whitney *U* test.

velocity, >150°) on both sides of each plane. Because accurate and consistent angles for vertical semicircular canal head rotations are difficult to obtain in the vHIT, an experienced, well-trained technician performed all maneuvers. The vHIT parameters used in the analysis were VOR gain, VOR gain asymmetry, and incidence of corrective saccades. The VOR gain in the three semicircular canals (anterior canal [AC]; horizontal canal [HC]; posterior canal [PC]; ipsilesional [i]; contralesional [c]) was calculated using the ratio of the area under the curve (AUC) for the eye velocity area to the head velocity area, which was automatically determined by the device. Abnormal VOR gain was defined as an HC <0.8, and AC and PC of 0.7[11] and/or the presence of covert and/or overt saccades. The gain asymmetry was calculated as the gain of each functionally paired semicircular canal (e.g., ipsilesional horizontal canal [iHC] and contralesional horizontal canal [cHC], ipsilesional anterior canal [iAC] and contralesional posterior canal [cPC], and ipsilesional posterior canal [iPC] and contralesional anterior canal [cAC]) according to the following equation: $Gs = [(Gc - Gi)/(Gc + Gi)] \times 100\%$, where Gc is the gain of the contralesional semicircular canal, and Gi is the gain of the ipsilesional semicircular canal. The overt (if the onset was after the end of the head rotation) or covert (if the onset was before the end of the head rotation) saccades used in the analysis were defined as more than 20% of trials exhibiting similar amplitude and latency during all vHIT trials, thus differentiating actual saccades from artifacts.

2.5 | Statistical analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences for Windows version 27.0; IBM Corp., Armonk, NY. Statistical significance was set at $p < .05$. Quantitative parameters are presented as mean ± standard deviation (SD). Categorical data were reported as numbers (percentages). The independent *t*-test or Mann–Whitney *U* test and chi-square or Fisher's exact test were used to compare the parameters in this study. Each evaluation method was selected based on the normality of the sample after the Shapiro–Wilk test. Receiver operating characteristic (ROC) curve analysis was performed to identify the parameters that were useful in distinguishing the VN and labyrinthitis groups.

TABLE 2 Pure-tone thresholds and speech discrimination scores in patients with VN and labyrinthitis.

	VN ($n = 23$)	Labyrinthitis ($n = 27$)	p-value
$iPTA_4$ (dB)	24.92 ± 19.14	89.21 ± 16.02	<.001[a]
iSD (%)	92.00 ± 8.82	6.48 ± 19.31	<.001[a]
$cPTA_4$ (dB)	25.33 ± 19.15	20.93 ± 14.12	.53[a]
cSD (%)	93.07 ± 8.61	93.93 ± 4.62	.65[a]

Note: Average pure-tone thresholds of 500, 1000, 2000, and 4000 Hz in the affected ear were defined as PTA_4.
Abbreviations: c, contralesional side; dB, decibel; i, ipsilesional side; PTA_4, pure-tone audiometry; SD, standard deviation (%); VN, vestibular neuritis.
[a]Mann-Whitney *U* test.

3 | RESULTS

3.1 | Clinical manifestation

Table 1 summarizes the demographics of the enrolled VN or labyrinthitis patients. No significant differences were observed in age, sex ratio, laterality of the lesion side, hypertension, cardiovascular disease, or time from symptom onset to test. The prevalence of diabetes was significantly higher in labyrinthitis patients ($p = .036$, chi-square test, Table 1). All enrolled patients showed spontaneous horizontal-torsional nystagmus beating toward the contralesional side. Twenty-two of 25 (88%) VN and 15 of 28 (54%) labyrinthitis patients showed definite corrective saccades in the bedside head-impulse test.

3.2 | Pure-tone audiometry

The average $iPTA_4$ was 24.92 dB HL, with 92% speech discrimination in the VN group and 89.21 dB HL with 6% speech discrimination in the labyrinthitis group ($p < .001$, Mann–Whitney *U* test, Table 2). Of the 25 VN patients, two showed mild bilateral hearing loss and three showed mild-to-moderate bilateral hearing loss, which was considered part of the aging process, with no clear audio-vestibular etiologies. Of the 28 labyrinthitis patients, 24 (85.7%) showed severe-to-profound hearing loss at the initial visit.

3.3 | Bithermal caloric test

Abnormal CW was observed in 19 patients (85.0%) in the VN group and 18 (67.0%) in the labyrinthitis group. The mean caloric weakness in the VN and labyrinthitis groups were 51.45% and 43.05%, respectively ($p = .647$, Mann–Whitney *U* test). DP was significantly increased in the VN group ($p = .04$, independent *t*-test, Table 3).

TABLE 3 Canal weakness (CW) in patients with VN and labyrinthitis.

	VN ($n = 23$)	Labyrinthitis ($n = 27$)	p-value
CW (%)	51.45 ± 27.40	43.05 ± 33.59	.647[a]
Abnormal CP (n, %)	19 (85.0%)	18 (67.0%)	.200[b]
DP	62.05 ± 51.02	20.70 ± 32.44	.004[c]

Abbreviations: CP, canal paresis; DP, directional preponderance; VN, vestibular neuritis.
[a]Mann-Whitney U test.
[b]Chi-square test.
[c]Independent t-test.

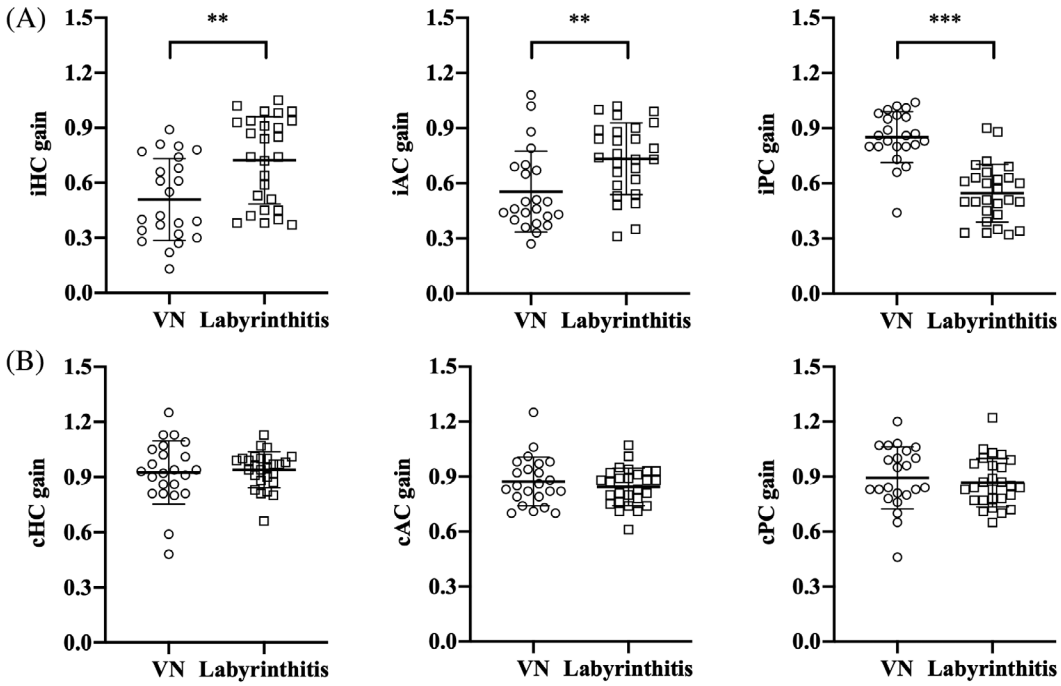

FIGURE 1 VOR gain of the (A) ipsilesional (i) and (B) contralesional (c) sides of three semicircular canals in patients with VN and labyrinthitis. Compared with the VN group, the VOR gain for labyrinthitis patients was reduced in the iPC, but relatively preserved in the iHC and iAC. The VOR gain of the contralateral sides showed no statistically significant differences. ***p-value <.001, **p-value <.01, *p-value <.05. AC, anterior semicircular canal; HC, horizontal semicircular canal; PC, posterior semicircular canal; VN, vestibular neuritis; VOR, vestibulo-ocular reflex.

3.4 | VOR gains and properties in VN and labyrinthitis

The quantitative differences in VOR gain between the groups are detailed in Figure 1 and Table 4. In the VN group, VOR gains were smaller on the iHC (0.51 ± 0.22) and iAC (0.55 ± 0.22), leading to marked asymmetry (Figure 2). However, the iPC VOR gain (0.85 ± 0.16) was within the pre-defined values of normality compared with that of HC and AC. In the labyrinthitis group, the VOR gain was 0.72 in the iHC, 0.73 in the iAC, and 0.55 in the iPC (Table 4 and Figure 1). Compared to that in VN patients, labyrinthitis patients showed relatively preserved gain in the iHC ($p = .002$, Mann–Whitney U test) and iAC ($p = .003$, Mann–Whitney U test). However, the iPC gain was dramatically decreased (0.85 ± 0.16 in VN and 0.55 ± 0.16 in labyrinthitis, $p < .001$, independent t-test) (Figure 1 and Table 4). The incidence of corrective saccades (covert or overt) is summarized in Table 4. VN patients showed frequent corrective saccades during the iHC and iAC examinations. Compared to those in VN patients, labyrinthitis patients showed more frequent corrective saccades during PC examination, which is similar to the VOR analysis results. The gain asymmetry (Gs) provides a more convincing validation of this variation in VOR properties (Figure 2). The Gs in VN was significantly increased in the HC (mean Gs of HC, 31.89 in VN vs. 15.19 in labyrinthitis, $p = .001$, Mann–Whitney U test) and AC (mean Gs of AC, 25.24 in VN vs. 9.28 in labyrinthitis, $p < .001$, Mann–Whitney U test); however, the robust Gs was observed in PC (mean Gs of PC, −0.66 in VN vs. 22.61 in labyrinthitis, $p < .001$, independent t-test, Figure 2). Consequently, we observed statistically significant differences in the VOR gain and incidence of corrective saccades on the ipsilesional side in all three semicircular canals between the groups.

The pattern of semicircular involvement (abnormal gain and/or presence of covert and/or overt saccades) in the enrolled patients is

TABLE 4 Vestibulo-ocular reflexes on video head-impulse test in patients with VN and labyrinthitis.

	VN ($n = 23$)	Labyrinthitis ($n = 27$)	p-value
iHC	0.51 ± 0.22	0.72 ± 0.24	.002[a]
Covert saccades	9 (39.1%)	7 (25.9%)	.318[b]
Overt saccades	22 (95.7%)	19 (70.4%)	.028[c]
Any saccades	22 (95.7%)	20 (74.1%)	.055[c]
iAC	0.55 ± 0.22	0.73 ± 0.20	.003[a]
Covert saccades	2 (8.7%)	2 (7.4%)	1.000[c]
Overt saccades	13 (56.5%)	6 (22.2%)	.013[b]
Any saccades	14 (60.9%)	6 (22.2%)	.005[b]
iPC	0.85 ± 0.16	0.55 ± 0.16	<.001[d]
Covert saccades	2 (8.7%)	4 (14.8%)	.674[c]
Overt saccades	7 (30.4%)	20 (74.1%)	.002[b]
Any saccades	9 (39.1%)	20 (74.1%)	.013[b]
cHC	0.93 ± 0.17	0.94 ± 0.10	.712[d]
Covert saccades	0	1 (3.7%)	1.000[c]
Overt saccades	7 (30.4%)	7 (25.9%)	.723[b]
Any saccades	7 (30.4%)	7 (25.9%)	.723[b]
cAC	0.87 ± 0.13	0.84 ± 0.10	.401[d]
Covert saccades	2 (8.7%)	0	.207[c]
Overt saccades	1 (4.3%)	0	.460[c]
Any saccades	3 (13.0%)	0	.09[c]
cPC	0.89 ± 0.17	0.87 ± 0.13	.543[d]
Covert saccades	1 (4.3%)	0	.460[c]
Overt saccades	3 (13.0%)	7 (25.9%)	.308[c]
Any saccades	4 (17.4%)	7 (25.9%)	.468[b]

Abbreviations: AC, anterior canal; c, contralesional side; HC, horizontal canal; i, ipsilesional side; PC, posterior canal; VN, vestibular neuritis.
[a]Mann-Whitney *U* test.
[b]Chi-square test.
[c]Fisher's exact test.
[d]Independent *t*-test.

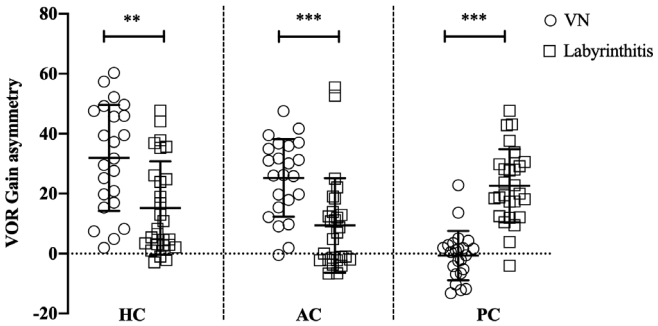

FIGURE 2 The asymmetry of VOR gain (Gs) in patients with VN and labyrinthitis. The Gs in VN was significantly increased in the HC (mean Gs of HC, 31.89% in VN vs. 15.19% in labyrinthitis, $p = .001$, Mann–Whitney *U* test) and AC (mean GS of AC, 25.24% in VN vs. 9.38% in labyrinthitis, $p < .001$, Mann–Whitney *U*-test). However, The Gs in PC was significantly decreased (mean GS of PC, 0.66% in VN vs. 22.61% in labyrinthitis, $p < .001$, independent *t*-test) compared with labyrinthitis patients.

summarized in Figure 3. In the VN group, 10 (43.5%) patients showed a pattern of HC and AC involvement, and nine (39.1%) showed semicircular canal involvement. However, in labyrinthitis, the pattern of semicircular canal involvement (40.7%) or isolated PC involvement (29.6%) was predominant (Figure 3).

3.5 | Cutoff value of parameters for distinguishing labyrinthitis from VN

ROC analysis was performed to determine which parameters were valuable for discriminating labyrinthitis from VN, including PTA_4, ipsilesional VOR gain, and Gs (Table 5). iPTA4 (AUC = 0.985), iPC gain (AUC = 0.928), and Gs in PC (AUC = 0.942) were robust examples. The optimal cutoff value of 54.38 dB for $iPTA_4$ provided the best sensitivity (93%) and specificity (94%). Labyrinthitis could be diagnosed with a sensitivity of 87% and specificity of 89% when the iPC gain

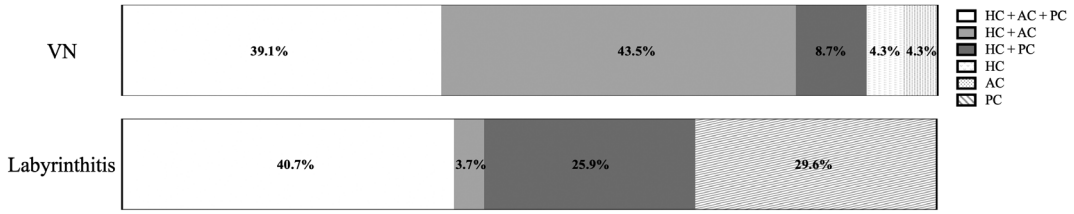

FIGURE 3 The pattern of semicircular involvement (abnormal gain and/or presence of covert and/or overt saccades) in enrolled patients. AC, anterior semicircular canal; HC, horizontal semicircular canal; PC, posterior semicircular canal; VN, vestibular neuritis.

TABLE 5 The ROC analysis for the PTA, VOR gain, and Gs to predict differentiation of labyrinthitis from VN.

							Asymptotic 95% Confidence Interval	
Variable	Area	Cutoff value	Sensitivity	Specificity	Std. error[a]	Asymptotic Sig[b]	Lower bound	Upper bound
$iPTA_4$	0.985	54.38 dB	93%	93%	0.014	0.000	0.957	1.000
iHC	0.758	0.62	63%	65%	0.067	0.002	0.626	0.889
iAC	0.745	0.67	67%	70%	0.075	0.003	0.598	0.891
iPC	0.928	0.71	87%	89%	0.039	0.000	0.850	1.000
Gs in HC (%)	0.775	22.37	65%	67%	0.065	0.001	0.646	0.903
Gs in AC (%)	0.820	16.62	74%	78%	0.062	0.000	0.698	0.941
Gs in PC (%)	0.942	9.94	89%	91%	0.034	0.000	0.876	1.000

Abbreviations: AC, anterior canal; dB, decibel; Gs, gain asymmetry; HC, horizontal canal; PC, posterior canal; PTA, pure-tone audiometry; ROC, receiver operating characteristics; VN, vestibular neuritis; VOR, vestibulo-ocular reflex.
[a]Under the nonparametric assumption.
[b]Null hypothesis: true area = 0.5.

was 0.71, and with a sensitivity of 89% and specificity of 91% when the Gs in PC was above 9.94% (Table 5).

4 | DISCUSSION

VN and labyrinthitis are two distinct auditory and/or vestibular disorders clinically distinguished by the presence or absence of SSNHL.[2] The presence of sudden, abrupt hearing loss, which may be the only clinical symptom to suggest labyrinthitis, clinically distinguishes labyrinthitis from VN. In the present study, the VOR properties of all semicircular canals were quantitatively investigated using vHIT. An attempt was also made to assess the properties of VOR parameters that could help differentiate labyrinthitis from VN. The following distinctive vHIT properties of labyrinthitis compared with VN were observed: (1) relatively preserved VOR gain in the ipsilesional HC and AC, (2) robust decrease in iPC gain, and (3) by employing the Gs in PC and iPC gain as well as PTA_4, labyrinthitis can be differentiated from VN. The overall VOR pattern in individuals with VN could be identified by a pronounced asymmetry caused by a robust gain reduction in iHC and iAC, while iPC gain was comparatively preserved.

Although the etiology of these two disorders is assumed to be distinct,[5] they may theoretically share some pathophysiology, including viral infection and vascular ischemia, which might be the common causes of both.[7,8] The most widely acknowledged cause of VN is viral infection,[15] although inner-ear vascular ischemia may provide a more convincing explanation for the clinical properties of labyrinthitis.[4,5,9]

Considering the distribution of the blood supply in the inner ear, ischemia of the internal auditory artery (IAA), common cochlear artery (CCA), or posterior vestibular artery (PVA) leads to cochlear and/or vestibular symptoms.[4,5] The IAA, which provides blood to the inner-ear organs, normally arises from the anterior inferior cerebellar artery.[16] The IAA splits into the CCA and anterior vestibular artery (AVA) inside the internal auditory canal. The main cochlear artery and vestibulocochlear artery, in turn, give rise to the PVA and cochlear ramus from the CCA. The AVA supplies blood to the utricle, ampullae of the AC and HC, and the superior saccule, whereas the PVA supplies blood to most of the saccule, ampulla of the PC, and part of the ampulla of HC and AC. Consequently, both vestibular and auditory symptoms are caused by ischemia of the IAA or CCA. IAA ischemia results in simultaneous hearing loss and semicircular canal dysfunction, whereas PVA ischemia results in localized PC dysfunction along with hearing loss. In the present study, 40.7% of labyrinthitis patients exhibited all canal involvement patterns, 29.6% isolated PC patterns, and 25.9% HC and PC involvement patterns (Figure 3). Additionally, the inner-ear vascular anatomy may properly account for the PC gain reduction and asymmetry in labyrinthitis patients, which was validated in the current study (Figures 1 and 2). This vascular etiology is further supported by the high incidence of DM in individuals with labyrinthitis (Table 1). Contrary to labyrinthitis, all canal or isolated involvement patterns seen in VN patients without hearing loss cannot be fully explained by inner-ear ischemia. The ischemia of the AVA or neural lesion of superior vestibular nerve can partially explain the HC and AC involvement pattern in the 10 VN patients (43.5%) in our study, but

other patterns, especially nine canal involvement patterns (39.1%), are difficult to describe, except for viral origin (Figure 3).[15] The present study also showed that increased Gs in PC and iPC gain reduction, measured as an excellent parameter that could distinguish labyrinthitis from VN, provided comparable sensitivity and specificity to the degree of hearing loss (Table 5). These results imply that PC supplied by PVA is more vulnerable to ischemia, suggesting that VN and labyrinthitis may not necessarily share the same pathophysiological processes.

Although several studies have investigated vestibular function in SSNHL with vertigo, which can be considered labyrinthitis, to our knowledge, our study is the first to compare VN and labyrinthitis as clear diagnostic criteria. The diagnosis of labyrinthitis was made only when there was a clear spontaneous nystagmus, not simply a vertiginous symptom accompanied by SSNHL. Similar to our study, Pogson et al. reported the audio-vestibular profiles of 27 patients, including 23 with labyrinthitis/labyrinthine infarctions who presented with acute vertigo with SSNHL. They demonstrated iPC gain reduction, which was explained by ischemia of the CCA or vestibulo-cochlear branches of the labyrinthine artery.[4] In a study of 30 SSNHL patients with vertigo and 22 VN patients, Yao et al. compared vHIT results and showed an iPC gain reduction in SSNHL with vertigo.[5] Unlike in our study, they did not demonstrate a difference in iPC gain between the groups. These differences were due to the possibility that the VN group in this study mostly comprised patients with superior or total type of involvement, and the isolated inferior type of VN was not properly included. The likely reason that our patient population did not include isolated inferior VN is that VN preferentially affects the superior or total vestibular nerve and rarely involves the inferior division only. The diagnosis of VN would have been missed in such patients because they would have exhibited mild dizziness, little spontaneous nystagmus, or normal caloric responses. Therefore, a patient with isolated PC gain reduction in AVS may not meet the clinical criteria for VN and should be investigated for other etiologies, such as central vestibular lesions or labyrinthitis.[17,18]

This study had several limitations. The patients were assessed retrospectively, which could have resulted in selection and information biases. The type of hearing loss, severity of vertigo, and other concomitant symptoms were not examined despite the possibility that they may have affected the findings. Finally, we did not evaluate the function of the utricle and saccule using ocular or cervical vestibular evoked myogenic potentials; therefore, the involvement of vestibular end organs was not fully explained.

5 | CONCLUSION

VN and labyrinthitis patients exhibit different degrees and patterns of vHIT involvement in all three semicircular canals, suggesting that the two disorders have distinct etiologies. Additional research is required to confirm these findings and to identify various etiologies.

AUTHOR CONTRIBUTIONS

Conceptualization: Gi-Sung Nam and Sung Il Cho. Data curation: Wonyong Baek and Min Seok Kim. Formal analysis: Min Seok Kim and Wonyong Baek. Funding acquisition: Gi-Sung Nam. Investigation: Gi-Sung Nam and Min Seok Kim. Methodology: Gi-Sung Nam. Supervision: Sung Il Cho. Validation: Gi-Sung Nam. Visualization: Gi-Sung Nam and MSK. Writing – original draft: Gi-Sung Nam. Writing – review and editing: Sung Il Cho.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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