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Semicircular canal dysfunction in profound sudden sensorineural hearing loss and its prognostic value: implications for the proper indication of hyperbaric oxygen therapy

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Vestibular involvement in sudden sensorineural hearing loss (SSNHL) is regarded as an indicator of poor prognosis. This study aimed to investigate the prognostic significance of semicircular canal dysfunction in profound SSNHL and evaluate the effectiveness of hyperbaric oxygen therapy (HBOT) accordingly. From October 1, 2020 to August 15, 2025, patients diagnosed with profound SSNHL (≥ 80 dB) and with available video head impulse test results were assessed. A total of 85 patients were included and divided into two groups based on HBOT compliance: the HBOT group (≥ 5 sessions, $n = 39$) and the non-HBOT group (< 5 sessions, $n = 46$). Semicircular canal function and treatment outcomes were compared. PSCC dysfunction was independently associated with about 14 dB poorer post-treatment mean hearing thresholds (76.00 ± 3.43 dB vs. 62.26 ± 3.75 dB, $p = 0.011$). In these patients with PSCC dysfunction, HBOT was associated with improved post-treatment mean hearing thresholds (74.32 ± 2.90 dB vs. 89.22 ± 3.17 dB; $p = 0.002$), with significant improvements at 0.5, 1, 2, and 4 kHz (all $p < 0.05$). PSCC dysfunction is associated with poorer hearing recovery in patients with profound SSNHL and may help identify a distinct clinical phenotype that is more likely to benefit from HBOT.

Keywords Sudden sensorineural hearing loss, Hyperbaric oxygen therapy, Semicircular canal, Vestibular disease, Prognostic factor

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency defined as a rapid onset of hearing loss of ≥ 30 dB across at least three contiguous frequencies within 72 h¹. The annual incidence is estimated at 10 to 30 per 100,000 individuals and increases with age^{2,3}. Despite extensive investigations, the etiology of SSNHL remains largely idiopathic in most cases⁴.

The major treatment strategy for SSNHL is systemic corticosteroids, combined with intra-tympanic dexamethasone injection (ITDI)^{1,4–6}. Steroids may have an effect on recovery from SSNHL, as inflammation and reactive oxygen species lead to inner ear hair cell loss and steroids can control them^{7–9}. In addition, hyperbaric oxygen therapy (HBOT) is also considered an effective treatment, especially in profound hearing loss^{10–12}.

Although treatment is administered appropriately, about 40% of patients do not recover^{13,14}. Many factors have been identified as being associated with SSNHL^{14,15}. The severity of hearing loss is a well-established prognostic factor, with profound SSNHL being associated with significantly poorer auditory recovery¹⁴. Vestibular symptoms such as vertigo are also negative prognostic indicators, suggesting more extensive inner ear involvement¹⁴. In recent studies, dysfunction of the posterior semicircular canal (PSCC), identified through the video head impulse test (vHIT), has emerged as a possible marker of poor prognosis¹⁶. This relationship may be explained by their shared end-arterial supply: the PSCC receives blood from the posterior vestibular artery, a terminal branch of the common cochlear artery, whereas the cochlea is perfused by another terminal branch of the same vessel. Because the inner-ear circulation lacks collateral pathways, any ischemia in the common cochlear

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artery territory can simultaneously compromise both the cochlea and the PSCC. Thus, PSCC involvement may reflect localized vascular compromise and serve as a clinical marker of poor prognosis in SSNHL.

HBOT has been introduced as an optional treatment for SSNHL, particularly in cases with limited response to steroid therapy^{17,18}. However, the optimal timing, target population, and overall efficacy of HBOT remain subjects of ongoing debate^{11,12,19–22}. The therapeutic rationale for HBOT lies in its ability to enhance oxygen delivery to the inner ear, reduce ischemic injury, and promote recovery in cases with suspected vascular etiology^{11,12}.

Given the vascular vulnerability of the PSCC and its association with poor hearing outcomes¹⁶, it is plausible that patients exhibiting PSCC dysfunction may represent a distinct subgroup of SSNHL with underlying ischemic etiology. Although a previous study identified that arterial stiffness was associated with poor prognosis of SSNHL, treatment for ischemic etiology was not comprehensively evaluated²³. Since HBOT is thought to exert its therapeutic effect by improving oxygenation and reducing ischemic damage within the inner ear, one may hypothesize that this subgroup could particularly benefit from adjunctive HBOT^{11,12}. This raises an important clinical question: can PSCC dysfunction serve not only as a prognostic marker for poor recovery, but also as a predictor of favorable response to HBOT in patients with profound SSNHL?

The present study aims to investigate whether PSCC dysfunction can serve as a prognostic biomarker for poor hearing recovery in patients with profound SSNHL, and to assess whether the presence of vestibular involvement can guide more appropriate use of HBOT in clinical practice.

Materials and methods

Inclusion of subjects

Patients diagnosed with SSNHL and hearing thresholds of 80 dB or higher—categorized as profound hearing loss²⁴—and treated at the tertiary referral centers between October 1, 2020 and August 15, 2025 were included. For the control group, patients managed at the secondary referral center where HBOT was unavailable, from January 2016 to December 2020, were enrolled. During this period, data were retrospectively reviewed, and patients with abnormal tympanic membrane findings on the affected side or those who lacked vHIT data were excluded. In terms of age, sex, underlying disease (hypertension and diabetes), and initial hearing thresholds, the two centers were comparable (age, $p = 0.432$; sex, $p = 0.112$; hypertension, $p = 0.822$; diabetes, $p = 0.147$; initial hearing thresholds, $p = 0.274$). Both centers followed a similar treatment approach, initiating systemic steroid therapy with prednisolone 60 mg followed by tapering, with salvage intratympanic dexamethasone injection when indicated, except for the addition of HBOT. There were no significant differences between the two centers in the time from onset to treatment ($p = 0.257$) or in the completion of ITDI cycles ($p = 0.118$).

This study was conducted after receiving approval from the Institutional Review Board of Hanyang University Hospital and Hanyang University Guri Hospital (IRB No. 2025-05-044, 2021-03-008). The requirement for informed consent was waived by the Institutional Review Board of Hanyang University Hospital and Hanyang University Guri Hospital due to the retrospective nature of the study. All methods were carried out in accordance with relevant guidelines and regulations.

Audiological test and video Head Impulse Test (vHIT)

The hearing thresholds at 0.25 kHz, 0.5 kHz, 1 kHz, 2 kHz, 4 kHz, and 8 kHz were evaluated. The mean hearing threshold was calculated using a weighted 6-division method, with the following formula: $(0.5 \text{ kHz} + 2 \times 1 \text{ kHz} + 2 \times 2 \text{ kHz} + 4 \text{ kHz})$ divided by 6. In addition, complete hearing loss was defined as 95 dB or greater²⁴. In addition, the word recognition score (WRS) was evaluated by experienced audiologists.

The vestibulo-ocular reflex (VOR) gains at high frequencies for all three semicircular canals were assessed using the video head impulse test (vHIT) with the ICS Impulse system (GN Otometrics, Taastrup, Denmark). The vHIT was performed as part of the pre-treatment evaluation. More than 20 unpredictable head impulses were delivered in the plane of each semicircular canal (SCC), with peak velocities ranging from 100° to 300°/second. The presence of catch-up saccades or a gain of less than 0.8 in the LSCC, and less than 0.7 in the PSCC or ASCC, was considered indicative of dysfunction^{25,26}.

Hyperbaric oxygen therapy (HBOT)

HBOT was administered at 2.0 ATA for 90 min. Previous studies indicate that the therapeutic efficacy of HBOT depends on cumulative exposure and that a minimum of 10 sessions is generally considered a complete treatment course¹². Since the number of patients who received 1–9 sessions was very small, and to ensure sufficient statistical power when analyzing dysfunction in each semicircular canal, patients were stratified using a five-session cutoff (half of the standard 10 sessions): an HBOT group, comprising individuals who completed at least 5 sessions, and a non-HBOT group, consisting of those who either received no HBOT or discontinued treatment before completing 5 sessions. To further validate the primary findings, a sensitivity analysis was performed by re-stratifying patients into three distinct groups based on the treatment dose: a control (0 sessions), and two treatment cohorts based on dose—insufficient (1–9 sessions) and sufficient (≥ 10 sessions) HBOT groups.

Treatment outcomes

Hearing recovery was evaluated using hearing thresholds measured at the final follow-up visit, including only those obtained thirty days or more after treatment. The degree of hearing recovery was classified into four categories according to Siegel's criteria as follows: (1) complete recovery, final hearing thresholds better than 25 dB regardless of the amount of hearing gain; (2) partial recovery, 15 dB or more hearing gain and final hearing thresholds between 25 and 45 dB; (3) slight recovery, 15 dB or more hearing gain and final hearing worse than 45 dB; and (4) no improvement, less than 15 dB hearing gain²⁷.

Statistical analysis

Chi-square test and Fisher's exact test were conducted to evaluate relationships between categorical variables. Student's t-test and Wilcoxon rank-sum test were used to compare continuous variables between two groups. For analyses based on dysfunction of each SCC, patients with combined SCC dysfunction were classified as normal or abnormal for each SCC individually. Multivariable analysis was conducted after adjusting for variables affecting final hearing outcomes. ANCOVA and Quade's ANCOVA were applied for multivariable comparisons between the two groups, and ordinal logistic regression was performed to adjust for covariates in the analysis of Siegel's criteria. To conduct multivariable analysis for continuous variables, a linear regression model was employed. All statistical analyses were performed using IBM SPSS Statistics, version 25.0 (IBM Corp., Armonk, NY, USA).

Results

Inclusion of subjects and demographics

Of the 133 patients with profound SSNHL without tympanic membrane abnormality, two were excluded due to vestibular schwannoma. Among the remaining patients, 105 with a follow-up duration of more than 4 weeks were selected. Subsequently, 18 patients who did not have vHIT data and two patients with uncontrolled diabetes were excluded. Finally, 85 patients were included in the analysis (Fig. 1).

The mean age of the included subjects was 55.75 ± 15.24 years, comprising 45 males and 40 females. The proportions of hypertension and diabetes were 29.41% ($n = 25$) and 30.59% ($n = 26$), respectively. Complete hearing loss at initial visit occupied 22.35% ($n = 19$).

Systemic steroids or ITDI were administered to all patients. Systemic steroids were administered 2.52 ± 2.83 days after onset. The ITDI was administered to all but two patients and the completion rate of ITDI (≥ 3 times) was 91.76%. In total, 39 patients received hyperbaric oxygen therapy (HBOT) consisting of five or more sessions, whereas the remaining 46 patients (including 3 patients receiving HBOT fewer than five times) either did not undergo HBOT or discontinued treatment before completing five sessions. Among patients who received HBOT, the mean time from SSNHL onset to treatment initiation was 8.77 ± 16.61 days (median, 5 days; interquartile

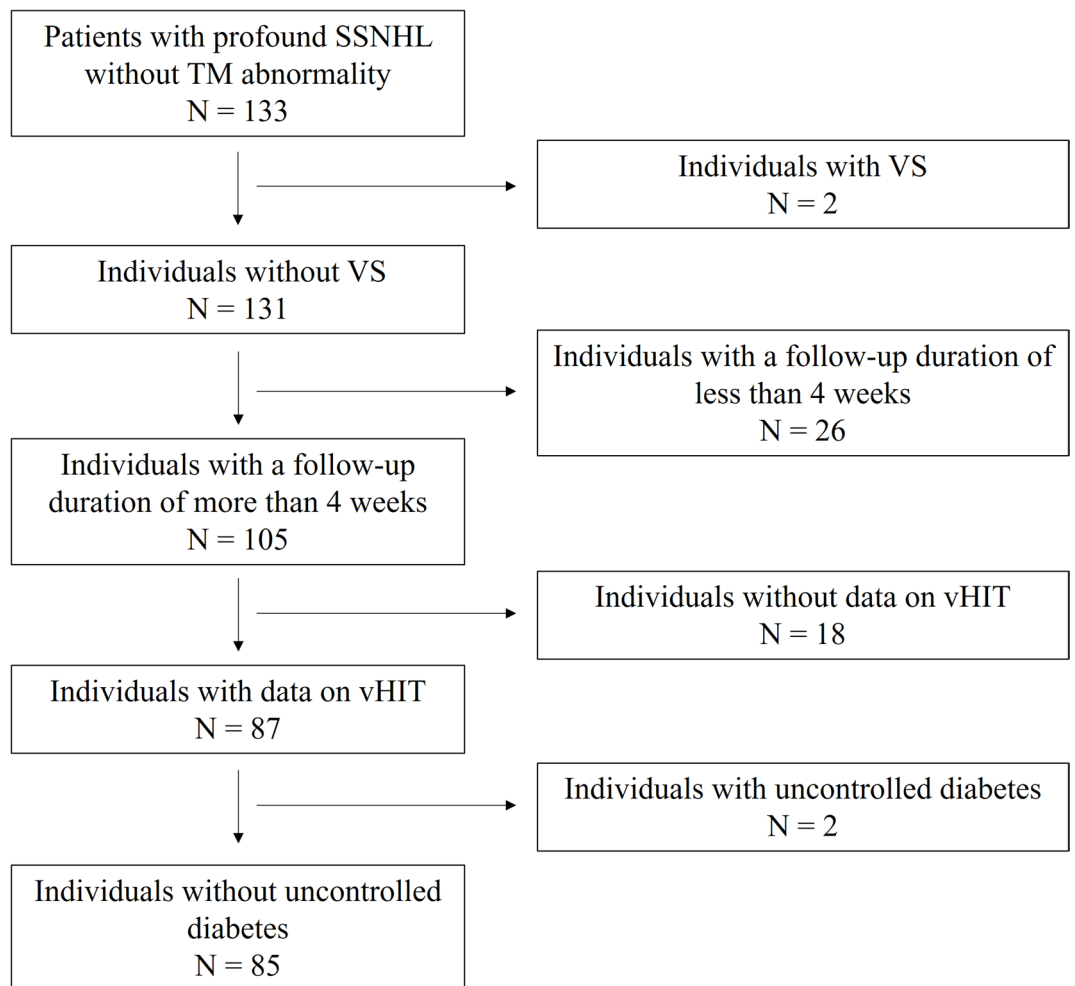


Fig. 1. Inclusion of subjects. SSNHL, sudden sensorineural hearing loss; TM, tympanic membrane; VS, vestibular schwannoma; N, number; vHIT, video head impulse test.

range, 2–8 days). The 88.24% ($n=75$) patients received treatment during hospitalization, while 11.76% ($n=10$) were treated as outpatients. The mean follow-up period was 311.89 ± 405.54 days after onset.

Between the HBOT and non-HBOT groups, age ($p=0.382$), sex ($p=0.555$), hypertension ($p=0.800$), diabetes ($p=0.166$), baseline audiological outcomes such as hearing thresholds ($p=0.443$) and WRS ($p=0.228$), systemic steroid initiation ($p=0.313$), and completion of ITDI ($p=0.593$) were not significantly different. In contrast, the proportions of dizziness ($p=0.022$), follow-up duration ($p=0.043$), ASCC dysfunction ($p=0.001$), and LSCC dysfunction ($p=0.001$) were significantly different between the groups (Table 1).

Among the demographic variables and medical history, age, diabetes, and initial complete hearing loss were associated with final hearing thresholds (age: $p<0.001$; diabetes: $p=0.026$; initial complete hearing loss, $p<0.001$).

Vestibular characteristics and hearing recovery according to vestibular dysfunction

The proportion of patients with dizziness was 61.18% ($n=52$) and initial complete hearing loss was significantly more frequent in individuals with dizziness (34.62%, $p<0.001$). The proportion of concurrent dysfunction in ASCC, LSCC, and PSCC was 16.47% ($n=14$), 41.18% ($n=35$), and 54.12% ($n=46$), respectively.

In terms of initial hearing loss according to vestibular dysfunction, PSCC dysfunction was associated with poorer initial hearing thresholds (100.45 ± 10.53 vs. 95.20 ± 9.18 dB in ears with normal PSCC function, $p=0.018$), while ASCC and LSCC dysfunction did not show an association with initial hearing thresholds (ASCC dysfunction: 100.83 ± 8.83 vs. 97.52 ± 10.46 dB in ears with normal ASCC function, $p=0.275$; LSCC dysfunction: 98.21 ± 10.71 vs. 97.98 ± 9.98 dB in ears with normal LSCC function, $p=0.917$) (Table 2). Initial complete hearing loss was more prevalent in individuals with PSCC dysfunction (34.78%) than in those without (PSCC: 7.69%, $p=0.003$), respectively. However, dysfunction of the LSCC and ASCC did not show a statistically significant association with the initial complete hearing loss (LSCC: $p=0.093$; ASCC: $p=0.505$). Furthermore,

	Overall ($n=85$)	HBOT ($n=39$)	Non-HBOT ($n=46$)	<i>p</i> -value
Age (year)	55.75 ± 15.24	57.33 ± 14.88	54.41 ± 15.57	0.382
Gender (male/Female)	45 : 40	22 : 17	23 : 23	0.555
Comorbidity				
Hypertension (%)	29.41% ($n=25$)	30.77% ($n=12$)	28.26% ($n=13$)	0.800
Diabetes (%)	30.59% ($n=26$)	23.08% ($n=9$)	36.96% ($n=17$)	0.166
Initial hearing status				
Hearing threshold (dB)	97.92 ± 10.27	96.99 ± 10.49	98.71 ± 10.13	0.443
Complete hearing loss (%)	22.35% ($n=19$)	23.08% ($n=9$)	21.74% ($n=10$)	0.883
Word recognition score (%)	0.89 ± 3.89	1.49 ± 5.31	0.40 ± 1.96	0.228
Dizziness (%)	61.18% ($n=52$)	74.36% ($n=29$)	50.00% ($n=23$)	0.022
Semicircular canal function				
ASCC dysfunction (%)	16.47% ($n=14$)	30.77% ($n=12$)	4.35% ($n=2$)	0.001
LSCC dysfunction (%)	41.18% ($n=35$)	58.97% ($n=23$)	26.09% ($n=12$)	0.001
PSCC dysfunction (%)	54.12% ($n=46$)	64.10% ($n=25$)	45.65% ($n=21$)	0.065
Days to systemic steroid initiation	2.52 ± 2.83	2.18 ± 2.26	2.80 ± 3.24	0.313
Completion of ITDI (≥ 3)	91.76%	92.31%	91.30%	0.593
Follow-up periods	311.89 ± 405.54	216.73 ± 228.64	377.61 ± 481.22	0.043
Hearing recovery				
Final hearing threshold (dB)	69.70 ± 28.69	67.82 ± 24.52	71.29 ± 31.98	0.582
Siegel's criteria (%)				0.202
Complete recovery	8.24% ($n=7$)	2.56% ($n=1$)	13.04% ($n=6$)	
Partial recovery	11.76% ($n=10$)	15.38% ($n=6$)	8.70% ($n=4$)	
Slight improvement	44.71% ($n=38$)	51.28% ($n=20$)	39.13% ($n=18$)	
No improvement	35.29% ($n=30$)	30.77% ($n=12$)	39.13% ($n=18$)	

Table 1. Demographics, medical history, and vestibular manifestations in the overall group and a comparison between the HBOT and non-HBOT groups. HBOT, hyperbaric oxygen therapy; n, number; ITDI, intratympanic dexamethasone injection; ASCC, anterior semicircular canal; LSCC, lateral semicircular canal; PSCC, posterior semicircular canal; Statistical analyses were conducted between the HBOT and non-HBOT groups, and statistically significant *p*-values are indicated in bold.

Vestibular problem (n of presence / n of absence)	Initial hearing thresholds (dB)			Final hearing thresholds (dB)		
	Presence	Absence	p-value	Presence	Absence	p-value
Dizziness (n = 52 / n = 33)	100.32 ± 10.01	94.14 ± 9.64	0.006	76.87 ± 24.49	58.38 ± 31.44	0.003
Semicircular canal dysfunction						
ASCC dysfunction (n = 14 / n = 71)	100.83 ± 8.83	97.52 ± 10.46	0.275	71.46 ± 19.90	69.35 ± 30.22	0.749
LSCC dysfunction (n = 35 / n = 50)	98.21 ± 10.71	97.98 ± 9.98	0.917	79.06 ± 20.33	63.14 ± 31.90	0.006
PSCC dysfunction (n = 46 / n = 39)	100.45 ± 10.53	95.20 ± 9.18	0.018	81.12 ± 19.91	56.22 ± 31.71	<0.001

Table 2. Initial mean hearing thresholds and final mean hearing thresholds according to dizziness and semicircular canal dysfunction. n, number; ASCC, anterior semicircular canal; LSCC, lateral semicircular canal; PSCC, posterior semicircular canal. Statistically significant values were indicated in bold.

the initial WRS was not significantly different according to ASCC dysfunction ($p=0.143$), LSCC dysfunction ($p=0.168$), or PSCC dysfunction ($p=0.108$).

Final hearing thresholds and WRS were analyzed according to SCC dysfunction, and LSCC and PSCC dysfunction were significantly associated with worse final post-treatment hearing thresholds (LSCC dysfunction: 79.06 ± 20.33 vs. 63.14 ± 31.90 dB in normal LSCC function, $p=0.006$; PSCC dysfunction: 81.12 ± 19.91 vs. 56.22 ± 31.71 dB in normal PSCC function, $p<0.001$) and WRS (LSCC dysfunction: 18.66 ± 28.44 vs. $37.78 \pm 41.37\%$ in normal LSCC function, $p=0.013$; PSCC dysfunction: 15.70 ± 25.84 vs. $46.67 \pm 42.49\%$ in normal PSCC function, $p<0.001$). In contrast, ASCC dysfunction did not show an association with final post-treatment hearing thresholds (71.46 ± 19.90 vs. 69.35 ± 30.22 dB in normal ASCC function; $p=0.749$) or WRS (22.29 ± 28.26 vs. $31.41 \pm 39.19\%$ in normal ASCC function, $p=0.533$) (Table 2).

Multivariable analysis controlling for variables affecting hearing outcomes, including age, diabetes, and initial complete hearing loss, demonstrated that PSCC dysfunction was independently associated with both a poorer post-treatment hearing threshold (76.00 ± 3.43 dB vs. 62.26 ± 3.75 dB in ears with normal PSCC function, $p=0.011$) and WRS (42.62 ± 5.61 vs. $19.13 \pm 5.12\%$ in ears with normal PSCC function $p=0.004$). In contrast, LSCC function was not significantly associated with either the post-treatment threshold (LSCC dysfunction: 73.99 ± 3.99 dB; normal LSCC function: 66.69 ± 3.31 dB, $p=0.172$) or WRS (LSCC dysfunction: $23.24 \pm 6.02\%$; normal LSCC function: $34.57 \pm 5.00\%$, $p=0.161$) (Fig. 2A).

Frequency-specific multivariable analysis further showed that PSCC dysfunction was associated with higher thresholds at 0.25, 2, 4, and 8 kHz (all $p<0.05$), while LSCC dysfunction showed significant differences at 0.25, 4, and 8 kHz (all $p<0.05$) (Fig. 2B and C).

Figure 3 showed that PSCC dysfunction is consistently linked to poorer hearing-recovery outcomes. Using Siegel's criteria, patients with PSCC dysfunction showed a markedly higher proportion of "no" or "slight" recovery and an absence of complete recovery (overall $p<0.001$, significant after Bonferroni correction), a pattern also observed in subjects with LSCC dysfunction ($p=0.005$, significant after Bonferroni correction).

Ordinary logistic regression analysis, adjusted for age, diabetes, and initial complete hearing loss, showed that LSCC and PSCC dysfunction were significantly associated with poorer hearing recovery ($p=0.026$ and $p=0.036$, respectively).

Effect of HBOT on hearing thresholds according to vestibular dysfunction

In the overall study population, HBOT did not significantly change the final hearing threshold (Table 1). When the analysis was broken down by SCC function, a distinct therapeutic advantage was identified for subjects with PSCC dysfunction (Table 3), whereas such an association was not observed in other SCC dysfunctions.

After adjustment for age, diabetes, and initial complete hearing loss, HBOT was associated with an ~15 dB lower (better) final post-treatment hearing threshold in the patients with PSCC dysfunction (74.32 ± 2.90 dB vs 89.22 ± 3.17 dB in non-HBOT group; $p=0.002$) (Fig. 4A). Frequency-by-frequency, the benefit centered on 0.5 kHz ($p=0.027$), 1 kHz ($p=0.002$), 2 kHz ($p=0.044$) and 4 kHz ($p=0.008$) (Fig. 4B). When the final hearing outcome was assessed according to Siegel's criteria, HBOT group exhibited a lower proportion of 'no improvement' compared to the non-HBOT group, although the difference was not statistically significant (Fig. 4C). Although the difference in WRS according to HBOT status was 13%, it was not statistically significant (HBOT: $21.65 \pm 5.10\%$; non-HBOT: $8.61 \pm 5.59\%$, $p=0.187$).

To evaluate the cumulative effect of HBOT, participants with PSCC dysfunction were classified into the control ($n=18$), insufficient HBOT ($n=7$), and sufficient HBOT groups ($n=21$). In the multivariable analysis, the sufficient HBOT group showed significantly better hearing thresholds than the control group at 1 kHz (69.45 ± 4.82 dB vs. 89.81 ± 5.23 dB, $p=0.004$), 2 kHz (77.64 ± 3.03 dB vs. 89.63 ± 3.28 dB, $p=0.018$), 4 kHz (80.26 ± 3.04 dB vs. 93.44 ± 3.30 dB, $p=0.007$), and the mean threshold (73.21 ± 3.22 dB vs. 88.01 ± 3.49 dB, $p=0.010$). Furthermore, the sufficient HBOT group had better mean thresholds at 2 kHz (77.64 ± 3.03 dB), 4 kHz (80.26 ± 3.04 dB), and 8 kHz (79.50 ± 2.53 dB) than the insufficient HBOT group (2 kHz: 93.76 ± 5.15 dB, $p=0.009$; 4 kHz: 100.36 ± 5.17 dB, $p=0.002$; 8 kHz: 89.50 ± 4.30 dB, $p=0.016$) (Fig. 4D and E).

Further sensitivity analysis was conducted using linear regression to examine the association between the number of HBOT sessions and final hearing thresholds in patients with PSCC dysfunction. In the multivariable analysis adjusting for age, diabetes, and initial complete hearing loss, final post-treatment hearing thresholds showed a negative association (better hearing outcomes) with the number of HBOT sessions ($\beta = -0.382$, 95% CI -1.738 to -0.445 , $p=0.001$) (Table 4).

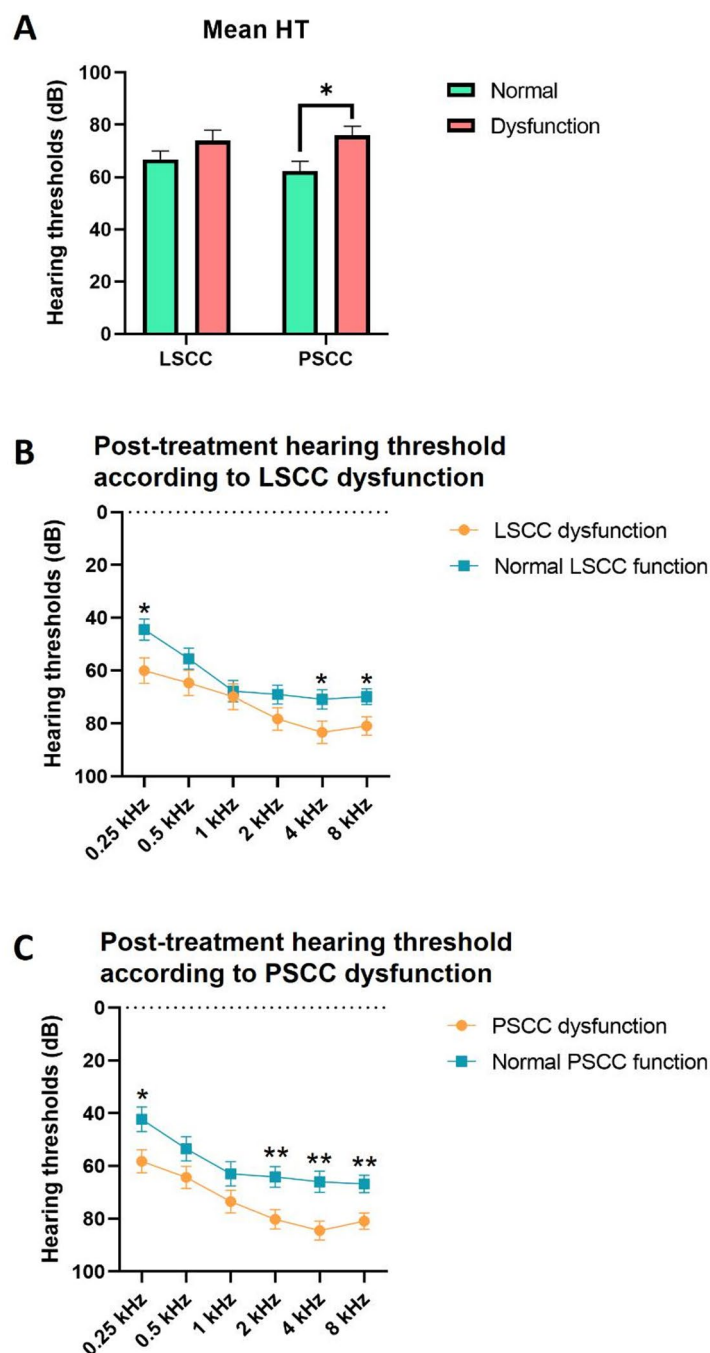


Fig. 2. Multivariable analysis of post-treatment hearing thresholds according to lateral semicircular canal (LSCC) or posterior semicircular canal (PSCC) function. (A) Mean hearing thresholds after treatment based on LSCC or PSCC function. Frequency-specific final hearing thresholds according to the presence or absence of LSCC (B) and PSCC (C) dysfunction. HT, hearing thresholds; LSCC, lateral semicircular canal; PSCC, posterior semicircular canal; *, $p < 0.05$; **, $p < 0.01$.

Discussion

The present study analyzed treatment outcome of profound SSNHL according to the HBOT treatment and vHIT results. The results can be summarized as follows: (1) PSCC dysfunction were associated with more severe baseline hearing loss; (2) LSCC and PSCC dysfunction independently predicted poorer post-treatment thresholds; and (3) only patients with PSCC dysfunction appeared to derive a significant (~ 15 dB) improvement in final hearing thresholds from HBOT, whereas HBOT may offer no significant benefit in ears without PSCC involvement.

The cochlea and vestibule receive their blood supply primarily from the labyrinthine artery. The labyrinthine artery gives rise to two major branches: the common cochlear artery and the anterior vestibular artery^{28,29}.

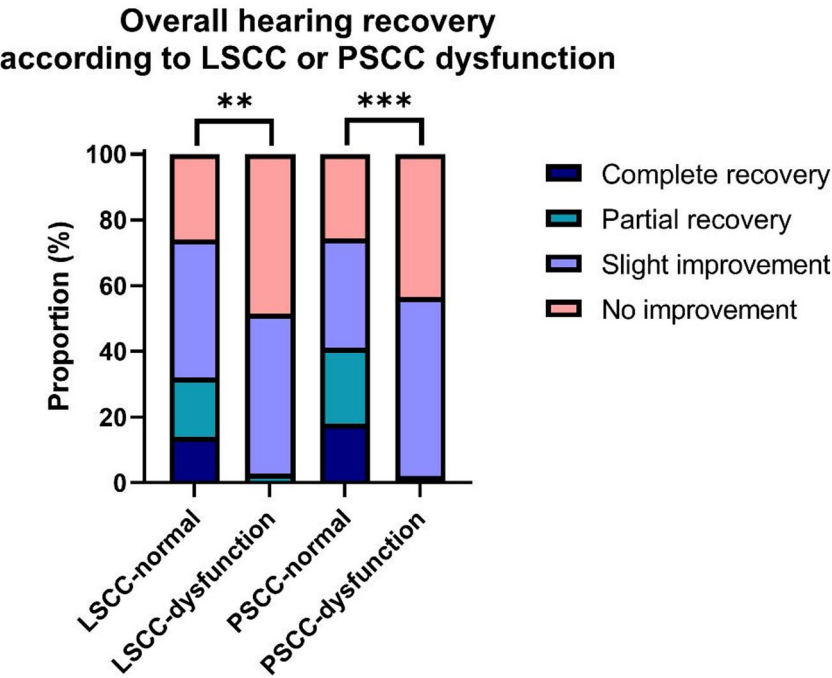


Fig. 3. Final hearing recovery based on Siegel's criteria according to lateral semicircular canal (LSCC) or posterior semicircular canal (PSCC) dysfunction. LSCC, lateral semicircular canal; PSCC, posterior semicircular canal; ASCC, anterior semicircular canal; *, $p < 0.0083$ after Bonferroni correction; ***, $p < 0.00017$ after Bonferroni correction.

Presence of SCC dysfunction (n of HBOT / n of non-HBOT)	Initial hearing thresholds			Final hearing thresholds		
	HBOT	Non-HBOT	p-value	HBOT	Non-HBOT	p-value
ASCC dysfunction						
Presence (n = 12 / n = 2)	100.56 ± 9.01	102.50 ± 10.61	0.791	69.69 ± 18.85	82.08 ± 31.23	0.791
Absence (n = 27 / n = 44)	95.80 ± 10.87	98.54 ± 10.20	0.308	66.99 ± 26.94	70.80 ± 32.28	0.311
LSCC dysfunction						
Presence (n = 23 / n = 12)	98.91 ± 10.91	96.88 ± 10.66	0.595	77.30 ± 20.81	82.43 ± 19.83	0.526
Absence (n = 16 / n = 34)	94.83 ± 9.50	99.36 ± 10.01	0.122	54.19 ± 23.50	67.35 ± 34.68	0.086
PSCC dysfunction						
Presence (n = 25 / n = 21)	99.83 ± 10.84	101.19 ± 10.37	0.655	75.53 ± 19.85	87.78 ± 18.26	0.036
Absence (n = 14 / n = 25)	92.44 ± 7.87	96.63 ± 9.63	0.222	54.05 ± 26.67	57.43 ± 34.67	0.141

Table 3. Comparison of initial and post-treatment hearing thresholds between the HBOT and non-HBOT groups within each semicircular canal function status. n, number; HBOT, hyperbaric oxygen therapy; SCC, semicircular canal; ASCC, anterior semicircular canal; LSCC, lateral semicircular canal; PSCC, posterior semicircular canal. Statistically significant values were indicated in bold.

The common cochlear artery further divides into the main cochlear artery and the vestibulo-cochlear artery, the latter of which supplies the PSCC, the basal turn of the cochlea, and the inferior saccule and utricle^{28,29}. Given that the diameter of labyrinthine artery was 0.2 mm and its branches had much smaller diameters, the cochlea and vestibule were vulnerable to ischemia from occlusion of those arteries³⁰. Therefore, occlusion of the common cochlear or vestibulo-cochlear artery may result in combined cochlear and vestibular dysfunction, manifesting as SSNHL with PSCC involvement. Several studies have shown that PSCC dysfunction is linked to more severe initial hearing loss and, in multivariate or cluster analyses, stands out as an independent predictor of hearing recovery in ISSNHL^{31,32}. A recent retrospective study of 49 severe-to-profound ISSNHL evaluated vestibular predictors of outcome, and the PSCC dysfunction on vHIT was the sole independent prognostic indicator of poor recovery, implicating a shared ischemic vulnerability of the cochlea and PSCC³³. The present findings showed approximately 14 dB worse final hearing thresholds and 23% worse WRS in patients with PSCC dysfunction, consistent with earlier studies. Interestingly, PSCC dysfunction showed a favorable response to HBOT, providing additional support to a vascular etiology for SSNHL.

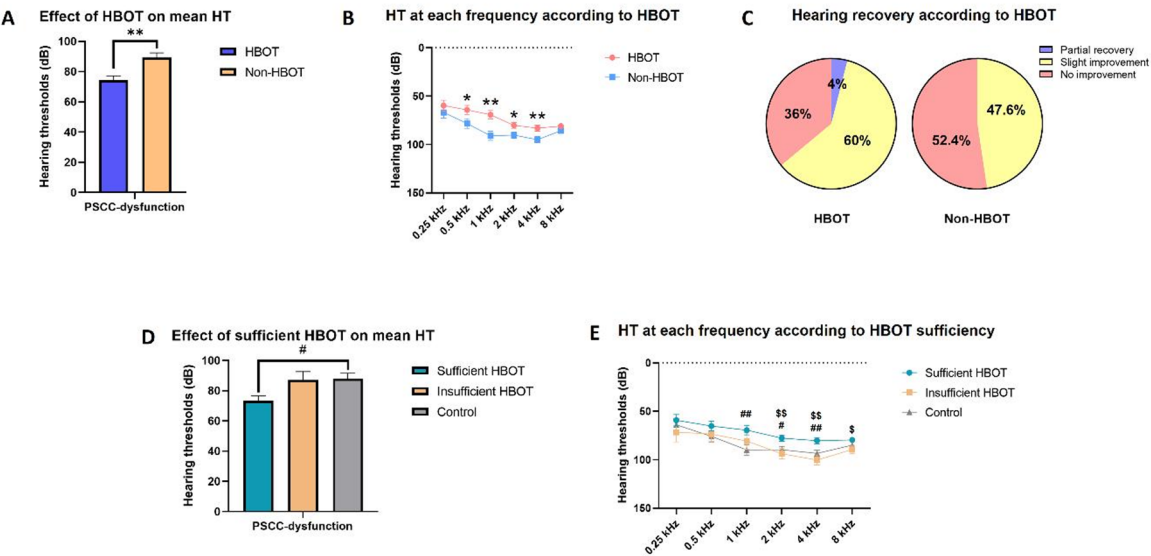


Fig. 4. Effects of hyperbaric oxygen therapy (HBOT) on final hearing thresholds and the degree of hearing recovery in patients with impaired posterior semicircular canal (PSCC) function. (A) Multivariable analysis of the effect of HBOT on mean hearing thresholds in patients with PSCC dysfunction. (B) Multivariable analysis of hearing thresholds at each frequency in patients with PSCC dysfunction, according to HBOT. (C) Final hearing recovery according to HBOT in patients with PSCC dysfunction based on Siegel’s criteria. (D) Multivariable analysis of the effect of sufficient (≥ 10 times) HBOT on mean hearing thresholds, comparing sufficient, insufficient (1–9 times), and no HBOT. (E) Multivariable analysis of hearing thresholds at each frequency in patients with PSCC dysfunction, according to adequacy of HBOT (sufficient ≥ 10 times; insufficient 1–9 times). HBOT, hyperbaric oxygen therapy; HT, hearing thresholds; *, $p < 0.05$ and **, $p < 0.01$ indicate significant differences related to HBOT status; #, $p < 0.05$ and ##, $p < 0.01$ indicate significant differences between the sufficient HBOT and control groups; \$, $p < 0.05$ and \$\$, $p < 0.01$ indicate significant differences between the sufficient and insufficient HBOT groups.

Variables	B	SE	β	95% CI	p-value
Age	0.299	0.168	0.224	-0.040, 0.637	0.082
Diabetes	-7.869	5.432	-0.197	-18.839, 3.102	0.155
Complete HL	26.348	4.629	0.637	16.999, 35.697	<0.001
N of the HBOT	-1.092	0.320	-0.382	-1.738, -0.445	0.001

Table 4. Multivariable linear regression analysis for final hearing thresholds. HL, hearing loss; N, number; HBOT, hyperbaric oxygen therapy.

HBOT may benefit SSNHL by transiently raising perilymph PO_2 , enhancing cochlear microcirculation, and dampening inflammatory cascades, thus preserving metabolically vulnerable hair cells and spiral ganglion neurons. Physiologically, pressurized 100% O_2 at 2–2.5 ATA drives a several-fold increase in dissolved plasma oxygen that diffuses across the round-window membrane and augments inner-ear tissue oxygenation, limits edema, and promotes angiogenesis and anti-apoptotic signaling³⁴. Clinically, an up-to-date umbrella review of nine systematic reviews/meta-analyses reported a pooled odds ratio of 1.37 (95% CI 1.17–1.61) favoring HBOT when added to standard therapy, albeit with moderate heterogeneity³⁵, while a 2021 JAMA meta-analysis likewise showed significant gains when HBOT was combined with steroids¹¹.

However, in the present cohort of profound SSNHL, HBOT failed to produce a statistically significant audiometric benefit. Retrospective series indicate that gains are greatest when treatment begins within 7–14 days of onset, whereas later initiation—common in routine practice—markedly attenuates efficacy³⁶. Other large retrospective data likewise show little overall advantage of HBOT in severe-to-profound SSNHL, yet report a clear improvement among patients with diabetes mellitus, a group prone to cochlear microangiopathy²⁰. These observations suggest that HBOT may exert its benefit only in a vascular-ischemic subtype of SSNHL—distinguished by systemic vasculopathy risk factors—rather than across all profound presentations. In this regard, the present study findings suggest that SSNHL with PSCC dysfunction may represent a subgroup that is more likely to benefit from HBOT. Anatomically, the cochlea and the PSCC are supplied by terminal branches of the common cochlear artery; this shared vascular territory likely underpins the association between PSCC dysfunction and the preferential improvement of hearing thresholds observed after HBOT. Furthermore, this study demonstrated that hearing thresholds can recover in ears with PSCC dysfunction when HBOT is

sufficiently administered (approximately ≥ 10 sessions), consistent with previous studies^{11,17,34,37}. Based on these findings, physicians should consider providing HBOT to patients with SSNHL and PSCC dysfunction and emphasize the importance of completing an adequate course of HBOT.

Despite appropriate medical therapy, including HBOT, most patients with profound SSNHL and PSCC dysfunction remained in the severe-loss range post-treatment; only one case achieved partial recovery, whereas 96% exhibited slight or no improvement. In addition, although patients who underwent HBOT showed approximately 13% higher WRS, the post-treatment WRS remained only slightly above 20%, and the difference was not statistically significant. Accordingly, individualized counselling that considers the better-hearing ear and introduces timely rehabilitation—cochlear implantation, CROS hearing aids, or bone-conduction devices—should be standard practice^{38,39}. Our data also corroborate earlier work identifying advanced age and a high initial pure-tone average as independent predictors of poor outcome¹⁴, likely reflecting age-related fragility of hair cells^{14,40} and cochlear microvasculature and the greater extent of cochlear injury in initially severe losses^{14,41}. Proactive discussion of these factors can facilitate realistic expectation setting and prompt referral for auditory rehabilitation.

This study also revealed that LSCC dysfunction may be associated with poor hearing recovery. As previous studies have identified, LSCC dysfunction may represent extensive damage to the inner ear, leading to poor hearing recovery^{42–44}. In contrast to PSCC dysfunction, subjects with LSCC dysfunction did not show significant hearing recovery after HBOT, possibly suggesting a pathophysiology distinct from vasculopathy. Further studies are needed to clarify the differing pathophysiology associated with combined SCC dysfunction.

To the best of our knowledge, this is the first study to implicate PSCC dysfunction in modulating the therapeutic response to HBOT in profound SSNHL. Several limitations, however, warrant consideration. First, because profound losses comprise only a small fraction of SSNHL patients, the sample size was necessarily limited, which may have limited adequate adjustment for confounders and reduced the statistical power to detect associations related to PSCC dysfunction. In particular, the insufficient HBOT subgroup was notably small, which may limit the interpretability of the dose-response analysis. Second, the retrospective design yielded non-uniform vestibular testing and considerable variation in the interval between symptom onset and HBOT initiation, introducing potential measurement and timing biases. Consequently, analysis of time to HBOT initiation as a covariate was warranted but could not be performed due to the insufficient sample size. Third, vHIT assessments were not repeated after therapy, precluding evaluation of PSCC functional recovery. Fourth, the use of historical controls from a different period and clinical setting may introduce potential confounding, and grouping patients according to HBOT adherence rather than randomization may have resulted in selection bias. Fifth, the detailed nature of vestibular symptoms could not be considered due to the limited number of patients. Sixth, the follow-up period varied substantially among patients because the follow-up intervals were not uniform, and therefore the final follow-up was used as the assessment time point. Consequently, a few patients were followed for longer periods, which may have influenced the evaluation of final hearing outcomes. Prospective, adequately powered studies with larger sample sizes, standardized vestibular protocols, fixed HBOT regimens, and uniform follow-up protocols are needed to validate these observations and to delineate which vestibular phenotypes derive the greatest benefit from HBOT.

Conclusion

PSCC dysfunction may represent a distinct clinical phenotype in profound SSNHL, associated with poorer hearing recovery yet greater apparent benefit from HBOT. vHIT-based assessment of PSCC function may therefore have value in identifying patients more likely to benefit from adjunctive HBOT. Prospective controlled studies are needed to validate this predictive role and clarify its implications for personalized treatment.

Data availability

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request and with approval from the Institutional Review Board.

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Competing interests

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