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Original Article

Insights Into Responses to Caloric and Head Impulse Stimulation in Lateral Semicircular Canal Benign Paroxysmal Positional Vertigo

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Objectives. The aim of this study was to investigate the pathophysiology of persistent geotropic direction-changing positional nystagmus (pGeo DCPN) by analyzing caloric responses and the clinical course in patients with pGeo DCPN and other variants of lateral semicircular canal benign paroxysmal positional vertigo (LSCC BPPV).

Methods. In this prospective case-control study, 101 patients diagnosed with pGeo DCPN (pGeo group, n=34), persistent apogeotropic (pApo) DCPN (pApo group, n=40), or transient geotropic (tGeo) DCPN (tGeo group, n=27) involving the LSCC were enrolled. The video head impulse test (vHIT) and caloric test were performed at diagnosis. If one or both tests were abnormal, follow-up testing was conducted after nystagmus resolution. Differences in test results and clinical courses among the LSCC BPPV types were analyzed and compared.

Results. The mean disease duration was significantly longer in patients with pGeo (23.0 days) than those with pApo (8.0 days) and tGeo (9.0 days) ($P<0.005$). All patients demonstrated normal vHIT gain. The pGeo group showed the highest canal paresis (CP) value (vs. pApo: $P=0.002$; vs. tGeo: $P<0.001$) and a higher frequency of abnormal CP (61.8%) than the pApo (22.5%) and tGeo (11.1%) groups. CP was predominantly ipsilesional (85.7%) in the pGeo group. Follow-up caloric tests indicated decreased CP in 72.7% of pGeo patients, with normalization in 36.4% following the disappearance of nystagmus.

Conclusion. The clinical course, caloric tests, and vHIT results indicate distinct pathophysiological mechanisms for different LSCC BPPV types. Cupular deflection due to buoyancy in pGeo DCPN plausibly leads to CP while preserving vHIT integrity.

Keywords. *Light Cupula; Benign Paroxysmal Positional Vertigo; Caloric Test; Vestibular Function Test*

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INTRODUCTION

Approximately 10%–30% of all benign paroxysmal positional vertigo (BPPV) cases originate from the lateral semicircular canal (LSCC) [1]. A subtype of LSCC BPPV, known as persistent direction-changing positional nystagmus (DCPN), is characterized by horizontally directed persistent nystagmus that changes direction with head rotations of 90° to the right or left in the supine position [2]. Persistent DCPN is categorized into apogeo-

tropic and geotropic types. Persistent apogeotropic (pApo) DCPN directs opposite to gravity during head rotation and is primarily caused by LSCC cupulolithiasis, also termed "heavy cupula", where otoconia adhere to the cupula [3]. In this situation, the cupula's specific gravity exceeds that of the LSCC endolymph, causing cupular deflection toward the utriculofugal direction in lesion-side-down positions, generating nystagmus away from the lesion side. In other head positions, the nystagmus is directed toward the lesion side because the cupula is deflected in the utriculopetal direction. Nystagmus persists due to the cupula's inability to return to neutral because of its increased specific gravity.

Conversely, persistent geotropic (pGeo) DCPN, known as "light cupula", aligns with gravitational force during head movements. Three primary theories explain pGeo DCPN: the lighter cupula theory posits that alcohol, lighter than water, diffuses into the cupula faster than into endolymph, temporarily reducing the cupula's density [4,5]. Pathological cupular degeneration may produce similar effects. The heavy endolymph theory proposes that acute inner ear insults, such as labyrinthine hemorrhage, hypoperfusion, or inflammation, increase endolymph density [6-8]. Leakage of blood plasma proteins or changes in macromolecules like sulfate proteoglycans might contribute to this effect [9,10]. Lastly, the light debris theory suggests lighter debris attaching to the cupula alters its density, supported by abrupt vertigo onset and typically unilateral involvement [11-14]. Hiruma and Numata [15] introduced the light cupula theory in 2004. Kim et al. [6] reported that 14.2% of patients with geotropic DCPN corresponded to light cupula, higher than previously anticipated .

The caloric test, a standard vestibular function test, separately evaluates the vestibulo-ocular reflex of each ear within a very low-frequency stimulation range. It employs thermal stimulation to induce convective flow of endolymph in the LSCC, causing cupular deflection and subsequently horizontal nystagmus. Canal paresis (CP) is calculated by comparing the maximum angular velocity of nystagmus induced by thermal stimulation of the right and left LSCC [16]. Therefore, an increased CP indicates an imbalance in bilateral LSCC function. Vestibular function tests, especially caloric tests, may show abnormal results during periods of cupulopathy, as explored by some researchers. Several studies have suggested that unilateral CP can occur in certain LSCC BPPV cases [17-20]. However, other studies have reported no significant differences in caloric responses between LSCC BPPV patients and controls [3,21,22]. Therefore, whether LSCC BPPV consistently exhibits CP remains a matter of debate, particularly given the scarcity of reports specifically on patients with pGeo DCPN BPPV.

Cupulopathy, including pApo and pGeo DCPN, theoretically may affect unilateral caloric responses due to the dynamic status of the cupula. Furthermore, increased specific gravity of the endolymph resulting from altered fluid composition, a suggested cause of pGeo DCPN, might alter caloric responses by influencing thermal expansion rates and convective efficiency. In contrast, the video head impulse test (vHIT), which assesses semicircular canal function in a high-frequency stimulation range by measuring the vestibulo-ocular reflex induced by rapid head rotations, likely remains normal in pGeo DCPN cases with light cupula, as angular acceleration significantly exceeds buoyant effects. Thus, this study aimed to investigate differences in caloric and vHIT responses at diagnosis among three LSCC BPPV groups and evaluate changes in these tests after nystagmus resolution, aiming to determine whether CP is temporary. Additionally, the study sought to infer the underlying pathophysiological mechanisms of pGeo and pApo DCPN LSCC BPPV based on the results and clinical courses. The findings will help clarify the distinct pathological mechanisms underlying different LSCC BPPV types.

HIGHLIGHTS

- All lateral semicircular canal benign paroxysmal positional vertigo patients showed normal video head impulse test result.
- In the persistent geotropic direction-changing positional nystagmus (pGeo DCPN) group, 61.8% showed unilateral caloric weakness.
- The majority of persistent apogeotropic DCPN and transient DCPN patients showed normal caloric test results.
- Caloric paresis was predominantly ipsilateral in pGeo DCPN patients.
- The degree of caloric asymmetry decreased after the resolution of pGeo DCPN in most patients with abnormal initial canal paresis values.

MATERIALS AND METHODS

Ethics statement

The study design was approved by the Institutional Review Board of Severance Hospital (No. 4-2021-1159). The requirement for informed consent was waived by the ethics committee due to the use of standard diagnostic approaches and treatment methods.

Patient enrollment

Between December 1, 2020, and May 1, 2023, patients diagnosed with three types of LSCC at Severance Hospital (Seoul, Korea), Gangnam Severance Hospital (Seoul, Korea), St. Vincent Hospital (Suwon, Korea), Myongji Hospital (Goyang, Korea), and Ilsan Paik Hospital (Goyang, Korea) were consecutively enrolled in this multi-center, prospective study. The three different types of LSCC BPPV are as follows; patients with persistent geotropic DCPN (pGeo), those with persistent apogeotropic DCPN (pApo), and those with transient geotropic DCPN (tGeo).

The inclusion criteria were as follows: (1) patients who agreed to undergo caloric test and vHIT on the same day of diagnosis

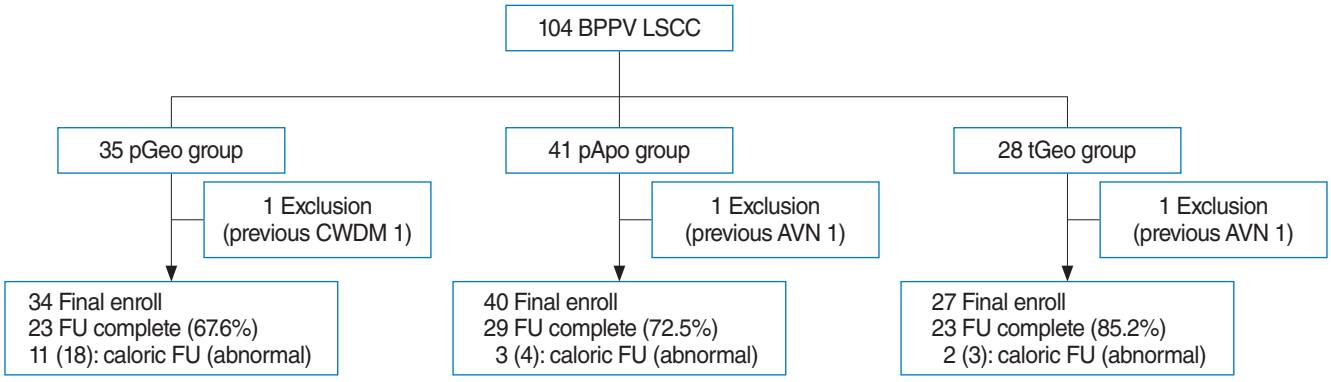

Fig. 1. Patient enrollment results for each group. A total of 104 patients were enrolled, and three patients were excluded (one per group) due to a surgical history of canal-wall-down mastoidectomy or a prior diagnosis of acute vestibular neuritis. BPPV, benign paroxysmal positional vertigo; LSCC, lateral semicircular canal; pGeo group, persistent geotropic direction-changing positional nystagmus group; pApo group, persistent apogeotropic direction-changing positional nystagmus group; tGeo group, transient geotropic direction-changing positional nystagmus group; CWDM, canal-wall-down mastoidectomy; AVN, acute vestibular neuritis; FU, follow-up.

and (2) patients who met the diagnostic criteria of pGeo, pApo, and tGeo DCPN (refer to "Diagnosis of pGeo or pApo DCPN and study design" subheading). Exclusion criteria included patients with (1) a previous history of vestibular disorders such as acute vestibular neuritis, labyrinthitis, Meniere's disease, and migraine, (2) a history of chronic otitis media or otologic surgery, (3) BPPV combined with acute sensorineural hearing loss, (4) change in nystagmus pattern during follow-up, (5) multi-canal BPPV, and (6) central lesion revealed by neurological exams or imaging studies [23]. The caloric and vHIT tests were routinely conducted on the same day of diagnosis. Patients with abnormal tests results were recommended to undergo follow-up tests after the disappearance of the nystagmus. Patient enrollment is shown in Fig. 1.

Diagnosis of pGeo or pApo DCPN and study design

All patients were asked about the onset time of positional vertigo before visiting our hospital through a detailed interview at initial visit. Then, patients underwent neurological and neuro-otological examinations with videonystagmography after history taking. The diagnosis of tGeo and pApo DCPN were made according to the diagnostic criteria for canalolithiasis and cupulolithiasis of the horizontal canal BPPV proposed by the Barany Society [24]. tGeo DCPN was diagnosed if the geotropic direction-changing nystagmus lasted <1 minute and pApo if the apogeotropic direction-changing nystagmus lasted >1 minute on the supine roll test. The pGeo DCPN was diagnosed if the geotropic nystagmus persisted for >1 minute on supine roll test and had a null point [25]. The affected side was determined by the bow and lean test, wherein the nystagmus was directed toward the affected side in the bowing position and toward the healthy side in the leaning position [26]. After diagnosis, a bithermal caloric test and vHIT were conducted before the repositioning maneuver on the same day. Patients with tGeo DCPN and pApo DCPN were treated using the Lempert maneuver [27] and cupulolith repositioning maneuver [28]. For patients with pGeo DCPN, we planned to adopt a wait-and-see approach rather than a repositioning maneuver because there is no universally accepted repositioning maneuver. This decision is also based on the prospective case controlled study by Ban et al. [29], which reported that the canal repositioning procedure for pGeo DCPN was not useful [30]. Regular follow-up appointments were scheduled at 1-week intervals at most participant hospitals until the nystagmus completely disappeared. Diseases duration was defined as the period from the onset of symptoms to the disappearance of positional nystagmus. Only one hospital was scheduled for short-term (1 or 2 days) follow-up clinic sessions until the nystagmus completely disappeared. For patients with abnormal caloric and vHIT tests results at the first visit, follow-up tests were recommended after the nystagmus resolved. The disease duration This protocol was established uniformly as clinical routine in participating clinics.

Vestibular function test

A conventional bithermal caloric test (Visual Eyes VNG; Micromedical Technologies) was performed for each ear using water irrigators, with the patient in a supine position with 30° of head flexion. Each ear canal was stimulated for 30 seconds, with a resting interval of 5 minutes between irrigation cycles. During irrigation, the induced nystagmus and slow-phase velocity were monitored using a video nystagmography system. CP was calculated using the Jongkees formula. A CP value ≥25% was regarded as pathological [23,31,32]. CP was calculated after compensating for spontaneous nystagmus if spontaneous nystagmus was detected.

For the vHIT (ICS Impulse, GN Otometrics), default software settings were used. To evaluate LSCC, the participants were seated upright with 30° of head flexion and instructed to gaze at a dot on a wall at a distance of 1 m. The head impulses were conducted by the same right-handed examiner with a peak velocity

range of 200°/sec–250°/sec, rotation angle of 15°–20°, and duration of 150–200 ms. A minimum of 20 horizontal head impulses were delivered randomly in the right and left directions. The vestibulo-ocular response mean gain, calculated automatically, and the presence of catch-up saccades were used as evaluation parameters in this study. A gain value of >0.8 for LSCC is considered normal [33,34].

Sample size and Statistical analysis

The sample size was determined by a priori power analysis for analysis of variance, with an effect size of 0.4, an alpha-error probability of 0.05, a beta-error probability of 0.2, and three groups. The total sample size was calculated to be 66, with each group having at least 22 participants. Considering an exclusion rate of 20%, the target number of participants in each group was set at 28. Enrollment for this study was capped when the fewest participants, the tGeo group, reached 28. The normality of the data was evaluated using the Shapiro-Wilks test. Fisher's exact test was used to evaluate the significance of differences in proportions between groups. The Kruskal-Wallis and post-hoc Dunn's tests assessed differences in CP values among the groups. Wilcoxon matched-pairs signed-rank test was used to evaluate before-and-after results. Statistical analyses were conducted using IBM SPSS version 25.0 (IBM Corp.) and Prism 8.0 (GraphPad Software). Data were presented as medians with interquartile ranges (IQRs) due to unequal distribution. Statistical significance was set at $P<0.05$.

RESULTS

Distribution and demographics of patients and follow-up results

A total of 104 patients were enrolled and divided into three groups based on the positional nystagmus type induced by the supine head roll test: pGeo (n=34), pApo (n=40), and tGeo (n=27) (Fig. 1, Table 1). Three patients were excluded due to a history of canal-wall-down mastoidectomy or a prior diagnosis of acute vestibular neuritis (Fig. 1). No significant differences were observed among groups regarding age, sex, or the time interval from symptom onset to diagnosis. However, disease duration (from dizziness onset to nystagmus resolution) significantly differed among the groups ($P=0.005$) (Table 1). Follow-up until nystagmus disappearance was completed in 23 patients (follow-up loss, 32.4%) in the pGeo group, 29 patients (follow-up loss, 27.5%) in the pApo group, and 24 patients (follow-up loss, 14.8%) in the tGeo group. The pGeo group demonstrated the longest disease duration (23.0 days; IQR, 12.0–53.0) compared to the pApo group (8.0 days; IQR, 2.5–29.5; Dunn's multiple comparisons test: $P=0.010$) and the tGeo group (9.0 days; IQR, 3.0–38.0; Dunn's multiple comparisons test: $P=0.016$) (Table 1).

Analysis of vHIT and bithermal caloric test

All enrolled patients had normal vHIT gain on the day of enrollment. However, caloric CP values were significantly higher in the pGeo group (26.0%, IQR=24.3%) compared to the pApo group (14.5%, IQR=18.9%; $P=0.002$) and the tGeo group (7.0%, IQR=13.6%; $P<0.001$) (Fig. 2A). The proportion of patients with initially abnormal CP was significantly greater in the pGeo group (21 of 34 patients, 61.8%) than in the pApo group (9 of 40 patients, 22.5%; $P=0.001$) and the tGeo group (3 of 27 patients, 11.1%; $P<0.001$). Within the pGeo group, abnormal CP was present on the affected side in 52.9% of patients, on the contralateral side in 8.8%, and was normal in 38.2% (Fig. 2B).

In the pGeo group, follow-up caloric testing after positional nystagmus resolution was performed in 11 of the 18 patients initially showing abnormal CP on the affected side. Among these, eight patients (72.7%) exhibited reduced CP at follow-up, with four patients (36.4%) returning to a normal CP range (<25%). In contrast, three patients (27.3%) showed increased CP at follow-up. However, no statistically significant difference was observed between initial and follow-up caloric tests in these 11 patients, mainly due to the three patients without a reduction in follow-up CP values ($P=0.206$) (Fig. 3). In the pApo and tGeo groups, follow-up caloric testing was conducted in three out of nine patients (33.3%) and two out of three patients (66.7%), respectively. Normalization of CP values at follow-up occurred

Table 1. Demographics, disease duration, and vHIT results of the enrolled patients

Variable	pGeo DCPN (n=34)	pApo DCPN (n=40)	tGeo DCPN (n=27)	*P*-value
Age (yr)	57.5 (51.2–70.0)	64.5 (54.2–75.7)	57.0 (49.0–64.0)	0.094
Male (%)	23.5	37.5	11.1	0.050
Diagnostic interval (day)	4.5 (1.25–15.5)	5.0 (0.0–14.0)	2.0 (0.7–13.0)	0.655
Disease duration (day)	23.0 (12.0–53.0)	8.0 (2.5–29.5)	9.0 (3.0–38.0)	0.005*
vHIT gain of affected LSCC	1.00 (0.93–1.04)	0.96 (0.84–1.01)	0.96 (0.91–1.04)	0.068
vHIT gain of contralateral LSCC	1.01 (0.95–1.08)	0.98 (0.72–1.04)	1.00 (0.92–1.03)	0.392

Values are presented as median (interquartile range). Diagnostic intervals the time from a patient's first symptoms to their diagnosis, disease duration: the period from the development of dizziness symptoms to the disappearance of nystagmus (data from 22, 29, and 24 patients who completed follow-up in the pGeo, pGeo, and tGeo DCPN groups, respectively).

vHIT, video head impulse test; pGeo, persistent geotropic; DCPN, direction-changing positional nystagmus; pApo, persistent apogeotropic; tGeo, transient geotropic; LSCC, lateral semicircular canal.

*$P<0.05$.

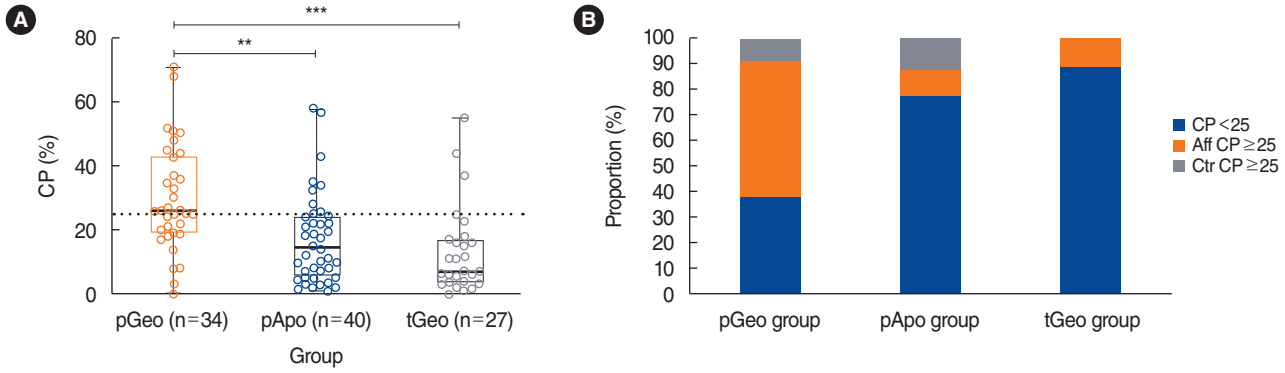

Fig. 2. Bithermal caloric test results of enrolled patients. (A) Box-and-whisker plot illustrating the distribution of canal paresis (CP), analyzed using the Kruskal-Wallis test with post-hoc Dunn's test. The median is indicated by the bar, and the interquartile range by the box. The dotted line denotes the normal CP cutoff value (<25%). (B) Proportion of patients with normal CP (blue bar), ipsilateral CP (orange bar), and contralateral CP (gray bar). pGeo group, persistent geotropic direction-changing positional nystagmus group; pApo group, persistent apogeotropic direction-changing positional nystagmus group; tGeo group, transient geotropic direction-changing positional nystagmus group; Aff, affected/ipsilateral; Ctr, control/contralateral. $^{**}P<0.01$, $^{***}P<0.001$.

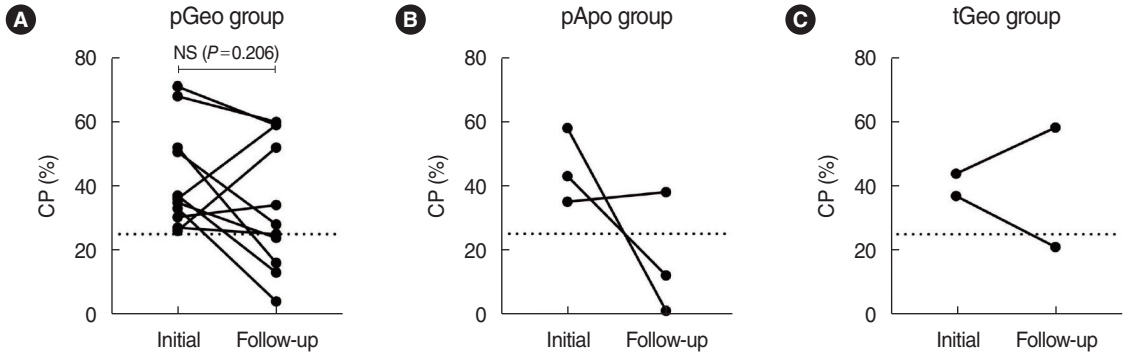

Fig. 3. Changes in canal paresis (CP) after follow-up. In the pGeo group, 11 patients initially showing abnormal CP on the affected side underwent a follow-up caloric test after positional nystagmus resolution. Some patients demonstrated normalized CP, although differences between initial and follow-up tests were not statistically significant. In the pApo and tGeo groups, only three and two patients with abnormal initial CP underwent follow-up caloric testing, respectively. PGeo group, persistent geotropic direction-changing positional nystagmus group; pApo group, persistent apogeotropic direction-changing positional nystagmus group; tGeo group, transient geotropic direction-changing positional nystagmus group; NS, not significant.

in two of the three pApo patients and one of the two tGeo patients.

DISCUSSION

This study revealed a higher incidence of CP and a longer disease duration in patients with pGeo DCPN than in those with other types of LSCC BPPV, despite all patients showing normal vHIT gain. Notably, CP predominantly occurred on the affected side (85.6% of abnormal CP cases), suggesting a potential link to the pathophysiology of pGeo DCPN. These observations imply distinct pathophysiological mechanisms between pGeo DCPN and pApo DCPN, as patients with pApo DCPN exhibited significantly lower CP values, fewer cases of abnormal CP, and shorter disease durations. Interestingly, most patients showed reduced CP, with some returning to normal levels in follow-up caloric tests conducted after positional nystagmus resolution. Considering clinical course and CP changes, pGeo may result from transient alterations in the cupula itself, whereas pApo might be related to otolith debris rather than cupular changes.

Few studies have reported caloric test outcomes specifically for pGeo DCPN of LSCC BPPV. Tang et al. [3] recently found CP in 20% of pGeo DCPN cases and none in pApo DCPN. Another study by Ichijo [22] indicated that 21% of pApo DCPN patients exhibited contralateral CP, whereas 21% of light cupula cases showed ipsilateral CP. The timing of the caloric test largely accounts for these differences: unlike our study, which performed caloric testing during active positional nystagmus, previous studies conducted caloric tests after nystagmus had subsided. Our findings confirmed a higher occurrence of abnormal CP in pGeo DCPN compared to pApo DCPN. Considering CP recovery at follow-up, 27.3% of patients in our study retained unilateral CP, a finding likely comparable to previous studies [3,22].

Bergenius and Tomanovic [11] and Tomanovic and Bergenius [35] reported abnormal CP in 13 of 20 pGeo DCPN patients. However, their study observed no clear correlation between decreased caloric response and the theoretical affected side determined by positional nystagmus direction in the supine position. Only three patients exhibited concordance between caloric weakness and the affected side identified via supine head-roll tests (geotropic nystagmus slow-phase velocity difference >10°/sec). These findings might not accurately reflect caloric responses influenced by gravitationally induced cupular deflection due to changes in cupular density. Furthermore, 40% of their patients had migraines, a condition commonly associated with positional nystagmus, possibly explaining the weak correlation between caloric test outcomes and nystagmus direction [36]. In contrast, our study excluded patients with migraines, demonstrating a stronger correlation between the affected side in pGeo DCPN and caloric weakness. CP directly reflects LSCC functional imbalance [16], and the caloric test fundamentally depends on cupular deflection induced by thermal changes in endolymph within the LSCC. Consequently, caloric-induced nystagmus velocity correlates with the extent of cupular deflection [37]. In pGeo DCPN patients, CP was primarily ipsilesional. A plausible hypothesis suggests cupular buoyancy counteracts thermal buoyancy from warm irrigation. Due to the ampulla's slight medial orientation relative to the gravity vector (earth-vertical axis), as evidenced by bow/lean tests, the light cupula deflects against gravity. This hypothesis could explain the caloric response normalization following pGeo DCPN resolution. Nevertheless, given persistent CP in many pGeo DCPN cases at follow-up, additional mechanisms might be involved. For example, underlying CP may predispose individuals to pGeo DCPN, or pGeo DCPN itself could induce lasting CP through sensory epithelium pathology or cupular degeneration. Clarifying these hypotheses requires further research involving large-scale prospective cohort studies and animal experiments.

Furthermore, alterations in endolymph properties consistent with the "heavier endolymph theory" might also contribute to CP alongside cupula buoyancy [25,38]. These theoretical alterations involve changes in the thermal expansion coefficient of the endolymph. Although direct evidence identifying specific components leaking into the endolymph is lacking, changes in thermal expansion could impact convection efficiency, potentially causing variations in caloric response. Additionally, altered endolymph viscosity might influence vHIT gain, given the pendulum model of the semicircular canal [37]. However, differences in vHIT gain between ears or groups were not observed in this study, suggesting that cupular buoyancy is a more plausible mechanism for CP in pGeo DCPN compared to changes in thermal expansion. Detailed profiles of factors increasing endolymph viscosity must be identified to further validate this hypothesis through modeling. Central vestibulopathies, such as vestibular migraine, can also cause pGeo DCPN [39]. Nonetheless, central vestibular contributions were excluded in this study based on the participant exclusion criteria.

Unlike caloric testing, vHIT results were normal and did not vary among groups. The very high angular acceleration (1,000–4,000°/sec^2) during vHIT may overpower cupular buoyancy effects, corresponding to lower angular acceleration [40,41]. Conversely, cupular buoyancy resembles low angular acceleration stimulation conditions, as even slight changes in cupula density (~10^{-4} g/cm^3) are sufficient to produce gravity-sensitive responses [42]. An alternative hypothesis relates to different responses of vestibular hair cell types to varying stimulation frequencies. Type I vestibular hair cells respond primarily to abrupt, high-frequency stimulation, whereas type II cells respond predominantly to low-frequency stimuli [43,44]. This difference could account for the discrepancy between vHIT (>2 Hz) and caloric testing (<0.01 Hz), similar to the "cell type theory" proposed to explain discrepancies observed in Meniere disease [45-47]. Thus, a lighter-density cupula might predominantly stimulate type II hair cells rather than type I hair cells.

The infrequent occurrence of abnormal CP in the pApo and tGeo DCPN groups compared to the pGeo DCPN group implies the following inference: in pGeo DCPN, the cupula deflects against gravity due to its lower specific gravity compared to endolymph. Given the significantly higher CP in pGeo DCPN, the cupular deflection appears more pronounced in this group. Reports on nystagmus velocity in pGeo DCPN remain limited, though eye velocity in pApo DCPN has been noted to be slower compared to tGeo DCPN [48]. Cupulolithiasis is generally accepted as the main mechanism of pApo DCPN [49]. The different locations of the cupula where otoliths attach, as well as varying amounts of otoliths, can result in variable CP values for pApo DCPN, with the frequency of abnormal CP values being higher than that of the canal and lower than that of pGeo DCPN. Given the shorter disease duration and better response to repositioning maneuvers in pApo DCPN, cupulolithiasis rather than intrinsic cupular changes is likely its primary pathophysiology. These hypotheses require direct evidence, given the absence of definitive animal models or cupular pathology data for precise modeling.

This study had several limitations. First, considerable loss of follow-up occurred despite clinicians' recommendations for regular outpatient visits and serial caloric tests in patients with initially abnormal CP. Second, variations in follow-up durations until nystagmus disappearance among institutions might have introduced discrepancies in BPPV resolution periods. However, the overall trends in resolution were consistent across institutions. Additionally, CP recovery was observed across pApo, tGeo, and pGeo groups, meaning that CP reversal cannot definitively be considered an exclusive characteristic of pGeo DCPN. Finally, caution is warranted when interpreting CP changes. Although caloric test reliability is high (coefficients >0.90 [50]); variability up to 15%–20% in CP has been documented [51].

Although conclusively determining the exact mechanism of

pGeo DCPN remains challenging, our study suggests that its mechanism may not simply mirror the opposite of pApo DCPN (light debris theory), which involves heavier otoconia attached to the cupula. This inference is supported by significantly higher CP values, poorer repositioning maneuver responses, and longer disease durations (median, 23.0 days) observed in this study, compared to durations under one week reported for pApo and tGeo DCPN by Shim et al. [29,52]. In conclusion, the caloric test findings across different LSCC BPPV types in this study suggest distinct pathophysiological mechanisms. Cupular deflection driven by buoyancy in pGeo DCPN could lead to CP while preserving normal vHIT results.

CONFLICT OF INTEREST

Sung Huhn Kim is an editorial board member of the journal but was not involved in the peer reviewer selection, evaluation, or decision process of this article. No other potential conflicts of interest relevant to this article were reported.

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Conceptualization: SHB, SHK (Sung Huhn Kim). Methodology: SHK (Sung Huhn Kim). Software: SHB. Visualization: SHB, SHK (Sung Huhn Kim). Formal analysis: SHB. Investigation: SHB. Resources: SHK (Sang Hyun Kwak). Data curation: SHB, SHK, JML, JHH, DBS, SHK. Supervision: SHK (Sang Hyun Kwak), SHK (Sung Huhn Kim). Project administration: SHB. Funding acquisition: SHK (Sung Huhn Kim). Writing–original draft: SHB, SHK (Sang Hyun Kwak). Writing–review & editing: SHB, SHK (Sang Hyun Kwak), SHK (Sung Huhn Kim). All authors read and agreed to the published version of the manuscript.

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