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ORIGINAL COMMUNICATION

# Characteristics of positional nystagmus in patients with horizontal canal canalolithiasis or cupulopathy

Hui Wang[1] · Qingxiu Yao[1] · Zhuangzhuang Li[1] · Dongzhen Yu[1] · Haibo Shi[1]

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## Abstract

**Objectives** Positional nystagmus can be related to various kinds of disorders. The current study aims to compare the direction-changing horizontal positional nystagmus (DCPN) characteristics in horizontal canal canalolithiasis (HC-canalolithiasis), heavy cupula of the horizontal canal (HC-Hcu), and light cupula of the horizontal canal (HC-Lcu), especially the temporal patterns of positional nystagmus in three disorders.

**Methods** 52 patients (22 males, 30 females; mean age, 49.6 years) presenting with geotropic or apogeotropic DCPN were enrolled, and they were divided into HC-canalolithiasis, HC-Hcu, or HC-Lcu groups according their nystagmus characteristics. We compared their latency, time constant, peak slow-phase velocity (SPV), time to reach peak SPV intensity ($T_{peak}$), and time to decay to half-peak intensity ($T_{1/2peak}$).

**Results** The time to reach peak SPV did not differ significantly between the HC-Hcu (23.1 ± 8.6 s) and HC-Lcu (24.4 ± 9.9 s) groups ($p = 0.733$), but was significantly longer than that of the HC-canalolithiasis group (5.4 ± 3.5 s; $p \leq 0.001$). The peak intensity did not differ among the canalolithiasis (36.4 ± 20.6°/s), HC-Hcu (30.1 ± 23.6°/s), and HC-Lcu (21.4 ± 12.7°/s) groups ($p = 0.133$). The onset latency also had no statistical difference among three groups ($p = 0.200$). The nystagmus patterns of HC-Lcu and HC-Hcu groups were similar, including latency, peak SPV intensity, $T_{peak}$, $T_{1/2peak}$, and SPV in 20 s, 40 s, 60 s, 80 s.

**Conclusions** The nystagmus characteristics of HC-Hcu and HC-Lcu are similar, except for the fact that movement was in opposite directions, suggesting that HC-Hcu and HC-Lcu may result from a similar pathophysiological mechanism (cupulopathy) differing from that underlying canalolithiasis.

**Keywords** Horizontal canal · Canalolithiasis · Heavy cupula · Light cupula

## Introduction

Benign paroxysmal positional vertigo (BPPV), the most common peripheral vestibular disorder, is characterized by recurrent attacks of brief positional vertigo (dizziness) and nystagmus elicited by a change in head position relative to gravity [1, 2]. The horizontal canal (HC) is the second most affected canal and HC-BPPV accounts for 5–30% of all BPPV cases [3, 4]. In patients with HC-BPPV, direction-changing positional nystagmus (DCPN) is typically observed upon turning the head to either side in the supine position and could be classified as geotropic or apogeotropic according to the direction of the DCPN [2]. In most patients, geotropic DCPN disappears within 30 s, which can be explained by a model of canalolithiasis, according to which otoconia dislodged from the otolith macula beds are trapped in a semicircular canal and move in response to changes in head position and eventually trigger vertigo attacks [5]. In contrast, apogeotropic nystagmus often lasts longer than 1 min and is thought to be the result of cupulolithiasis (heavy cupula) of the horizontal canal (HC-Hcu), which is caused by dislodged otoconia attached to the cupula, renders it sensitive to gravity [2, 6, 7]. Probably, canalolithiasis of the horizontal canal

Hui Wang, Qingxiu Yao and Zhuangzhuang Li contributed equally to this manuscript.

✉ Dongzhen Yu
drdzyu@126.com

✉ Haibo Shi
hbshi@sjtu.edu.cn

[1] Department of Otolaryngology, Shanghai Jiao Tong University Affiliated Sixth People's Hospital. Shanghai Key Laboratory of Sleep Disordered Breathing, Shanghai 200233, China

may also cause apogeotropic horizontal positional nystagmus. This would be the case when otoconia are located in the anterior part of the horizontal canal (close to the cupula), in these patients, the nystagmus is transient and transformation from apogeotropic to geotropic positional nystagmus can be observed during diagnostic positional maneuvers [2, 6].

Recently, patients with persistent geotropic DCPN without latency or fatigability have been reported [7-9]. These cases are similar to the HC-Hcu, although the nystagmus was in opposite direction. Imai et al. [10]reported that the relationship between slow-phase velocity (SPV) and the angle of head rotation in patients with persistent geotropic nystagmus was linearly symmetrical to the patients with persistent apogeotropic nystagmus with respect to the neutral head position (position where the nystagmus disappeared), suggesting that persistent geotropic and apogeotropic nystagmus share a similar pathophysiology. Previous studies have shown that persistent apogeotropic nystagmus was caused by HC-Hcu, whereas persistent geotropic nystagmus was thought to be due to a light cupula of the HC (HC-Lcu) [9, 10].

Previous studies in patients with HC-Lcu found nystagmus duration longer than 1 min without latency or fatigability [7-9]. In a previous study, we extended on these findings by assessing changes in nystagmus intensity over time in nine patients [11]. The nystagmus had the crescendo-decrescendo temporal intensity pattern similar to typical BPPV, and also had latency and fatigability. To further characterize persistent geotropic nystagmus, we performed the roll test in additional 12 subjects with HC-Lcu and compared the nystagmus with that elicited by HC-canalolithiasis and HC-Hcu. We characterized the nystagmus in 52 patients with DCPN and compared the temporal patterns of positional nystagmus in HC canalolithiasis, HC-Hcu, and HC-Lcu to quantitatively analyze the dynamic changes of positional nystagmus in three groups.

## Materials and methods

This study was carried out in accordance with the recommendations of Ethics Committee of Shanghai Jiao Tong University affiliated sixth People's Hospital (2016-105-(1)). The protocol was approved by the Ethics Committee of Shanghai Jiao Tong University affiliated sixth People's Hospital. All subjects gave written informed consent in accordance with the Declaration of Helsinki.

The study included 52 patients (22 males and 30 females; mean age, 49.6 years; range, 18–69 years) diagnosed with HC-canalolithiasis, HC-Hcu, or HC-Lcu in the Department of Otolaryngology at Shanghai Jiao Tong University Affiliated Sixth People's Hospital, between March 2017 and October 2018. A detailed history was obtained for all patients. The average disease duration was calculated as the time from the onset of vertigo to the clinic visit. The Dix-Hallpike maneuver and supine roll test were performed on each patient. HC canalolithiasis and HC-Hcu were diagnosed according to the criteria formulated by the Bárány Society [2], whereas the HC-Lcu diagnosis was based on the presence of persistent (lasting > 1 min) geotropic nystagmus during the roll test [12]. According to Ewald's law, the affected side was that showing more vigorous nystagmus in patients with HC canalolithiasis and HC-Lcu, whereas the affected side was opposite in patients with HC-Hcu.

The nystagmus was recorded in four head positions. Dix-Hallpike maneuver was performed first, briefly, the head is first turned 45º to left, the patient is then rapidly brought into a left head-hanging position with the head 20–30º below the bed. The patient is next returned to the upright position with the head facing forward, then rotated 45º to the right and repeated the procedure for the right ear. Bilateral supine head roll test was performed then, patients lay supine with the head 30º up from the earth horizontal plane, the patient's head was then turned by about 90º to left and right sides successively. The nystagmus was recorded using an infrared illuminated, vision denied VNG system (VertiGoggles-M, ZEHNIT Medical Technology, Shanghai, China). A 640 × 480 pixel@60FPS infrared camera is placed conjugate to the patient's iris and produces a digital recording that is transmitted to a PC for real-time monitoring and analysis of horizontal and vertical eye movements based on pupil tracking. In addition to test results, calibration values are saved and are available for review. All recordings are available for review and analysis in the review/analysis mode of the software, during review and analysis, horizontal and vertical eye movement traces are displayed, slow-phase velocity of individual nystagmus beat is measured by the software.

Given the characteristics of DCPN, only data from the roll test were assessed. The recordings began when the patients' head reached target positions and were termed as $T_0$, patients' head maintained at target positions until positional nystagmus disappeared (HC-canalolithiasis group) or for longer than 60 s (HC-Hcu and HC-Lcu groups). The onset of nystagmus was measured from the first clearly visible SPV beat, which was termed as $T_{onset}$. The latency time was defined as the time from $T_0$ to $T_{onset}$. The SPV toward left was defined as positive, and toward right was defined as negative. The peak intensity was determined by averaging the SPV of the maximum three beats, and the peak SPV of the affected and unaffected sides were recorded. The SPV was measured at 20, 40, 60, and 80 s in the HC-Hcu and HC-Lcu groups using the same method as peak SPV. One patient each in HC-Hcu and HC-Lcu groups was recorded shorter than 80 s, so they were excluded for calculating 80 s SPV. The time constant (TC) was defined as the time for the

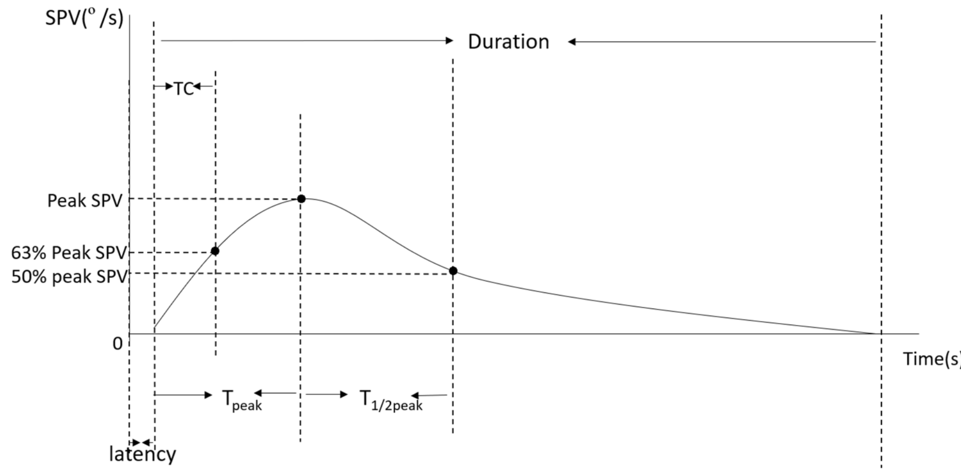


**Fig. 1** Graphic illustrations of different time parameters. TC: time constant, the time it takes for the response to reach 63% of the peak slow-phase velocity, $T_{peak}$: the time it takes for the response to reach peak intensity; $T_{1/2peak}$: the time it takes for the response decreases to half-peak intensity SPV: slow-phase velocity

**Table 1** Demographic and nystagmus characteristics of three types of patients

| Diagnosis` | Sex (M:F) | Age(y) | Peak SPV (°/s) Vigorous side | Peak SPV (°/s) Less side | Latency(s) | TC(s) | $T_{peak}$(s) | $T_{1/2peak}$(s) |
|---|---|---|---|---|---|---|---|---|
| HC-Ca | 9:14 | 52.8 ± 11.4 | 36.4 ± 20.6 | 10.8 ± 10.4 | 1.0 ± 2.6 | 2.7 ± 2.4 | 5.4 ± 3.5 | 7.8 ± 4.3 |
| HC-Hcu | 11:6 | 44.5 ± 15.8 | 30.1 ± 23.6 | 8.2 ± 7.4 | 1.9 ± 2.7 | 14.6 ± 5.8 | 23.1 ± 8.6 | 32.5 ± 9.9 |
| HC-Lcu | 2:10 | 50.5 ± 14.2 | 21.4 ± 12.7 | 9.9 ± 4.4 | 2.8 ± 3.5 | 11.9 ± 5.6 | 24.4 ± 9.9 | 27.1 ± 13.4 |

*HC-Ca* canalolithiasis of horizontal canal, *HC-Hcu* cupulolithiasis of heavy cupula, *HC-Lcu* cupulolithiasis of light cupula, *M* male, *F* female

response to reach approximately 63% of the peak SPV. The time for SPV to reach peak intensity ($T_{peak}$), and the time to decay to half-peak ($T_{1/2peak}$) were calculated, schematic diagram was shown in Fig. 1.

## Statistical analysis

The descriptive data are expressed as mean ± standard deviation. The sex rate among groups were compared by chi-square test, the ages, disease duration, latency were using one-way ANOVA. The peak SPV of the affected and unaffected ears were compared using Wilcoxon's paired test. SPV, TC, $T_{peak}$, $T_{1/2peak}$ among groups were compared using the Kruskal–Wallis test and post hoc tests. SPV at 20, 40, 60, and 80 s between the HC-Hcu and HC-Lcu groups were compared using the Mann–Whitney U test (median, interquartile ranges and 95% confidence intervals were exhibited). All statistical analyses were conducted using SPSS version 22.0 (SPSS/PC, Chicago, IL, USA).

# Results

The study included 52 patients (22 males and 30 females; mean age, 49.6 years; age range, 18–69 years) diagnosed with HC canalolithiasis, HC-Hcu, or HC-Lcu. The medical histories obtained for each patient revealed one case suffered from BPPV long time ago, one case occurred vertigo 40 years ago, one case of sudden sensorineural hearing loss, and one case tinnitus and deafness caused by trauma. None of the remaining 48 patient experienced symptoms related to vertigo or otorhinolaryngologic disease. The average disease duration in the HC-canalolithiasis, HC-Hcu, and HC-Lcu groups were 8.5 ± 5.9, 14.7 ± 12.5, and 8.6 ± 8.5 days, respectively. There were no difference in ages and disease duration among three groups ($p = 0.188$, $p = 0.116$, respectively), however, the sex rate were not well matched ($p = 0.033$). Table 1 lists the patient demographic and nystagmus characteristics of each group.

Characteristics of HC-canalolithiasis group.

Of the 52 patients, 23 (14 females and 9 males; average age, 52.8 ± 11.4 years) were diagnosed with HC-canalolithiasis. Geotropic DCPN was observed immediately after lying with the affected ear down in 19 patients, and the remaining four patients had latencies of several seconds (average latency, 1.0 ± 2.6 s). The SPV profile of a canalolithiasis was presented in Fig. 2. All patients had a crescendo-decrescendo pattern of intensity, the TC was 2.7 ± 2.4 s, the duration of positional nystagmus was 25.0 ± 6.3 s, and the peak intensity was 36.4 ± 20.6°/s at 5.4 ± 3.5 s and decayed to half-peak at 7.8 ± 4.3 s. Rolling the patient onto the unaffected side, a less intense geotropic nystagmus (peak SPV 10.8 ± 10.4°/s) was observed ( Fig. 3).

Characteristics of HC-Hcu group.

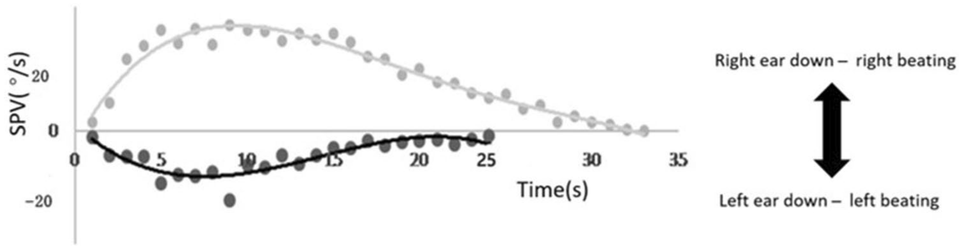


**Fig. 2** SPV profile of positional nystagmus in each direction in a subject with right HC-canalolithiasis. With the affected and unaffected ears down, geotropic nystagmus was recorded, note the nystagmus pattern of crescendo-decrescendo with greater SPV to the right, the SPV returned to baseline by 33 s

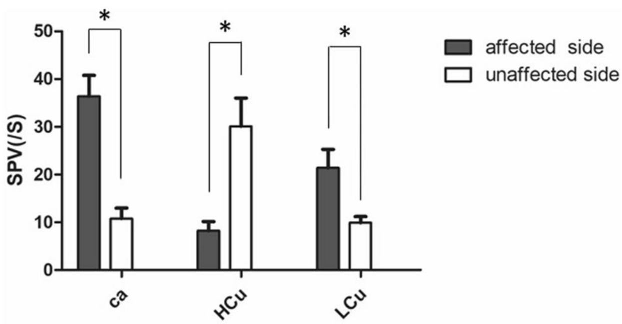


**Fig. 3** The comparisons of peak SPV in affected and unaffected sides of three groups

The HC-Hcu group included 17 patients (6 females and 11 males; mean age, 44.5 ± 15.8 years). According to Ewald's law, the side with less vigorous nystagmus was considered to be the affected side. Upon lying with the unaffected ear down, apogeotropic DCPN was observed almost immediately after onset (average latency, 1.9 ± 2.7 s, range, 0–10 s), the recorded time of positional nystagmus was 94.0 ± 27.2 s. These patients also had the crescendo-decrescendo pattern of intensity similar to typical BPPV. The SPV profile of a HC-Hcu patient was presented in Fig. 4. The TC was 14.6 ± 5.8 s, the peak intensity was 30.1 ± 23.6°/s at 23.1 ± 8.6 s, and $T_{1/2peak}$ was 32.5 ± 9.9 s. Rolling the patient onto the affected side produced a less intense apogeotropic nystagmus (peak SPV 8.2 ± 7.4°/s; Fig. 3).

Characteristics of HC-Lcu group.

In total, 12 subjects (10 females and 2 males; mean age, 50.5 ± 14.2 years) were diagnosed with HC-Lcu. Upon lying with the affected ear down, a horizontal geotropic nystagmus was observed with the average onset latency of 2.8 ± 3.5 s (range, 0–10 s), and the recorded time of positional nystagmus was 86.5 ± 23.8 s. The positional nystagmus showed a pattern of slow build-up and gradual decay, maintained at slow SPV, similar to that observed in the HC-Hcu group. The SPV profile of a HC-Lcu patient was exhibited in Fig. 5. With the affected ear down, the nystagmus peaked at 24.4 ± 9.9 s with a peak intensity of 21.4 ± 12.7°/s. The TC was 11.9 ± 5.6 s, $T_{1/2peak}$ was 27.1 ± 13.4 s. Rolling the patient onto the unaffected side, a less intense geotropic nystagmus (peak SPV 9.9 ± 4.4°/s; Fig. 3) was observed.

The duration of nystagmus was markedly shorter, the $T_{peak}$ and $T_{1/2peak}$ occurred earlier in patients with canalolithiasis compared with cupulopathy groups (HC-Hcu and HC-Lcu). However, the peak intensity did not differ significantly among groups ($p = 0.133$). The onset latency also had no difference among three groups ($p = 0.200$). The SPV pattern of HC-Lcu and HC-Hcu are similar, including SPV at 20 s, 40 s, 60 s, and 80 s (Table 2), peak intensity, TC ($P = 0.514$), $T_{peak}$ ($p = 0.733$), $T_{1/2peak}$ ($p = 0.336$).

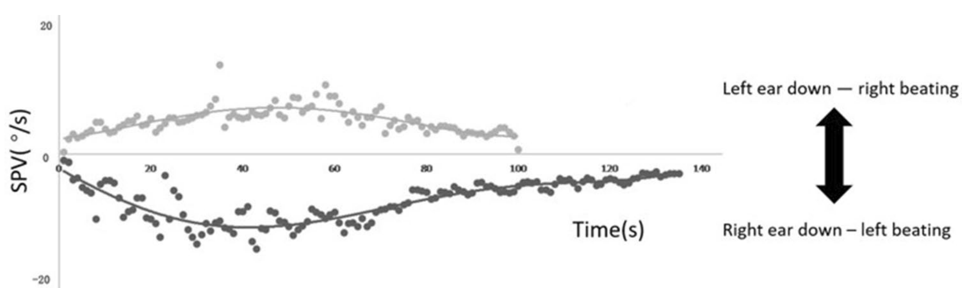


**Fig. 4** SPV profile of positional nystagmus in each direction in a subject with left HC-Hcu. With the affected and unaffected ears down, apogeotropic nystagmus was recorded, note the slow build-up and gradual decay with greater SPV to the left, the $T_{peak}$ was at 40 s, the $T_{1/2peak}$ was 30 s, and the TC was 13 s

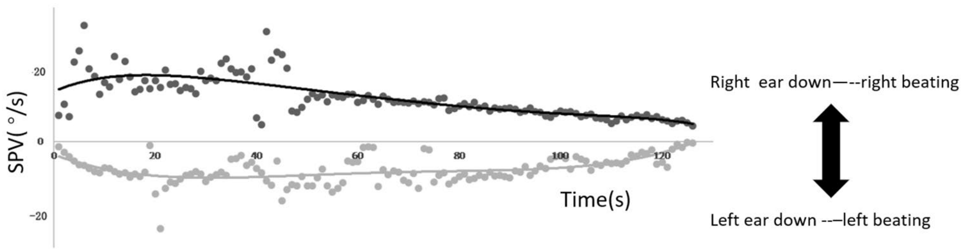


**Fig. 5** SPV profile of positional nystagmus in each direction in a subject with right HC-LCu. Geotropic nystagmus was recorded with the affected and unaffected ears down, note the nystagmus pattern of build-up and gradual decay with greater SPV to the right, the $T_{peak}$ was at around 20 s, and the $T_{1/2peak}$ was 40 s, and the TC was 8 s

**Table 2** The comparisons of SPV at 20, 40, 60, 80 s in vigorous sides between Hcu and Lcu

| | HC-Hcu ($n=17$) | | HC-Lcu($n=12$) | | $P$* |
|---|---|---|---|---|---|
| | Median (25–75%) | 95%CI | Median (25–75%) | 95% CI | |
| 20 s | 10.8 (8.3–29.9) | 9.6–33.2 | 8.2 (5.1–19.4) | 6.3–18.1 | 0.263 |
| 40 s | 15.2 (7.3–29.5) | 12.2–34.9 | 10.5 (5.4–16.8) | 6.6–17.6 | 0.117 |
| 60 s | 9.8 (5.5–23.6) | 7.8–25.4 | 7.7 (3.3–12.8) | 5.2–11.0 | 0.283 |
| 80 s | 5.7 (3.5–10.55) | 3.6–18.6 | 5.0 (1.9–7.5) | 2.8–6.9 | 0.272 |

*CI* confidence interval

## Discussion

We compared nystagmus characteristics in patients with HC-canalolithiasis, HC-Hcu, and HC-Lcu, and found that the positional nystagmus in three disorders all present a crescendo–decrescendo pattern. The nystagmus duration was markedly shorter and the $T_{peak}$ and $T_{1/2peak}$ occurred earlier in the HC-canalolithiasis group than HC-Hcu and HC-Lcu groups. However, the peak intensity did not differ significantly among groups. The nystagmus onset latency also had no statistical difference among groups. The nystagmus pattern, including the peak intensity, duration, TC, $T_{peak}$, $T_{1/2peak}$, and SPV at 20, 40, 60, and 80 s, was similar in patients with HC-Lcu and HC-Hcu, suggesting that the conditions may result from similar underlying mechanism, which differ from that involved in canalolithiasis.

The heavy cupula theory is widely accepted as an explanation of HC-Hcu; however, the mechanism underlying HC-Lcu remains controversial. One hypothesis is the heavy endolymph theory, which states that the specific gravity of the endolymph may increase due to an acute insult to the inner ear, such as labyrinthine hemorrhage, inner ear hypoperfusion, or inflammation, leaving the specific gravity of the cupula lower than surrounding endolymph [8] [9]. However, if this theory is correct, it is difficult to explain why the HC is commonly affected, but involvement of the posterior and anterior canals is rare. Moreover, the heavy endolymph theory suggests that the angle of the neutral position should be approximately 20º, however, Ichijo et al. [13]reported that the neutral position varied markedly among subjects (HC-Hcu range, 5–89º, and HC-Lcu range, 5–85º), the authors argued that their findings supported the light debris theory, according to which light particles attach to the HC cupula and thereby make the cupula light. We found that the nystagmus patterns of HC-Lcu and HC-Hcu were similar in TC, latency, peak SPV intensity, $T_{peak}$, $T_{1/2peak}$, or SPV at 20, 40, 60, and 80 s. Thus, the light debris theory is a more plausible explanation for these findings. Most patients in HC-Lcu were prescribed with vestibular suppressants for the symptomatic relief, vertigo and positional nystagmus resolved within 1 week in most patients [9], Several studies indicated that repositioning maneuvers were not effective in HC-Lcu [8, 14], which was different from HC-Hcu, so the exact mechanism of HC-Lcu is yet to be elucidated.

The dynamics of build-up and decay of positional nystagmus associated with cupulopathy can be explained by the dynamics of the horizontal vestibulo-ocular reflex (VOR). The stimulus in patients with cupulopathy is similar to a constant angular acceleration that results in deflection of the cupula in an exponentially increasing manner first and then maintained at a constant level. However, the VOR response does not parallel the deflection of the cupula, which builds up rapidly before deflection of the cupula reaches a constant level and then decays slowly during the rest period [15, 16], this mechanism explains the rapid build-up in patients with cupulopathy. In normal human subjects, the average TC of the horizontal VOR is about 12 s, which is consistent with the rapid build-up of the static positional nystagmus observed in the present study. The gradual decay in SPV after reaching the peak response in patients with cupulopathy may be explained by central adaptation. Estimates of the central adaptation TC of the horizontal VOR in normal human subjects is in the range of 80–100 s [15-19], which is approximately consistent with the slow rate of decay observed in our study.

## Study limitations

Our study has several limitations. Our sample size may not be sufficient, and selection bias should be considered when interpreting our results. Furthermore, Because not all of the patients in HC-Hcu and HC-Lcu groups underwent brain magnetic resonance imaging, cases of central lesions may have been missed, but given the characteristics of nystagmus and the absence of neurological findings, such as ataxia, spontaneous downbeat nystagmus, gaze-evoked nystagmus, and saccadic hypermetria, the likelihood that DCPN was caused by central lesions is low.

## Conclusions

Our detailed comparison of the nystagmus characteristics in HC-canalolithiasis, HC-Hcu, and HC-Lcu revealed that nystagmus patterns were identical in patients with HC-Hcu and HC-Lcu, except for the fact that movement was in opposite directions. This finding suggests that HC-Hcu and HC-Lcu may result from a similar pathophysiological mechanism, which differs from that underlying canalolithiasis.

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### Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

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