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Vestibulo-ocular Reflex Gain Asymmetry in Unilateral Ménière's Disease: Insights from HIMP and SHIMP Tests and Correlations with Audio-vestibular Findings

Original Investigation

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Abstract

Objective: The purpose of this study was to calculate the vestibulo-ocular reflex (VOR) gain asymmetry ratios obtained from head impulse paradigm (HIMP) and suppression HIMP (SHIMP) tests in patients with unilateral definite Ménière's disease (MD) and to investigate their relationships with clinical, audiologic and vestibular parameters.

Methods: The study included 35 (18 female, 17 male) unilateral MD patients with a median age of 50 (24-65). All patients underwent pure tone audiometry, video-nystagmography, including caloric test, and video head impulse test. The VOR gain asymmetry indices were computed and the demographic, clinical and audio-vestibular variables were analyzed.

Results: Mean HIMP lateral canal VOR gain asymmetry ratio was -11.27±25.276 and mean SHIMP lateral canal VOR gain asymmetry was -15.63±23.993. While differences in caloric response asymmetry ratios were observed among hearing-loss severity groups (p=0.05), HIMP and SHIMP asymmetry ratios did not differ. VOR gain asymmetry ratios showed significant differences among dizziness handicap inventory groups, SHIMP saccade group and visually enhanced VOR saccade group (p<0.05). Significant correlations were found between SHIMP and caloric asymmetry ratios with air-conduction pure tone averages (r=-0.337, p=0.047 and r=-0.358, p=0.035), and between HIMP lateral canal asymmetry and hearing at 500 Hz (r=-0.362, p=0.032).

Conclusion: Our study confirmed that VOR gain asymmetry assessed by SHIMP and HIMP differs across hearing levels; and further that caloric response asymmetry is also correlated with audio-vestibular parameters in patients with unilateral MD.

Keywords: Ménière's disease, vertigo, caloric test, head-impulse, vestibulo-ocular reflex

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Introduction

Ménière's disease (MD) is an idiopathic inner ear disorder characterized by episodic vertigo, fluctuating sensorineural hearing loss, tinnitus, and aural fullness. The underlying pathophysiology is commonly attributed to endolymphatic hydrops. Most patients are initially

unilateral, but bilaterality can increase to 50% with disease progression (1). A careful history taken during and between attacks, clinical evaluation, and vestibular testing are crucial for diagnosis. The diagnostic criteria for the disease were defined by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) in 1995 and revised in 2015 (2).

Vestibular system functions are generally evaluated by testing the two most basic reflexes, the vestibulo-ocular reflex (VOR) and the vestibulo-spinal reflex (VSR). The major tests for VOR are the video head-impulse test (vHIT), the video-nystagmography (VNG) and the caloric test, and for VSR the vestibular-evoked myogenic potentials and the postugrography. Vestibular testing in MD often shows discrepancies between low-frequency and high-frequency results due to the differential sensitivities of the tests. The most significant finding in vestibular testing for MD is unilateral vestibular hypofunction detected on bi-thermal caloric testing (3). However, normal caloric test results can be obtained even during attacks in nearly half of the patients. There are two paradigms: the conventional head impulse paradigm (HIMP) and the suppression HIMP (SHIMP). vHIT quantifies high-frequency angular VOR and yields canal-specific gain and gain asymmetry with characterization of overt/covert catch-up saccades. The SHIMP test has emerged in recent years as an alternative to HIMP to overcome the difficulties in calculating VOR gain due to covert saccades (4).

During VNG, a low frequency test, suppression VOR is used to evaluate how well the cerebellum can suppress VOR when visual fixation is present. Impaired VOR suppression points to cerebellar dysfunction; recovery of suppression over time reflects central compensation after peripheral loss (5). Visually enhanced VOR (vVOR) probes visuo-vestibular integration, with a moving visual target during head motion, normal subjects show near-unity vVOR gain. Deviations inform the balance between visual and vestibular drive and may flag specific disorders (6).

However, data on VOR gain asymmetry in all semicircular canal planes in MD and how it correlates with demographic, clinical and audio-vestibular findings remain limited. The aim of this study was to investigate the HIMP and SHIMP VOR gain asymmetry and to evaluate correlations with clinical parameters in unilateral MD patients with canal paresis.

Methods

The study was approved by the Clinical Research Ethics Committee of the University of Health Sciences Türkiye, Bursa Yüksek İhtisas Training and Research Hospital (approval no: 2011-KAEK-25 2023/04-10, date: 19.04.2023). Informed consent was obtained from all the patients and subjects. The patients included in the nested cohort prospectively were those diagnosed with definite MD according to the guidelines of the Equilibrium Committee of the AAO-HNS (2).

The study was conducted with a nested cohort of a certain disease and without a control group as the study investigated the asymmetry between the affected ear and the self-control ear individually in each patient. Power analysis for two-tailed nonparametric testing with Cohen's d=0.50 (medium effect), α=0.05, power=0.80 features, the minimum sample size was calculated as 32 patients.

Patients

Individuals diagnosed with MD who presented to the ear, nose and throat outpatient clinics of a tertiary teaching hospital for routine follow-up between May 2023 and November 2023 were included in the study. Previous files of the patients were reviewed retrospectively, and demographics, disease-related clinical data, and audio-vestibular test results were noted. Pure-tone audiometry, VNG (including caloric testing), and vHIT (HIMP and SHIMP) results and tinnitus handicap inventory (THI) and dizziness handicap inventory (DHI) scores obtained at their visits in the inter-attack period were also recorded.

The inclusion criteria were:

1. age between 18 and 65 years

2. previous diagnosis of unilateral MD

3. had visited the clinic at earliest 1-month since the last attack

4. MD with unilateral canal paresis and normal ear examination

The exclusion criteria were:

1. external ear or middle ear pathology detected in otoscopy

2. type B and C tympanogram

3. conductive or mixed type hearing loss in pure-tone audiometry

4. pathological findings other than endolymphatic hydrops in magnetic resonance imaging

5. disconjugate eye movements

6. history of ototoxic or vestibulo-otoxic medication

7. central pathology

8. overlap syndrome, associating other vestibular disorders

Demographic characteristics such as age, sex, employment status, smoking, alcohol use, and comorbidity of the patients

were recorded. Anamnesis was obtained by questioning the onset time, course, and accompanying symptoms of hearing loss. In the physical examination, middle ear effusion, infection, and some neoplasms were evaluated, and a comprehensive evaluation of cranial nerves and cerebellar functions was done.

Audio-vestibular Tests

All audiometric evaluations were conducted in single-walled silent booths. Tests in the booth were performed using a clinical audiometer (Interacoustics AC40, Denmark). Pure tone air-conduction thresholds (ACT) in the range of 250-8000 Hz were obtained with supra-aural headphones (Telephonics TDH-50), while bone-conduction thresholds at 500-8000 Hz range were obtained with a bone vibrator (Radioear B71). The pure-tone average (PTA) was calculated as the mean threshold of 500, 1000, 2000, and 4000 Hz. Tympanometry was performed with a 226 Hz probe tone at 80 dB intensity. Acoustic reflexes were assessed at 500, 1000, 2000, and 4000 Hz using a 100 dB stimulus.

All participants underwent the VNG battery including bithermal caloric test and the vHIT at lateral, right anterior-left posterior and left anterior-right posterior canal planes. Vestibular tests were recorded with the impulse control system (ICS)-impulse version 4.0 (Otometrics A/S, Taastrup, Denmark) VNG and vHIT device and ICS AirCal (GN Otometrics, Taastrup, Denmark). Caloric tests were carried out with air stimulation at 50°C and 24°C for 60 seconds. Canal paresis and directional superiority were calculated using Jongkees' formula [(total right ear response - total left ear response/total right + left ear response) x 100] for canal paresis. For canal paresis, the difference in nystagmographic response between both sides was determined as at least 20%.

Calculation of VOR Gain and Caloric Response Asymmetry

The asymmetry ratios for VOR gains of lateral, posterior and anterior semi-circular canals were calculated with the formula below:

Asymmetry ratio (%) =2x (ipsilateral canal VOR gain - contralateral canal VOR gain)/(ipsilateral canal VOR gain + contralateral canal VOR gain) x100.

The asymmetry ratios for caloric responses were calculated with the formula below:

Asymmetry ratio (%) =2x (ipsilateral total caloric response - contralateral total caloric response)/(ipsilateral total caloric response + total contralateral caloric response) x100.

In this formula and in all audio-vestibular test results, ipsilateral refers to the affected ear (diseased ear).

Quality of Life Assessment

The Turkish version of the THI assesses the emotional, catastrophic, and functional effects of tinnitus (7). The survey consists of 25 questions: a "yes" answer scores 4 points, "sometimes," scores 2 points, "no," scores 0 points (8). The classification according to total scores are: "no or slight handicap (0-16)," "mild handicap (18-36)," "moderate handicap (38-56)," "severe handicap (58-76)," and "catastrophic handicap (78-100)."

The DHI defined by Jacobson and Newman (9) and whose reliability and validity have been studied in Turkish was applied (10). The DHI consists of 25 questions regarding the presence of dizziness to determine the physical, emotional, and functional effects of vestibular disorders during the performance of certain movements. Dizziness is scored as 4 if always present, 2 if sometimes present and 0 if never present. The total score varies between 0 (no disability) and 100 (maximum disability) and three classes are defined indicating mild (0-30), moderate (31-60) and severe (61-100) degrees (10).

Statistical Analysis

The distribution of continuous variables was assessed using the Shapiro-Wilk test. Depending on the results of the normality analysis, continuous variables were summarized as mean±standard deviation (SD) for normally distributed data, or as median with minimum-maximum values for non-normally distributed data. Categorical variables were presented as absolute numbers (n) and percentages (%). In the comparisons of continuous variables between the groups, the Mann-Whitney U test or the Kruskal-Wallis test was used as the variables did not show normal distribution. When statistically significant differences were found in more than two group analyses, subgroup analyses were performed using the Dunn-Bonferroni post-hoc tests. For comparisons between groups for categorical variables, Pearson's chi-square test or the Fisher-Freeman-Halton test was used. For relationships between continuous variables, Spearman's correlation test was applied as normal distribution was not provided regarding asymmetry ratios. The SPSS (IBM SPSS Statistics for Windows, Version 28.0. Armonk, NY: IBM Corp.) program was used for statistical analyses and type I error rate was taken as 5% and a p-value <0.05 was considered statistically significant.

Results

The demographic and clinical data are summarized in Table 1. The definitive analysis of audio-vestibular findings are given in Table 2.

There were significant differences in SD (p<0.001), DHI (p=0.006), lateral canal VOR gains (p=0.036) and caloric responses (p=0.036) between the hearing-loss severity groups (Table 3). There were no significant differences with respect to VOR gain asymmetry ratios in lateral, posterior and anterior canals in HIMP, and lateral canal VOR gain asymmetry ratio in SHIMP (p>0.05). The caloric response asymmetry ratio difference was at significance level between the hearing groups (p=0.05).

Figure 1 shows the distributions of asymmetry ratios in significant categoric variables. In DHI groups, patients with normal hearing showed less asymmetry in SHIMP and caloric tests (p=0.035 for both). Lateral canal HIMP and SHIMP asymmetry ratios and caloric response asymmetry ratios differed significantly between the SHIMP and vVOR saccade groups (p=0.025, p=0.002, and p=0.002; p=0.001, p<0.001, and p=0.002, respectively). No meaningful

Table 1. Demographic and disease characteristics of patients

Age; mean±SD	48.94±10.508
Median (min-max)	50 (24-65)
Sex; n (%)	
Female	18 (51.4%)
Male	17 (48.6%)
Employment status; n (%)	
Yes	21 (60%)
No	14 (40%)
Smoking; n (%)	
Yes	7 (20%)
No	28 (80%)
Alcohol use; n (%)	
Yes	8 (22.9%)
No	27 (77.1%)
Comorbid disease; n (%)	
Yes	8 (22.9%)
No	27 (77.1%)
Autoimmune disease; n (%)	
Yes	2 (5.7%)
No	33 (94.3%)
Ménière's disease duration; mean±SD (months)	90.66±56.338
Median (min-max)	96 (12-276)
Ménière's disease side	
Right	21 (60%)
Left	14 (40%)
Attack duration; mean±SD (minutes)	100.29±137.466
Median (min-max)	60 (15-840)
Attack severity; mean±SD (VAS)	3.49±0.612
Median (min-max)	3 (2-5)

Min: Minimum, Max: Maximum, SD: Standard deviation, VAS: Visual analogue scale

Table 2. Definitive analysis of audio-vestibular findings

		n (%)
SNHL degree		
Very mild		8 (22.9%)
Mild		9 (25.7%)
Moderate		6 (17.1%)
Moderate-severe		8 (22.9%)
Severe		2 (5.7%)
Very severe		2 (5.7%)
THI group		
Mild		16 (45.7%)
Moderate		10 (28.6%)
Severe-catastrophic		9 (25.7%)
DHI group		
Mild		7 (20%)
Moderate		16 (45.7%)
Severe		12 (34.3%)
Ipsilateral SHIMP		
Saccade, none		11 (31.4%)
Saccade, present		24 (68.6%)
Ipsilateral vVOR		
Saccade, none		25 (71.4%)
Saccade, present		10 (28.6%)
Ipsilateral SVOR		
Suppression reflex, none		1 (2.9%)
Suppression reflex, present		34 (97.1%)
	Mean±SD	**Median (min-max)**
THI	40.86±21.422	42 (10-84)
DHI	53.37±20.429	54 (16-86)
Ipsilateral PTA (dB)	47.23±26.149	41.25 (18-118)
Contralateral PTA (dB)	18.18±14.330	12.5 (5-66)
Speech discrimination (%)		
Ipsilateral	70.05±26.308	76 (0-96)
Contralateral	93.71±10.551	100 (48-100)
SHIMP VOR gains		
Ipsilateral LC	0.79±0.173	0.86 (0.30-0.98)
Contralateral LC	0.91±0.102	0.90 (0.76-1.20)
HIMP VOR gains		
Ipsilateral AC	0.93±0.190	0.90 (0.56-1.42)
Contralateral AC	0.93±0.190	0.94 (0.50-1.32)
Ipsilateral PC	0.83±0.192	0.87 (0.25-1.29)
Contralateral PC	0.90±0.167	0.88 (0.61-1.34)
Ipsilateral LC	0.84±0.192	0.89 (0.30-1.16)
Contralateral LC	0.92±0.110	0.93 (0.66-1.11)
Caloric test responses		
Ipsilateral	14.91±7.905	14 (2-34)
Contralateral	35.09±12.657	32 (15-62)
Asymmetry ratios		
HIMP LC VOR gain	-11.27±25.276	-8.38 (-87.85-23.91)
HIMP PC VOR gain	-9.67±29.030	-8.79 (-104.76-51.71)
HIMP AC VOR gain	-0.20±24.003	-2.76 (-53.85-66.67)
SHIMP LC VOR gain	-15.63±23.993	-12.76 (-86.79-12.29)
Caloric response	-83.54±38.523	-76.36 (-177.78-38.10)

AC: Anterior canal, DHI: Dizziness handicap inventory, HIMP: Head impulse paradigm, LC: Lateral canal, Min: Minimum, Max: Maximum, PC: Posterior canal, PTA: Pure-tone average, SD: Standard deviation, SHIMP: Suppression HIMP, SNHL: Sensorineural hearing loss, VOR: Vestibulo-ocular reflex, SVOR: Suppression VOR, THI: Tinnitus handicap inventory, VAS: Visual analogue scale, vVOR: Visually-enhanced VOR

significant differences were found in vertical canal gain asymmetry ratios, even diminished VOR gains were observed in the ipsilateral posterior canal in patients with severe hearing loss (Table 3, Figure 1).

The findings of the correlation analysis of asymmetry ratios with audiological and vestibular status of the cases are shown in Table 4. When the relationships between HIMP lateral canal asymmetry ratios and clinical parameters

Table 3. Comparative analysis findings of hearing groups

	Normal (n=8)	**Mild HL (n=15)**	**Moderate HL (n=8)**	**Severe HL (n=4)**	
	n (%)				**p-value**
Sex					
Female	6 (75%)	9 (60%)	3 (37.5%)	0	0.071[a]
Male	2 (25%)	6 (40%)	5 (62.5%)	4 (100%)	
DHI group					
Mild	4 (50%)	3 (20%)	0	0	
Moderate	4 (50%)	7 (46.7%)	3 (37.5%)	2 (50%)	0.083[a]
Severe	0	5 (33.3%)	5 (62.5%)	2 (50%)	
Ipsilateral SHIMP					
Saccade, none	1 (12.5%)	4 (26.7%)	4 (50%)	2 (50%)	0.304[a]
Saccade, present	7 (87.5%)	11 (73.3%)	4 (50%)	2 (50%)	
Ipsilateral vVOR					
Saccade, none	8 (100%)	12 (80%)	4 (50%)	1 (25%)	0.015[a]
Saccade, present	0	3 (20%)	4 (50%)	3 (75%)	
Median (min-max)					
Age	45.5 (32-59)	47 (24-65)	52.5 (31-62)	51.5 (48-64)	0.332[b]
Duration	72 (12-120)	96 (12-192)	108 (36-144)	66 (17-276)	0.841[b]
THI	28 (10-50)	36 (10-80)	44 (20-66)	59 (44-84)	0.118[b]
DHI	29 (16-52)	58 (22-84)	66 (34-86)	60 (58-80)	**0.006[b]**
Speech discrimination					
Ipsilateral	92 (84-96)	80 (64-92)	56 (44-68)	0 (0-48)	**<0.001[b]**
Contralateral	100 (100-100)	100 (84-100)	88 (80-96)	84 (48-100)	**<0.001[b]**
SHIMP gains					
Ipsilateral LC	0.8 (0.8-0.9)	0.8 (0.3-0.9)	0.8 (0.4-0.9)	0.6 (0.6-0.7)	0.200[b]
Contralateral LC	0.9 (0.7-1)	0.9 (0.7-1)	0.9 (0.7-1.2)	0.8 (0.7-0.9)	0.618[b]
HIMP gains					
Ipsilateral AC	0.9 (0.9-1.3)	0.9 (0.7-1.36)	0.8 (0.7-1.1)	0.7 (0.5-1.42)	0.270[b]
Contralateral AC	0.9 (0.8-1.1)	0.9 (0.5-1.3)	0.8 (0.6-1.3)	0.7 (0.5-1)	0.267[b]
Ipsilateral PC	0.9 (0.7-1)	0.8 (0.5-1)	0.8 (0.2-1.2)	0.5 (0.5-0.8)	0.051[b]
Contralateral PC	1 (0.61-1.3)	0.9 (0.7-1.2)	0.8 (0.7-1.1)	0.7 (0.7-1.1)	0.361[b]
Ipsilateral LC	0.9 (0.8-1.1)	0.8 (0.3-1)	0.8 (0.3-1)	0.6 (0.4-0.9)	0.055[b]
Contralateral LC	1 (0.8-1.1)	0.9 (0.7-1.1)	0.8 (0.8-1)	0.8 (0.6-0.9)	**0.036[b]**
Caloric test					
Ipsilateral	18.5 (11-34)	14 (2-31)	15.5 (6-32)	8 (3-14)	0.136[b]
Contralateral	29 (20-56)	37 (17-58)	30 (24-59)	20.5 (15-62)	0.241[b]
Asymmetry ratios					
HIMP LC VOR gain	-1.9 (-17.4-17.2)	-6.7 (-87.8-16)	-15.5 (-79.3-23.9)	-14.3 (-59.7-20.3)	0.546[b]
HIMP PC VOR gain	-2.5 (-35-34)	-6.5 (-47.1-27.5)	-0.1 (-104.7-51.7)	-30.4 (-47-12.1)	0.188[b]
HIMP AC VOR gain	7 (-16.9-37.9)	-7.4 (-24-66.6)	-5.9 (-53.8-31.2)	2.4 (-28.5-40)	0.885[b]
SHIMP LC VOR gain	-7.7 (-13.9-11.1)	-12 (-86.7-12.2)	-20.2 (-50.4-12.2)	-23.1 (-30.7-7.7)	0.422[b]
Caloric response	-52.6 (-80-40)	-80 (-177.7-38.6)	-77.3 (-137.5-38.1)	-99.5 (-153.8-66.6)	**0.050[b]**

[a]: Fisher-Freeman-Halton test, [b]: Kruskal-Wallis test. AC: Anterior canal, DHI: Dizziness handicap inventory, HIMP: Head impulse paradigm, LC: Lateral canal, Min: Minimum, Max: Maximum, PC: Posterior canal, SHIMP: Suppression HIMP, SNHL: Sensorineural hearing loss, VOR: Vestibulo-ocular reflex, SVOR: Suppression VOR, THI: Tinnitus handicap inventory, vVOR: Visually-enhanced VOR

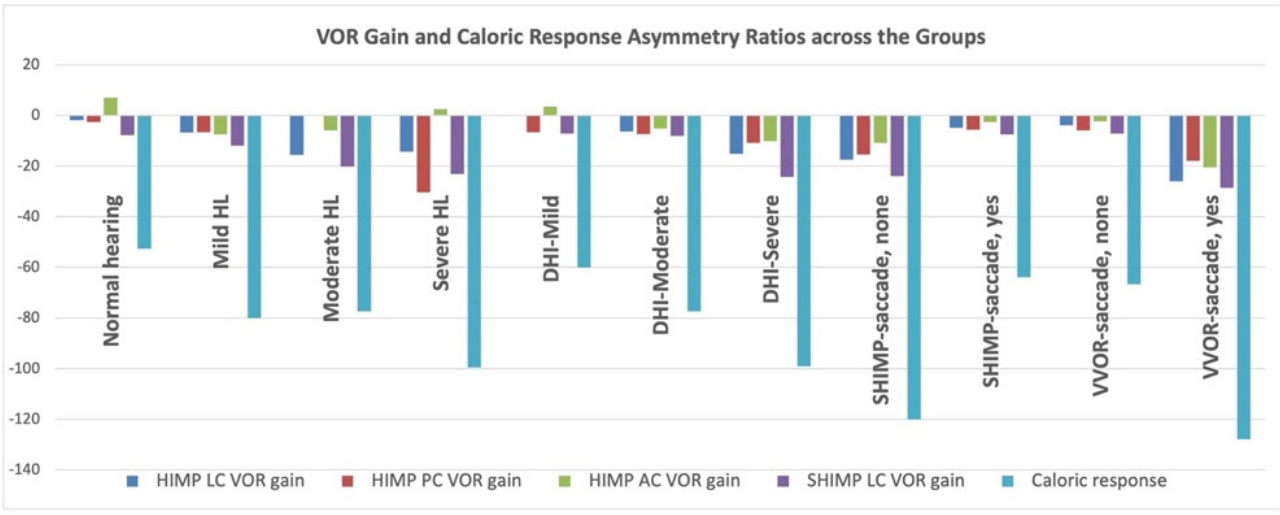

Figure 1. Summary of the comparative analysis of asymmetry ratios between the hearing, dizziness, and saccade groups
HL: Hearing loss, DHI: Dizziness handicap inventory, HIMP: Head impulse paradigm, SHIMP: Suppression HIMP, VOR: Vestibulo-ocular reflex, vVOR: Vissualy-enhanced VOR, LC: Lateral canal, PC: Posterior canal, AC: Anterior canal

were examined, there were weak negative correlations with ACT at 500 Hz (rho=-0.362, p=0.032), DHI (rho=-0.349, p=0.040) and positive correlations with HIMP ipsilateral anterior, lateral and contralateral posterior canal VOR gains (Table 4). SHIMP lateral canal VOR gain asymmetry ratios were negatively correlated with ACT at 500 Hz, 4000 Hz (rho=-0.342, p=0.044; rho=-0.322, p=0.044), ipsilateral PTA (rho=-0.337, p=0.047) and DHI (rho=-0.441, p=0.008), plus, positively correlated with HIMP ipsilateral lateral canal VOR gains (rho=0.381, p=0.024). There were negative correlations between caloric asymmetry ratio and ACT at 1000 Hz (rho=-0.411, p=0.014), DHI (rho=-0.458, p=0.006), plus, high level of positive correlations with ipsilateral and contralateral HIMP lateral canal VOR gains (rho=0.580, p≤0.001; rho=0.434, p=0.009) were observed.

Discussion

The presented study contributes to the evidence and demonstrates VOR asymmetry in unilateral MD and its associations with other clinical characteristics of the disease. Our findings support the idea that MD not only produces fluctuating vestibular hypofunction at low frequency levels but also leads to measurable and sometimes persistent asymmetries in VOR gain. We found significant differences among the hearing groups with respect to contralateral lateral canal VOR gains and caloric response asymmetry. However, VOR gain asymmetry ratios for vertical canals and SHIMP did not differ significantly by hearing groups. Both HIMP and SHIMP lateral canal asymmetry and caloric response asymmetry also varied significantly across saccade-based (SHIMP, vVOR) subgroups. Vertical canal asymmetry was largely nonsignificant, though in those with severe hearing loss there was a trend toward reduced ipsilateral posterior canal gain. In correlation analyses, lateral HIMP asymmetry correlated negatively with ACT at 500 Hz and DHI and positively with ipsilateral anterior, lateral, and contralateral posterior canal VOR gains. Lateral SHIMP asymmetry correlated negatively with ACT 500 Hz, 4000 Hz, ipsilateral PTA, and DHI, and positively with HIMP ipsilateral lateral VOR gain. Caloric asymmetry correlated negatively with ACT at 1000 Hz and DHI, and positively with ipsilateral and contralateral HIMP lateral VOR gains.

Further, the observation of a reduced or absent lateral canal VOR gain asymmetry does not necessarily indicate recovery of the affected ear. Rather, it may reflect either early bilateral vestibular involvement or central compensatory mechanisms that normalize eye movement responses. Over time, the contralateral vestibular apparatus may also exhibit subclinical dysfunction, reducing the apparent inter-aural asymmetry. Simultaneously, central vestibular compensation can recalibrate the VOR, further masking deficits that were initially unilateral (11,12). Therefore, reliance solely on lateral canal asymmetry ratios may underestimate the true disease burden in long-standing MD, and the lack of asymmetry should prompt careful consideration of potential bilateral progression rather than assuming functional recovery.

The pathophysiological basis of VOR asymmetry in MD remains complex. It was highlighted that vHIT VOR responses could differ significantly between the affected and unaffected sides in unilateral MD patients, reflecting both peripheral vestibular damage and central compensatory mechanisms (11). We have shown that VOR asymmetry is not a static feature but could be one that evolves over time as the disease progresses (Table 4). Dynamic changes in VOR gain and asymmetry emphasize the fluctuating nature of

Table 4. Correlation analysis of variables for VOR gain and caloric response asymmetry ratios

	Correlations with VOR gain and caloric response asymmetry ratios				
	HIMP LC	**HIMP PC**	**HIMP AC**	**SHIMP LC**	**Caloric**
Variables	**rho/p**	**rho/p**	**rho/p**	**rho/p**	**rho/p**
Age	-0.302/0.078	-0.148/0.396	-0.098/0.574	-0.102/0.561	-0.246/0.154
Disease duration	-0.094/0.589	0.166/0.341	-0.147/0.398	0.064/0.713	**-0.345/0.042**
Average attack duration	-0.118/0.499	0.036/0.837	-0.131/0.453	-0.120/0.494	-0.271/0.115
Average attack severity	0.014/0.938	-0.017/0.924	0.275/0.110	-0.087/0.619	-0.101/0.563
ACT 500 Hz	**-0.362/0.032**	-0.175/0.314	-0.143/0.412	**-0.342/0.044**	-0.306/0.074
ACT 1000 Hz	-0.278/0.105	-0.147/0.400	-0.211/0.223	-0.301/0.079	**-0.411/0.014**
ACT 2000 Hz	-0.178/0.306	-0.242/0.161	-0.110/0.530	-0.287/0.095	-0.320/0.061
ACT 4000 Hz	-0.162/0.353	-0.219/0.206	-0.020/0.911	**-0.322/0.044**	-0.269/0.118
ACT 8000 Hz	-0.088/0.617	-0.212/0.221	0.068/0.696	-0.293/0.088	-0.147/0.398
Ipsilateral PTA	-0.243/0.160	-0.190/0.275	-0.138/0.430	**-0.337/0.047**	**-0.358/0.035**
Contralateral PTA	-0.102/0.561	**-0.350/0.039**	**0.370/0.029**	-0.023/0.895	-0.138/0.428
Ipsilateral SD	0.219/0.207	0.116/0.508	0.241/0.163	0.271/0.115	0.328/0.054
Contralateral SD	0.095/0.589	0.065/0.711	-0.090/0.609	-0.025/0.887	0.205/0.237
THI	-0.235/0.175	-0.151/0.386	0.021/0.906	-0.274/0.111	-0.186/0.286
DHI	**-0.349/0.040**	-0.025/0.889	-0.154/0.376	**-0.441/0.008**	**-0.458/0.006**
HIMP ipsilateral AC	**0.392/0.020**	-0.119/ 0.497	**0.519/0.001**	0.043/0.808	**0.354/0.037**
HIMP contralateral AC	0.104/0.553	**0.445/0.007**	**-0.333/0.050**	-0.089/0.612	0.156/0.371
HIMP ipsilateral PC	0.274/0.111	**0.650/<0.001**	-0.209/0.229	-0.007/0.967	**0.363/0.032**
HIMP contralateral PC	**0.480/0.003**	**-0.470/0.004**	**0.589/<0.001**	0.251/0.146	**0.367/0.030**
HIMP ipsilateral LC	**0.604/<0.001**	0.284/0.098	0.094/0.590	**0.381/0.024**	**0.580/<0.001**
HIMP contralateral LC	0.114/0.513	0.298/0.082	-0.175/0.315	0.216/0.212	**0.434/0.009**
SHIMP ipsilateral LC	**0.337/0.048**	0.097/0.581	0.112/0.520	**0.806/<0.001**	0.221/0.202
SHIMP contralateral LC	0.147/0.401	0.301/0.079	-0.215/0.214	0.026/0.884	0.080/0.647
Ipsilateral CR	0.273/0.112	0.281/0.102	0.041/0.814	0.312/0.068	**0.711/<0.001**
Contralateral CR	-0.180/0.300	0.045/0.798	-0.165/0.344	0.033/0.852	-0.192/0.270

AC: Anterior canal, ACT: Air-conduction threshold, CR: Caloric response, DHI: Dizziness handicap inventory, HIMP: Head impulse paradigm, LC: Lateral canal, PC: Posterior canal, PTA: Pure-tone average, SHIMP: Suppression HIMP, THI: Tinnitus handicap inventory, SD: Standard deviation

cochlea-vestibular dysfunction and its potential progression (12). These findings align with our observations; a decrease in both ipsilateral and contralateral lateral canal VOR gains suggests that careful monitoring of asymmetry trajectories could provide insights into disease activity and prognosis. As the disease is characterized by a fluctuating pattern of symptoms that wax and wane, vHIT gains and saccades may show the compensation state of the patient at the time of admission.

As vHIT measures the VOR gains during high-velocity rotatory head impulses, a consensus has not been reached on its role in the follow-up of MD (13). While HIMP is good for detecting VOR deficits (presence of corrective saccades), SHIMP is better for assessing how much vestibular reserve remains (presence/absence of compensatory saccades), and the caloric test shows lateral canal paresis and canal asymmetry. Together, they give a complementary picture of fluctuating and progressive dysfunction in MD (14).

Nevertheless, the dissociation between caloric deficits and relatively preserved vHIT (HIMP) is reported in many patients: caloric abnormalities are more frequent, whereas vHIT gain abnormalities (or asymmetry) are less common in the interictal period (15,16). Our data support that VOR gain asymmetry in HIMP (lateral, vertical canals) and SHIMP do not differ across hearing loss severity, indicating that gain asymmetry is not sensitive to the severity of hearing loss in MD, at least in our cohort; although the patients with normal hearing showed less asymmetry in HIMP lateral canal VOR gains and caloric responses. The correlations between the asymmetry ratios and the other variables showed that when ipsilateral weakness is more prominent DHI and hearing thresholds are increased and VOR gains, and caloric responses are diminished.

The absence of significant vertical canal asymmetry differences is also in line with many reports: in MD, the lateral canal is most often implicated, whereas vertical

canals tend to remain relatively spared in interictal testing (17). The slight gain reduction in the ipsilateral posterior canal in severe hearing loss may reflect the early spread of endolymphatic hydrops involvement beyond the lateral canal in advanced disease. Pathophysiologically, this may reflect hydrops affecting the posterior labyrinth or secondary damage to vertical canal hair cells. Along with the lateral canal, the posterior canal was the most frequently abnormal canal on vHIT, in almost 56% of peripheral vestibulopathy cases in a study (18). The distribution profiles of canal involvement from multiple reports confirm that the posterior canal is often involved (sometimes more than horizontal) in MD patients (19).

A study conducted with 36 patients with definite MD found that the most frequent gain reduction in vHIT was in the posterior canal of the affected ear and in the coupled superior canal of the "unaffected" ear (20). In another study, it was shown that over time, VOR gain in vertical canals (superior and posterior) declined, whereas the horizontal canal gain remained relatively stable in the same interval. The authors interpreted this as evidence that vertical canal deterioration could precede or outpace horizontal canal decline in early to mid-stages of MD (21).

Importantly, posterior canal hypofunction on vHIT can be asymmetric and isolated and may persist between attacks, providing an additional diagnostic marker when lateral canal asymmetry has normalized due to bilateral progression. Detecting saccades and reduction in unilateral posterior canal VOR gain offers two key advantages: sensitivity to early or subclinical involvement, and diagnostic help. Since accompanying ipsilateral posterior canal hypofunction is unusual in other peripheral disorders, its presence strongly supports MD in the context of fluctuating audio-vestibular symptoms.

Another dimension is the role of SHIMP in MD. By design, SHIMP presents a head-fixed moving target that the subject must pursue, thereby eliciting anti-compensatory saccades; SHIMP is less influenced by covert saccades that can contaminate HIMP gain measures (4). Thus, VOR gain asymmetry assessed by SHIMP and HIMP provides complementary information in unilateral MD. Asymmetry in SHIMP was also not significantly stratified by hearing groups in our cohort, but correlations suggest that even when covert saccades are suppressed, SHIMP is still sensitive to audiometric severity in MD. SHIMP asymmetry seems to be correlated better with hearing-loss severity, possibly indicating higher sensitivity to residual vestibular dysfunction. In isolation, vHIT (gain) asymmetry may lack power, but when combined with saccadic behavior, it becomes more sensitive to vestibular dysfunction. For example, in the absence of saccades in SHIMP, we observed greater asymmetry in caloric responses. This observation aligns with the calls in literature to emphasize the importance of saccades rather than simple gain cut-offs (17). Our findings also depicted that asymmetry ratios are significantly increased in the presence of saccades in vVOR, which marks vestibular loss in MD (22).

With the progression of unilateral MD, the caloric-vHIT pattern tends to shift, which may reflect the deterioration of endolymphatic hydrops and vestibular hair cell impairments (12,23). In the early stages of MD, vHIT is often normal because this test assesses high-frequency vestibular fibers, and the disease initially affects primarily low-frequency systems (17,24). However, as the disease progresses, vHIT gain may decrease, and a significant gain reduction may occur in the affected ear. In some patients, changes in the VOR response may be observed in the contralateral ear due to subclinical effects or central compensation (23).

Even in cases of unilateral disease, VOR asymmetry is not pathognomonic of MD; rather, it should be interpreted in the context of the status and progress of the disease. Parameters suggesting incomplete compensation can contribute to chronic imbalance and reduced quality of life in MD patients. Our results support this idea, as persistent VOR asymmetry in unilateral MD may reflect insufficient central compensation. Periodic dysfunction of the VOR varies over time, and VOR gain may vary not only in the affected ear but also in the contralateral ear, and this can be assessed by VOR gain asymmetry. Taken together, larger study samples will highlight whether VOR asymmetry is a sensitive marker of unilateral vestibular dysfunction in MD, with diagnostic, prognostic, and rehabilitative implications. Future prospective studies should clarify how fluctuations in VOR asymmetry relate to clinical symptomatology and whether targeted interventions can mitigate long-term disability.

In our study, greater symmetry (less asymmetry) was associated with better hearing and less dizziness handicap. This matches clinical intuition: more preserved vestibular symmetry corresponds with milder functional impairment. However, MD is progressive and not rarely bilateral; longitudinal cohorts report contralateral ear involvement in up to 30-50% over 10-20 years (20). As the disease evolves, contralateral canal responses may deteriorate, resulting in diminished asymmetry ratios despite ongoing symptoms, as was observed in our study. Clinically, loss of asymmetry in a clinically unilateral case should raise suspicion for bilateral vestibular involvement rather than recovery. Thus, the lateral canal asymmetry ratio may underestimate disease burden in long-standing MD.

Study Limitations

Because testing was done at single timepoints with a single-center design, the influence of recent asymmetry dynamics

could not be standardized to the attack or interictal period of MD. A limitation of applying the results to clinical practice is that relying on the lateral canal asymmetry ratio alone may be misleading when contralateral involvement occurs or the disease progresses. Additionally, decreased VOR gain asymmetry over time may be an important clue in diagnosing bilateral MD.

Conclusion

VOR gain asymmetry assessed with SHIMP and HIMP offers complementary diagnostic value in unilateral MD. Both VOR gain and caloric response asymmetry rates could serve as adjunct markers for evaluating vestibular dysfunction in MD.

Ethics

Ethics Committee Approval: The study was approved by the Clinical Research Ethics Committee of the University of Health Sciences Türkiye, Bursa Yüksek İhtisas Training and Research Hospital (approval no: 2011-KAEK-25 2023/04-10, date: 19.04.2023).

Informed Consent: Informed consent was obtained from all the patients and subjects.

Footnotes

Authorship Contributions

Surgical and Medical Practices: N.G.Y.A., B.E., Concept: N.G.Y.A., B.E., Design: N.G.Y.A., B.E., Data Collection and/or Processing: N.G.Y.A., B.E., Analysis or Interpretation: N.G.Y.A., B.E., Literature Search: N.G.Y.A., B.E., Writing: N.G.Y.A., B.E.

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Main Points

- While there are relationships between head impulse paradigm (HIMP) and suppression HIMP (SHIMP) vestibulo-ocular reflex (VOR) gains and hearing thresholds at low frequency and dizziness handicap inventory, the caloric test correlates more closely with audiological impairment and symptom burden in unilateral Ménière's disease (MD).
- SHIMP provides complementary insight, and anti-compensatory saccades might be useful for disease monitoring.
- Incorporating SHIMP and visually enhanced VOR saccade metrics and asymmetry indices into clinical reports could improve the early detection of progression.
- Given these observations, vHIT gain asymmetry alone should not be interpreted as a marker of MD severity; instead, integrated vestibular profiling is warranted.

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