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Video Head Impulse Testing in Relapsing Remitting Multiple Sclerosis: Evaluation of Canal Gains, Asymmetry, and Corrective Saccades with Cerebellar Lesions

Emel Tahir[1], Asuman Küçüköner[1], Esra Kavaz Uştu[1] ✉, Sedat Şen[2] and Murat Terzi[2]

[1] *Ondokuz Mayıs University Faculty of Medicine Department of Otorhinolaryngology, Samsun, Turkey*

[2] *Ondokuz Mayıs University Faculty of Medicine Department of Neurology, Samsun, Turkey*

ABSTRACT

Objective: This study aimed to compare video head impulse test (vHIT) metrics in relapsing–remitting multiple sclerosis (RRMS) versus controls, test differences by cerebellar MRI status within RRMS, and explore clinical/MRI correlates of corrective saccades. Methods: Single-center prospective study including 35 adults with RRMS and 35 age-/sex-matched controls. Disability was quantified by EDSS; a blinded neuroradiologist classified cerebellar involvement. The MS cohort was categorized into two subgroups according to the occurrence of cerebellar lesions. Each group participated in a vHIT to assess VOR gain. Results: RRMS participants had low disability (mean EDSS 0.95). Compared with controls, RRMS showed lower VOR gain in the right anterior/posterior, left posterior, and left lateral canals and higher asymmetry across RALP, LARP, and lateral planes ($p = 0.012$, 0.024, < 0.001). Corrective saccades differed between groups only in the left anterior canal ($p = 0.025$). Within RRMS, cerebellar-positive versus cerebellar-negative subgroups did not differ for gain or asymmetry (all $p > 0.05$); corrective-saccade prevalence was similar (33.3% vs 35.0%; chi-square $p = 0.918$). In regression, cerebellar involvement, relapse count, EDSS, and disease duration were not associated with recurrent corrective saccades. Conclusion: vHIT reveals canal-selective gain reductions and elevated asymmetry in patients with mildly disabled RRMS, but these abnormalities do not differ by cerebellar MRI status.

KEYWORDS

multiple sclerosis, vertigo, video head impulse test.

1 Introduction

Multiple sclerosis (MS) is a chronic immune-mediated demyelinating disorder of the central nervous system and a primary cause of non-traumatic impairment in young adults (Reich et al., 2018). Dizziness, imbalance, and episodic vertigo are common across the disease course and may even be presenting features, motivating objective vestibular assessment early on (Cochrane et al., 2021). Vertigo may manifest as the initial symptom of multiple sclerosis in 4-20% of instances (de Lima Evangelista et al., 2023).

Notably, global lesion burden on conventional MRI does not consistently track functional handicap; infratentorial involvement —particularly brainstem and cerebellar disease —often carries disproportionate clinical weight (Barkhof, 2002). Studies utilizing a broad spectrum of laboratory vestibular tools - oculomotor recordings, vestibular-evoked myogenic potentials, and posturography - showed that patients with MS produced abnormal results when compared to healthy controls in general (Gür et al., 2022). Analyzing the performance of individuals with MS on objective diagnostic vestibular tools can enhance our comprehension of vestibular function in MS more effectively (Cochrane et al., 2021). The vestibular-ocular reflex (VOR), mediated by the semicircular canals, is commonly assessed by video-head impulse testing (vHIT) (Halmagyi et al., 2008). vHIT findings in patients with MS have been heterogeneous: some studies show no impairment, whereas others report abnormal gains and/or frequent corrective saccades in a meaningful subset (Pavicic et al., 2017; Pavlović et al., 2017; Shandiz et al., 2021). Such variability likely reflects lesion topography, oculomotor comorbidities (e.g., internuclear ophthalmoplegia), and analytic choices; critically, mean gain may remain near normal while velocity-dependence and saccadic patterning are altered by brainstem–cerebellar dysfunction (Pavlović et al., 2017).

The neuroanatomical rationale for interrogating vHIT in MS is strong. Vestibular nuclei in the brainstem integrate semicircular-canal input and project to the cerebellar flocculus/paraflocculus, which calibrate VOR gain and shape corrective saccades (Kheradmand and Zee, 2011). Brainstem pathology can disrupt VOR timing and increase reliance on corrective saccades, and vHIT abnormalities have been associated with brainstem lesions in MS (Halmagyi et al., 2008; Pavlović et al., 2017). Cerebellar pathology may leave mean gain within normative bounds yet alter its dependence on

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* Corresponding author Esra Kavaz Uştu, dresrakvz@gmail.com

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head velocity and modify the latency and clustering of corrective saccades (Shemesh and Zee, 2019). In addition, ocular motor pathway disease—most notably internuclear ophthalmoplegia (INO) due to medial longitudinal fasciculus involvement — introduces disconjugacy that can bias vHIT readouts (Nerrant and Tilikete, 2017). Accordingly, vHIT features beyond mean gain represent biologically plausible and potentially more sensitive markers of infratentorial dysfunction in MS. Despite multiple vestibular investigations in MS, comparatively few studies have stratified vHIT outcomes by cerebellar involvement while accounting for brainstem lesions and basic oculomotor confounders (Mañago et al., 2017). Understanding the effect of cerebellar lesions on vestibular function is critical because it may aid in the early detection and management of balance issues in MS. Addressing this gap may reconcile inconsistent reports and refine the clinical utility of vHIT in MS.

The main aim of this study was to evaluate the efficacy of the vHIT in assessing vestibular function in individuals with RRMS and to determine if vHIT parameters varied between people with MS with and without cerebellar involvement. Furthermore, we were interested in comparing these vHIT values between MS patients and a healthy control group to determine the amount of vestibular dysfunction in MS. The secondary end point of this study is to determine the role of vHIT as a diagnostic tool in detecting vestibular dysfunction caused by MS by relating vHIT findings to disease severity and brain magnetic resonance imaging MRI results.

We do not position vHIT as a stand-alone diagnostic for MS-related vestibulopathy; rather, we test its value as a complementary physiological measure that may capture infratentorial dysfunction beyond what global lesion counts convey.

2 Material and Methods

2.1 Study design

This single-center prospective case-control study was performed at the Otorhinolaryngology Department of Ondokuz Mayıs University in compliance with the Declaration of Helsinki. The Institutional Clinical Research Ethics Committee granted approval (approval no. 2021/503). Informed written consent was obtained from all subjects before enrollment.

2.2 Participants

We recruited 35 persons with RRMS, diagnosed according to the McDonald criteria, alongside 35 age- and sex-matched healthy controls. Demographic data, relapse frequency, disease duration, and neurological examination results were documented for patients with RRMS. The Expanded Disability Status Scale (EDSS) was employed to quantify disability.

2.3 MRI Acquisition and Lesion Classification

Routine clinical brain MRI scans were reviewed for research purposes. A board-certified neuroradiologist, blinded to clinical symptoms and all vHIT results, classified lesion topography as brainstem involvement (present/absent) and cerebellar involvement (present/absent). When available, total T2/FLAIR lesion counts were noted; these imaging studies were acquired independently of the present study.

2.4 Video Head Impulse Test (v-HIT)

Prior to vHIT, all patients were evaluated neurologically by the Neurology Department (S.Ş. and M.T.), and ocular motor examination findings were reviewed clinically. No clinically evident internuclear ophthalmoplegia or other overt ocular motor abnormality that could interfere with reliable vHIT interpretation was present at the time of testing in the patients. The vHIT was performed using EyeSeeCam (Interacoustics, Assens, Denmark) with monocular high-speed video-oculography (sampling 150–250 Hz). During the test, participants were asked to focus on a fixed target located approximately 1.2 meters away. All tests were conducted by a single researcher (A.K.). First, the device was calibrated. During this procedure, the patient was asked to focus on five points in front of him, each spaced 8.5° apart. The dots were created using a laser and a diffraction grating mounted on a pair of goggles. The fixation order was randomized, and the participant was instructed not to focus on each dot for more than one second. If any calibration error was detected, the researcher could repeat this process.

The examiner stood behind the seated participant and delivered unpredictable low-amplitude head impulses (≈10–20°) in the horizontal plane targeting peak head velocity ≥150°/s. With the patient's gaze fixed on the target, the head was pushed at an angle of 10-20 degrees in the horizontal plane and at a speed of 150°/s. The head thrust was performed at a moment and in a direction that the patient did not expect. These movements were performed in different planes: right, left, right anterior-left posterior (RALP), and left anterior-right posterior (LARP). A minimum of 10 impulses per direction was acquired. Traces contaminated by blinks, recording drop-outs, or suspected goggle slippage were excluded a priori. The device automatically determined the patient's vestibulo-ocular reflex (VOR) gain. VOR gain was defined as the ratio of the angular velocity of head movement to the angular velocity of eye movement at ~60 ms after impulse onset (device default). VOR gain ≥ 0.8 was accepted as the standard reference value for horizontal channels and ≥ 0.7 for vertical channels. In addition to canal-wise mean gain, corrective saccades (covert and overt saccades) were recorded, and the speed and time of these movements were analyzed. Corrective saccades were automatically detected by the device and visually confirmed by the examiner. Covert saccades occur during the head impulse (before head velocity returns to zero), and overt saccades occur after the head impulse. For each canal, we extracted saccade latency (ms) from impulse onset, peak velocity (°/s), amplitude (°), and saccade rate (percentage of impulses containing ≥1 corrective saccade). A pathological vHIT response was prespecified as (a) canal gain below the reference threshold or (b) recurrent corrective saccades, defined as corrective saccades present in ≥50% of impulses for that canal with peak velocity >100°/s. Saccade asymmetry was calculated if the observed corrective saccades differed in frequency and intensity between right and left canal movements. Asymmetry was assessed by comparing VOR gains and saccades in both directions, and cases with a significant difference were considered asymmetric (Halmagyi et al., 2017; Psillas et al., 2022)

2.5 Statistical Analysis

The R Project software was utilized for statistical data analysis. The Kolmogorov-Smirnov and Shapiro-Wilk tests were utilized to ascertain whether the data follows a normal distribution. Non-parametric tests were employed when the variables had non-normal distribution. The variables were expressed as mean ± standard deviation, median (minimum-maximum), or median interquartile range (IQR). The independent samples t-test was utilized for regularly distributed data, whereas the Mann-Whitney U test was

employed for non-normally distributed variables. Chi-square tests (Pearson's Chi-square or Fisher's exact test) were conducted to assess categorical data. Binomial logistic regression models (dependent variable = presence/absence of corrective saccades) were developed to ascertain if cerebellar lesions, attack frequency, EDSS score, and disease duration affect the identification of corrective saccades in MS patients. All tests were two-tailed, and p-values less than 0.05 were deemed statistically significant.

2.5.1 *Sample Size Calculation*

The sample size was calculated using G*Power (v3.1.7). A prior study (Ertugrul et al., 2020) generated an effect size of 1.315. A post-hoc power analysis revealed that a sample of 70 participants in two equally sized groups achieved a power (1- β) of 0.999.

3 Results

We analyzed 35 patients with RRMS and 35 healthy controls. No significant difference was found between the patient (male/female=5/30; mean age=45.0±11.7) and control group (male/female=10/25; mean age=41.0±10.1) in terms of age and sex distribution (p=0.066 and p=0.145, respectively). In patients with RRMS, mean disease duration was 7.04 ± 5.47 years; EDSS ranged 0–3.5 (mean 0.95), and the number of attacks was 2.62 (1-8). MRI identified cerebellar lesions in 15/35 (42.9%) patients. Table 1 summarizes the demographic information from the MS and control groups.

Compared with controls, the MS group showed statistically significantly lower VOR gain in the right anterior, right posterior, left posterior, and left lateral canals (Table 2). Canal-pair asymmetry indices were higher in MS for RALP, LARP, and lateral canals (Table 3; p = 0.012, 0.024, and <0.001, respectively).

While no overt and covert saccades were observed in healthy controls, they were observed in 18 patients in the MS group. Overt saccades were present in 10 recordings, whereas covert saccades were present in 13 recordings; specifically, 5 recordings had overt-only saccades, 8 had covert-only saccades, and 5 had both overt and covert saccades. When the distribution of corrective (covert/overt) saccades between the groups was analyzed, only the presence of corrective saccades in the left anterior canal (n=6) was higher than in the control group (p=0.025), as there was no difference between the groups in the other canals (p values>0.05) (Table 4).

MS patients were divided into two groups based on the presence or absence of cerebellar lesions, and their VOR gains were analyzed. No statistically significant difference was found between these two subgroups in terms of VOR gains in any channel (p values

Table 1 Descriptive Characteristics of the Groups

	MS (n = 35)	HC (n = 35)	p
Age (Mean ± SD)	45.0 ± 11.7	41.0 ± 10.1	0.066[a]
Sex (M/F)	5/30	10/25	0.244[b]
EDSS Score (Mean (min-max))	0.95(0-3.5)	-	
Disease Duration (years) (Mean ± SD)	7.04 ± 5.47	-	
Number of attacks (Mean (min-max))	2.62(1-8)	-	
Cerebellar lesions on MRI (n (%))	15 (42.9%)	-	

[a]independent samples t test, [b]Chi square test with Yates correction

Abbreviations: n, number; M, male; F, female; MS, patients with multiple sclerosis; HC, healthy controls; SD, standart deviation; min, minimum; max, maximum; EDSS, Expanded Disability Status Scale

Table 2 VOR Gains of MS and Healthy Controls.

SSC		Mean ± SD	Median (IQR)	p value
Right Anterior	MS	0.95 ± 0.33	0.97 (0.35)	0.032[a]
	HC	1.09 ± 0.16	1.14 (0.19)	
Left Anterior	MS	1.01 ± 0.37	1.06 (0.56)	0.181[a]
	HC	1.10 ± 0.13	1.14 (0.18)	
Right Lateral	MS	0.96 ± 0.33	0.95 (0.27)	0.597[b]
	HC	0.98 ± 0.14	0.97 (0.14)	
Left Lateral	MS	0.85 ± 0.27	0.87 (0.29)	< 0.001[b]
	HC	1.04 ± 0.15	1.03 (0.09)	
Right Posterior	MS	0.96 ± 0.41	0.94 (0.46)	0.013[b]
	HC	1.12 ± 0.16	1.13 (0.18)	
Left Posterior	MS	0.89 ± 0.39	0.84 (0.38)	< 0.001[b]
	HC	1.09 ± 0.16	1.13 (0.17)	

[a] independent samples t test,[b] Mann Whitney U test. Bold prints in p value column indicates a statistically significant difference between patients with multiple sclerosis and healthy controls.

Abbreviations: VOR, vestibüloocular reflex; MS, patients with multiple sclerosis; HC, healthy controls; SSC, semicircular canal; SD, standart deviation; IQR, interquartile range.

Table 3 Comparison of Paired Channel Asymmetries Between Groups.

	MS Median IQR	HC Median IQR	p[a]
RALP Asymmetry	7.0 (9.50)	3.0 (4.50)	0.012
LARP Asymmetry	12.0 (15.0)	4.0 (5.0)	0.024
Lateral Asymmetry	7.0 (8.50)	5.0 (4.0)	<0.001

a Mann Whitney U test. Bold prints in p value column indicates a statististically significant difference between patients with multiple sclerosis and healthy controls.

Abbreviations: RALP, right anterior and left posterior canal; LARP, left anterior and right posterior canal; MS, patients with multiple sclerosis; HC, healthy controls; IQR, interquartile range.

Table 4 Comparison of the Groups in terms of the Numbers of Patients with ≥ 1 Corrective (Overt/Covert) Saccades

	MS (n)	HC (n)	p[a]
Right Anterior	5	0	0.054
Left Anterior	6	0	0.025
Right Lateral	3	0	0.239
Left Lateral	1	0	0.999
Right Posterior	5	0	0.054
Left Posterior	2	0	0.493

[a]Fisher's exact test

Abbreviations: n, number; MS, patients with multiple sclerosis; HC, healthy controls range.

>0.05) (Figure 3). No difference was found between these two subgroups in terms of LARP, RALP and lateral channel asymmetries (p=0.228, p=0.841 and p=0.223 respectively) (Figure 4). While 5 (33.3%) of 15 patients with cerebellar lesions had corrective saccades, 7 (35.0%) of 20 patients without cerebellar lesions had corrective saccades. There was no significant correlation between the presence of corrective saccades and the presence of cerebellar lesion (Chi-square test, p=0.918). In the logistic regression models established to investigate whether the presence of cerebellar lesion, number of attacks, EDSS score, and disease duration affect the detection of corrective saccades in MS patients, no association was found between any of these variables and the presence of corrective saccades (p=0.748, p=0.310, p=0.607, and p=0.904, respectively).

4 Discussion

In this case–control cohort of patients with RRMS who had low disability (mean EDSS about 1.0), we found two main patterns. First, some semicircular canals showed lower VOR gain in MS than in healthy controls (right anterior, right posterior, left posterior, and left lateral). Second, canal-pair asymmetry was higher in MS across RALP, LARP, and lateral planes. Corrective saccades (covert or overt) were more frequent only for the left anterior canal. Within the MS cohort, vHIT metrics did not differ between patients with versus without cerebellar involvement on MRI, and cerebellar status did not predict recurrent corrective saccades in adjusted models.

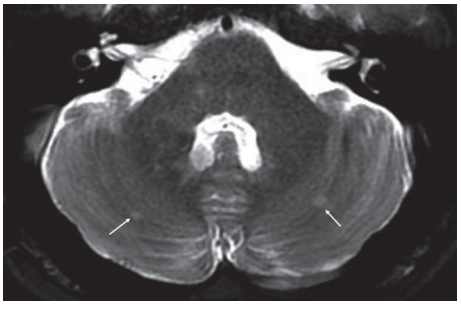


Figure 1 Presence of cerebellar plaque on magnetic resonance imaging.

Published vHIT studies in MS do not all show the same thing. (Ertugrul et al. 2020) reported canal-specific gain reductions in childhood-onset; (Pavlović et al. 2017) linked vHIT abnormalities to brainstem lesions; (Grove et al. 2022) found that worse vHIT metrics accompany higher disability; Eğilmez et al. (2022) suggested vHIT and cVEMP can be informative even when MRI shows no brainstem lesion; (Wagner et al. 2022) showed that compensatory saccade behavior differs between peripheral hypofunction and MS; and (Tramontano et al. 2024) correlated VOR abnormalities with balance/gait impairment. Our results sit in the middle of this spectrum: small but real gain reductions in several canals, apparent asymmetry, and only a limited difference in corrective saccades. This pattern supports the idea that we should not look only at mean gain but also at how gain changes with head speed and at saccade timing and clustering, especially in central nervous system disorders.

We compared MS patients with and without cerebellar MRI lesions because the cerebellar flocculus helps fine-tune VOR gain and shapes corrective saccades. However, cerebellar damage can leave the average gain close to normal while changing more subtle features (for example, how gain depends on head-impulse velocity, or how early and how consistently corrective saccades appear). Since our primary analyses focused on mean canal gain and the presence/absence of corrective saccades, we may have overlooked more subtle cerebellar effects. In addition, our cerebellar subgroups were small (15 vs 20), which reduces statistical power. By contrast, brainstem lesions act directly on vestibular-nuclear timing and typically increase the need for catch-up saccades; several studies - including (Pavlović et al. 2017)- link vHIT abnormalities to brainstem involvement. Therefore, a "no difference by cerebellar status" result is biologically consistent with the literature and suggests that future studies should stratify by brainstem lesions (or model brainstem and cerebellum together) and should prespecify velocity-

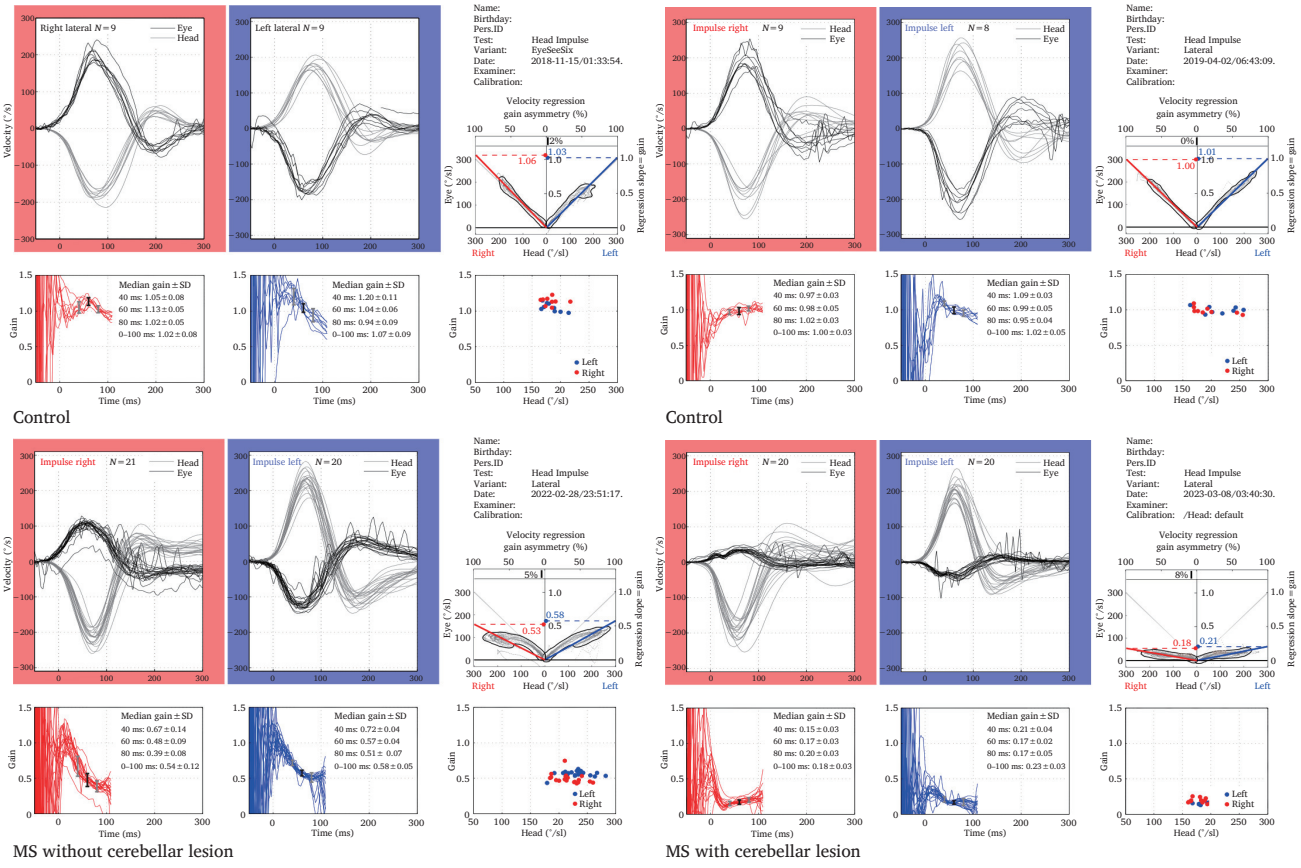


Figure 2 vHIT findings of one patient in the control group (normal gain) two patient with and without cerebellar lesions in the MS group (abnormal gain).

binned gain and saccade microstructure outcomes.

MS commonly affects both visual afferents and ocular motor control. Shandiz et al. investigated direct visual acuity (DVA) and vHIT in MS patients, concluding that vHIT was a more reliable test than MRI findings alone, especially in patients with brainstem lesions. Their study showed that VOR function was independent of disease duration (Shandiz et al., 2021). This finding aligns with the present study, where vHIT was used to assess a wide range of central vestibulopathies, particularly in the context of MS. At the same time, vHIT outputs can be biased by INO-related disconjugacy and technical artifacts; we therefore standardized calibration, matched head-velocity distributions, and excluded blink/drop-out/slippage traces.

Vertigo manifests as a presenting symptom in 4-20 % of patients with MS (de Lima Evangelista et al., 2023). Plaques in the brainstem, especially in the fourth ventricle, may disrupt otolithic projections from the vestibular nuclei to the superior cerebellar peduncle, resulting in positional vertigo (Karatas, 2008). Our work demonstrated that vHIT yielded markedly more anomalous outcomes in MS patients than in healthy controls, as indicated by reduced VOR gains and the occurrence of overt/covert saccades. The findings indicate possible demyelinating lesions in the vestibular nerve's root exit region, vestibular nucleus, or deep cerebellar areas that govern the VOR; nevertheless, the current study lacks sufficient conclusiveness to definitively identify the lesion locations.

Other studies have also explored the role of vHIT in assessing vestibular dysfunction in MS. (Ertugrul et al. 2020) found that the vestibulo-ocular system was impaired in childhood-onset MS patients, with significantly lower VOR gains in certain semicircular canals compared to healthy controls. (Pavlović et al. 2017) demonstrated that vHIT might identify brainstem pathology in MS patients, given the strong correlation between brainstem MRI lesions and bilateral pathology on vHIT for the lateral semicircular canal. These studies highlight the potential of vHIT as a diagnostic tool in MS, particularly for detecting brainstem lesions.

We do not propose vHIT as a stand-alone, lesion-localizing diagnostic in MS. We position it as a complementary physiological tool that can flag infratentorial dysfunction even when mean gain is near normal. Elevated asymmetry and specific corrective-saccade patterns can support earlier and more targeted vestibular rehabilitation. The ability of vHIT to test all six canals at physiological head speeds also makes it practical in ENT and neurology clinics.

Our study has several strengths: matched healthy controls, blinded neuroradiology review, and a single vHIT platform with explicit quality control. It also has several limitations, including a single centre, modest subgroup sizes, monocular eye recording, and a cross-sectional design. Importantly, although all patients had RRMS and low EDSS, the mean disease duration was about 7 years, so we should not claim “early-stage” involvement. Another limitation is that there is no single, universally accepted standard screening protocol for subtle or subclinical intranuclear ophthalmoplegia (INO) to detect potential abnormal ocular movements. Development of a standardized INO assessment framework may improve methodological consistency and strengthen the interpretation of vHIT findings in future studies. Finally, we ran many canal-wise comparisons; therefore, single-canal findings (like the left anterior canal for corrective saccades) should be viewed with caution and confirmed in larger samples. Reporting effect sizes and controlling for multiple testing helps, but replication is still

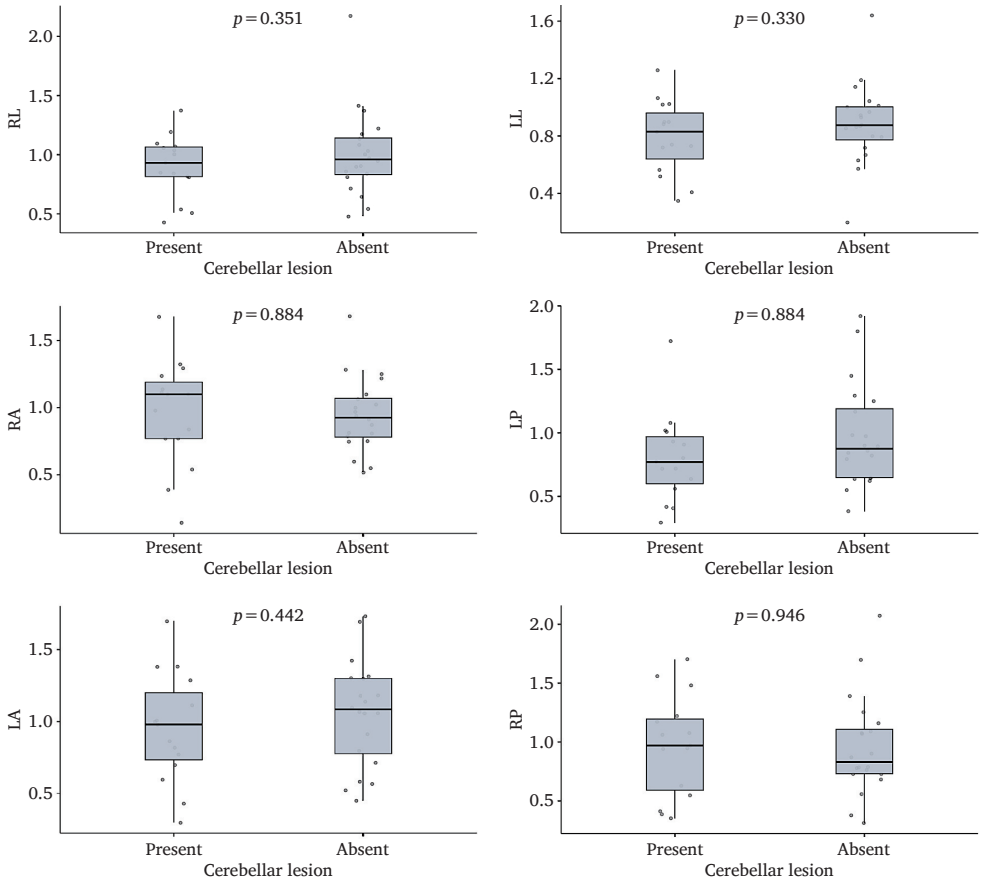


Figure 3 Vestibulo-ocular refkels (VOR) gains semicircular canals according to the presence/absence of cerebellar lesions. *Abbreviations:* RL, right lateral; LL, left lateral; RA, right anterior; LP, left posterior; LA, left anterior; RP, right posterior

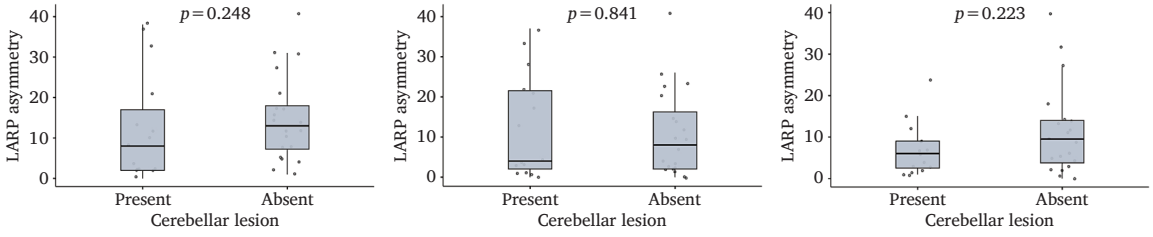


Figure 4 Left anterior-right posterior (LARP), right anterior-left posterior (RALP) and lateral channel asymmetries according to the presence/absence of cerebellar lesions.

needed.

Future studies should be larger and preferably multicentre. They should predefine velocity-binned gain and saccade microstructure (latency, clustering) as outcomes, include a simple oculomotor screen to detect INO, and stratify clearly by brainstem as well as cerebellar lesion load. Combining vHIT with other tests (for example, VEMP, ocular-motor recordings, posturography) will help connect MRI topography with high-speed vestibular physiology and with symptoms such as dizziness and imbalance.

5 Conclusion

In mildly disabled RRMS, vHIT demonstrated canal-selective reductions in VOR gain and elevated canal-pair asymmetry relative to controls, while corrective saccades differentiated groups in a single canal. These physiological abnormalities did not differ by cerebellar lesion status, suggesting that mean-gain-based vHIT measures may be relatively insensitive to cerebellar involvement or that subgroup power was insufficient to detect subtle effects. Given prior associations between vHIT abnormalities and brainstem pathology, future work should prioritize brainstem-focused stratification and velocity-dependent/saccadic metrics. We support the use of vHIT as a cost-effective, feasible adjunct for monitoring vestibular dysfunction in RRMS and for informing timely vestibular rehabilitation —without over-interpreting it as a lesion-localizing diagnostic.

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Data availablity

The data underpinning the conclusions of this work are publicly accessible in Mendeley Data Storage as follows: Tahir Emel (2026), 'Vestibulocular reflex manifestations of relapsing remşitting multiple sclerosis with or without cerebellar involvement', Mendeley Data, V1, doi: 10.17632/hkgzm6cnx2.1. The data has been sub-

mitted to the journal and can be shared at any time upon request.

Ethical approval

The study was conducted in compliance with the ethical norms of the Helsinki Declaration and its subsequent amendments, receiving approval from the Institutional Clinical Research Ethics Committee. (OMUKAEK 2021/503).

Informed consent

Informed written consent was obtainedfrom all subjects before enrollment.

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Disclosure of interests

The data underpinning the conclusions of this work are publicly accessible in Mendeley Data Storage as follows: Tahir Emel (2026), 'Vestibulocular reflex manifestations of relapsing remşitting multiple sclerosis with or without cerebellar involvement', Mendeley Data, V1, doi: 10.17632/hkgzm6cnx2.1. The data has been submitted to the journal and can be shared at any time upon request.

CRediT authorship contribution statement

Emel Tahir: Conceptualization, Validation, Methodology, Formal analysis, Investigation, Writing - original draft.

Asuman Küçüköner: Investigation, Resources, Methodology, Writing - review & editing.

Esra Kavaz Uştu: Investigation, Resources, Writing - review & editing

Sedat Şen: Resources, Investigation, Writing - review & editing

Murat Terzi: Supervision, Writing - review & editing.

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