

Vestibular dysfunction in migraine: effects of associated vertigo and motion sickness

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Abstract The mechanisms of vestibular migraine and motion sickness remain unknown. The aims of this study were to determine interictal vestibular dysfunction in migraineurs according to associated dizziness/vertigo and motion sickness, and to find out whether impaired uvulonodular inhibition over the vestibular system underlies the vestibular symptoms and signs by measuring tilt suppression of the vestibulo-ocular reflex (VOR). One hundred and thirty-one patients with migraine [65 with vestibular migraine (MV), 41 with migrainous dizziness (MD), and 25 with migraine only (MO)] and 50 normal controls underwent evaluation of vestibular function. Motion sickness was assessed using the motion sickness susceptibility questionnaire (MSSQ) and subjective scale. Compared with normal controls and MO group, patients with MV/MD showed increased VOR time constant (TC) and greater suppression of the post-rotatory nystagmus with forward

head tilt. The mean MSSQ score and subjective scale were highest in MV group, followed by MD, MO, and controls ($p = 0.002$, $p < 0.001$). Multiple linear regression model analyses revealed that motion sickness is an independent factor of TC prolongation ($p = 0.024$). Twenty-eight (21.4%) patients with migraine also showed perverted head shaking nystagmus and 12 (9.2%) had positional nystagmus. In view of the increased tilt suppression of the VOR, we speculate that dysfunction of the nodulus/uvula may not account for the prolonged TCs in MD/MV. Instead, innate hypersensitivity of the vestibular system may be an underlying mechanism of motion sickness and increased TC in MD/MV. The increased tilt suppression may be an adaptive cerebellar mechanism to suppress the hyperactive vestibular system in migraineurs.

Keywords Migraine · Vertigo · Motion sickness

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Introduction

In migraine, headache frequently coexists with symptoms of vestibular dysfunction, which include dizziness/vertigo, motion sickness and gait instability [1–5]. However, the mechanisms of migraine-related vestibular symptoms remain unknown. Patients with migraine-related dizziness often show directional preponderance during caloric stimulation and rotational testing [6], excessive postural sway in challenging circumstances [7], and a higher prevalence of peripheral vestibular dysfunction than would be expected in patients with purely central vestibular abnormalities [8, 9]. A recent report on migraine-related dizziness also found reduced semicircular canal-ocular reflex gain, a slightly larger modulation component of the otolith-ocular reflex during constant velocity off-vertical axis rotation

(OVAR), and increased postural sway during optic flow stimulation [10].

Migraineurs are more likely to suffer from motion sickness following milder vestibular stimulation [11]. Since motion sickness per se may affect the results of vestibular function tests, the abnormal vestibular findings in migraine-related dizziness should be interpreted in the context of co-morbid motion sickness [12, 13]. For example, compared with normal controls, the motion sickness group may show increased gain, decreased phase lead, and increased or decreased time constant (TC) of the vestibulo-ocular reflex (VOR) [12, 13]. However, previous studies did not attempt to analyze migraine-related vestibular dysfunction according to co-morbid dizziness/vertigo or motion sickness, and did not correlate the vestibular dysfunction with severity of motion sickness using quantitative assessment. In view of the recent reports on the relationship between velocity storage and motion sickness [14–16], we measured vestibular TC in patients with migraine according to the presence of dizziness/vertigo and motion sickness. When the head is pitched forward at the onset of post-rotatory nystagmus, the time constant of the VOR is diminished [17]. This tilt suppression of the VOR is mediated by the cerebellar nodulus and ventral uvula, and is eliminated after surgical ablation of those structures in animals [17] and in patients with midline cerebellar lesions [18]. Furthermore, the nodulus and uvula control the orientation vectors, TC, and habituation of the VOR [17, 19], all of which are known as critical factors in producing motion sickness [15–17]. Accordingly, we also evaluated tilt suppression of the post-rotatory nystagmus to determine whether impaired uvulonodular inhibition over the velocity storage is a mechanism of vestibular dysfunction in migraine and motion sickness. Our hypothesis is that impaired cerebellar inhibition over the VOR may underlie the vestibular hypersensitivity in migraine, which may explain the frequent association of vestibular symptoms and motion sickness in this disorder.

Methods

Subjects

At the Dizziness Clinic of Seoul National University Bundang Hospital, 131 consecutive patients with migraine were recruited from March to November 2006. The patients were classified into 65 (49.6%, 63 women) migrainous vertigo (MV), 41 (31.3%, 38 women) migrainous dizziness (MD), and 25 (19.1%, 18 women) migraine only (MO) [3]. MV was defined when the patients met the definite and probable diagnostic criteria proposed by Neuhauser et al. [3]. The MD group included the patients with dizziness/

vertigo which cannot be explained by diseases other than migraine but did not meet the diagnostic criteria of MV. Patients in the MO group did not report dizziness/vertigo. Subjects with migraine were also required to report active migraine, defined as at least two migraine episodes during the previous 12 months. Fifty healthy subjects (27 women, age range = 20–70, mean age \pm SD = 39.9 \pm 14.4) were recruited for normal controls from the local community after excluding those with a history of migraine, dizziness/vertigo, or other neurological disorders.

This study was performed in accordance with the declaration of Helsinki and was approved by the Committee for Medical Ethics of Seoul National University Bundang Hospital. Informed consent was obtained from each patient.

Evaluation of dizziness/vertigo and motion sickness

The degree of dizziness/vertigo and motion sickness (single-item motion sickness susceptibility questions) was classified as “0 = not at all”, “1 = slightly”, “2 = moderately”, and “3 = very much so”. The motion sickness group included the participants who responded as 1–3. The severity of motion sickness was also assessed using the motion sickness susceptibility questionnaire (MSSQ), which is a sum of total sickness score during childhood (<12 years of age, MSSQ-A) and during adulthood over the last 10 years (MSSQ-B) [20].

Neurotologic evaluations

During the headache-free period, patients underwent oculography, bithermal caloric tests, and the rotatory chair test. Further investigations such as MRI and laboratory tests were performed when considered appropriate.

Oculography

Nystagmus was recorded with a video-oculography system (SMI, Teltow, Germany) [21]. Spontaneous nystagmus with/without fixation and gaze-evoked nystagmus in the horizontal ($\pm 30^\circ$) and vertical ($\pm 20^\circ$) planes were recorded.

Post-head-shaking nystagmus (HSN) was induced using a passive head-shaking maneuver. After grasping the participant's head firmly on both sides with the head pitched forward by approximately 30° , the head was shaken horizontally in a sinusoidal fashion at a rate of 2.8 Hz with approximate amplitude of $\pm 10^\circ$ for 15 s. Detailed methods and normative data have been described previously [21, 22].

To induce positional nystagmus, patients laid supine from sitting and turned their heads to either side while in

supine. Then patients were moved from a supine to a sitting position and the head was bent down [23]. Patients were also subjected to right and left Dix-Hallpike maneuvers and the straight head hanging test [24].

Bithermal caloric tests

Seventy (53.4%) patients underwent binaural alternate irrigation for 25 s with 50 ml of cold and hot water (30 and 44°C) for caloric tests [18]. Asymmetry of vestibular function was calculated using Jongkees' formula and caloric paresis was defined by the response difference of 25% or more between the ears [18]. To determine caloric hyper-responsiveness, average peak slow phase velocity (SPV) was calculated from the peak SPV obtained under the four different stimulating conditions. The performance of caloric tests in only 70 patients was mainly due to the limited availability of the caloric tests in our laboratory and was not affected by the severity of the symptoms. Indeed, there were no differences in sex ratio, severity of motion sickness, and proportion of each migraine group between the patients with and without caloric tests.

Rotatory chair test

Horizontal VOR was measured by using the CHARTR[®] rotary vestibular test system (ICS Medical, IL, USA). For evaluation of VOR, the subjects underwent sinusoidal rotation about a vertical axis at harmonic frequencies ranging from 0.01 to 0.32 Hz with a peak angular velocity of 50°/s at each frequency. The patient's head was pitched forward 30° from the vertical plane during VOR testing. The velocity of eye movement elicited was compared with the velocity of the stimulus (assumed to be the same as head velocity). The resultant velocity curve was then analyzed using Fourier analysis. At each frequency phase shift, gain, and asymmetry were measured. To induce visually-enhanced VOR (VVOR) during sinusoidal oscillation (frequency = 0.04 Hz, peak angular velocity = 50°/s), the patients were instructed to see a whole-field optokinetic stimulation with alternating black and white stripes.

For the velocity-step test, patients were subjected to a series of velocity steps, both to the right and to the left. For a leftward velocity step, the subjects underwent an angular acceleration of 100°/s² for 1 s, rotation to the left for 60 s at a constant velocity of 100°/s, and deceleration to 0°/s within 1 s. The above steps were repeated for a rightward rotation. In response to the stimuli, TCs of the per- and post-rotatory nystagmus were calculated. The nystagmus beats were measured and plotted using a SPV algorithm. The algorithm then used a Gauss–Newton method to calculate the best fit curve for the slow phase beats, fitting to the exponential formula: $V = Ae^{bt} + C$, where V means

velocity; A , a constant related to the magnitude of the response; b , the time constant of the system; t , time; e , a constant (the base of natural logarithms); and C , offset from the baseline (spontaneous nystagmus offset from zero). The eye motion was detected with electrodes and digitized at 160 Hz with a frequency response of 0–30 Hz.

To evaluate tilt suppression of the VOR, the patients were asked to pitch their heads forward at the end of the step rotation stimuli in each direction. The patients were monitored during the testing using an infrared camera, and the data from inadequate head tilt were excluded from analyses. TC was calculated again as described above. The effectiveness of tilt suppression of the post-rotatory nystagmus was expressed by the tilt suppression index (TSI) that was calculated by the equation $TSI (\%) = [(TC \text{ without head tilt} - TC \text{ with head tilt}) / TC \text{ without head tilt}] \times 100$. Normative data were obtained from 50 healthy volunteers (23 men, age range = 20–70 years, mean age = 39.9 ± 14.4).

Statistical analyses

Statistical analyses included χ^2 and Fisher's exact tests for dichotomous variables, and t test and one-way ANOVA (with Tukey B corrections for multiple comparisons) for continuous variables. To find out the independent factors contributing to prolongation of TC, we also performed a multiple linear regression analysis (Enter method, SPSS version 12.0) after the categorical variables for severity of dizziness and motion sickness were transformed into new parameters indicating the presence (score ≥ 1) or absence (score 0) of those symptoms. Results were considered significant at $p < 0.05$.

Results

Demographic characteristics of the patients

The mean age did not differ between the patients and normal controls (41.7 ± 12.3 vs. 39.9 ± 14.4 years, $p > 0.05$, t test). Compared with normal control, women were more common in the migraine group (90.8 vs. 54.0%, $p < 0.001$, χ^2 test). The duration of dizziness/vertigo varied from minutes to several days.

Motion sickness

The migraine group included more persons susceptible to motion sickness (subjective scale ≥ 1) than the normal controls (88.5 vs. 45.0%, $p < 0.001$, χ^2 test). The mean MSSQ score (Fig. 1a) and subjective scales (Fig. 1b) were highest in the MV group, followed by MD, MO and the

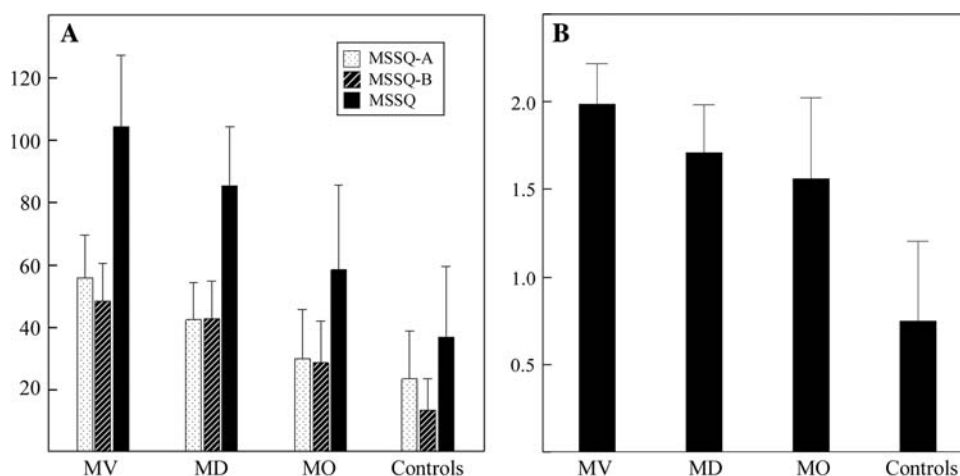


Fig. 1 The motion sickness susceptibility questionnaire (MSSQ) scores (a) and subjective scale (b) are highest in the MV group, followed by MD, MO and the control group (MSSQ, $p = 0.002$; MSSQ-A, $p = 0.013$; MSSQ-B, $p = 0.005$; subjective scale, $p < 0.001$, one-way ANOVA with Tukey B corrections for multiple

comparisons). MSSQ-A is concerned with childhood experiences of travel and motion sickness before the age 12 while MSSQ-B is related to experiences of travel and motion sickness over the last 10 years. MSSQ is the sum of MSSQ-A and MSSQ-B [20]. MV migrainous vertigo, MD migrainous dizziness, MO migraine only

control group. These differences were also observed in MSSQ-A and MSSQ-B (Fig. 1a). Furthermore, patients with MV/MD were more common in the motion sickness group than in the group without motion sickness (84.5 vs. 53.3%, $p = 0.009$, χ^2 test).

Oculography

Three patients showed spontaneous nystagmus which was right beating ($2.3^\circ/s$) in one with MV and downbeating (2.8 and $3.3^\circ/s$) in two with MD. No one showed gaze-evoked nystagmus.

HSN was observed in 46 patients (34.1%); 21 (32.0%) with MV, 18 (43.0%) with MD, and 7 (25.0%) with MO (χ^2 test, $p = 0.390$). HSN was horizontal ($n = 18$), vertical ($n = 18$), both horizontal and vertical ($n = 8$), or mixed horizontal, vertical and torsional ($n = 2$). The direction of vertical HSN was downbeat in all the patients. Overall, 28 (21.4%) patients with migraine showed perverted HSN. Twelve patients showed positional nystagmus, which was horizontal ($n = 7$), downbeat ($n = 2$), upbeat ($n = 2$), and mixed horizontal and downbeat ($n = 1$) during various positioning maneuvers.

Caloric test

Caloric responses were normal without caloric paresis or directional preponderance in all the 70 patients tested. However, eight (11.4%) of them (7 with MV, 1 with MD, and none with MO) showed increased response (average slow peak velocity $> 41.3^\circ/s$), based on our normal data

(mean \pm SD = $25.1 \pm 8.1^\circ/s$). There was no difference in the proportion of patients with caloric hyper-responsiveness between the groups with and without increased VOR TCs (22.2 vs. 9.8%, $p = 0.272$), and between those with and without pHSN (7.1 vs. 12.5%, $p = 1.000$).

Rotation test

Compared with normal controls, the migraineurs (MV, MD and MO) exhibited increased VOR gain only at 0.02 Hz of stimulation while the phase did not differ among the groups (Fig. 2a). The gains and phases of the VOR and VVOR did not differ among the groups, either. In contrast, the VOR TCs were increased in MV/MD, compared with those in MO and normal controls (Fig. 3a). However, tilt suppression of the VOR was greater in MV/MD (Fig. 3a).

The patients with motion sickness showed higher VOR gains at 0.02, 0.04, and 0.16 Hz rotations than those without motion sickness while the phases of the VOR did not differ between the groups (Fig. 2b). The VOR TCs were also increased in the motion sickness group while the tilt suppression of the VOR did not differ between the groups (Fig. 3b). Furthermore, the increase of TCs in the motion sickness group was in proportion to the severity of motion sickness (subjective scale) even though the correlation coefficient was small (Pearson $r = 0.2$, $p = 0.004$, Fig. 4).

The patients with perverted HSN also exhibited increased VOR gains at 0.02, 0.04, and 0.16 Hz rotations without difference in the VOR phases, compared with

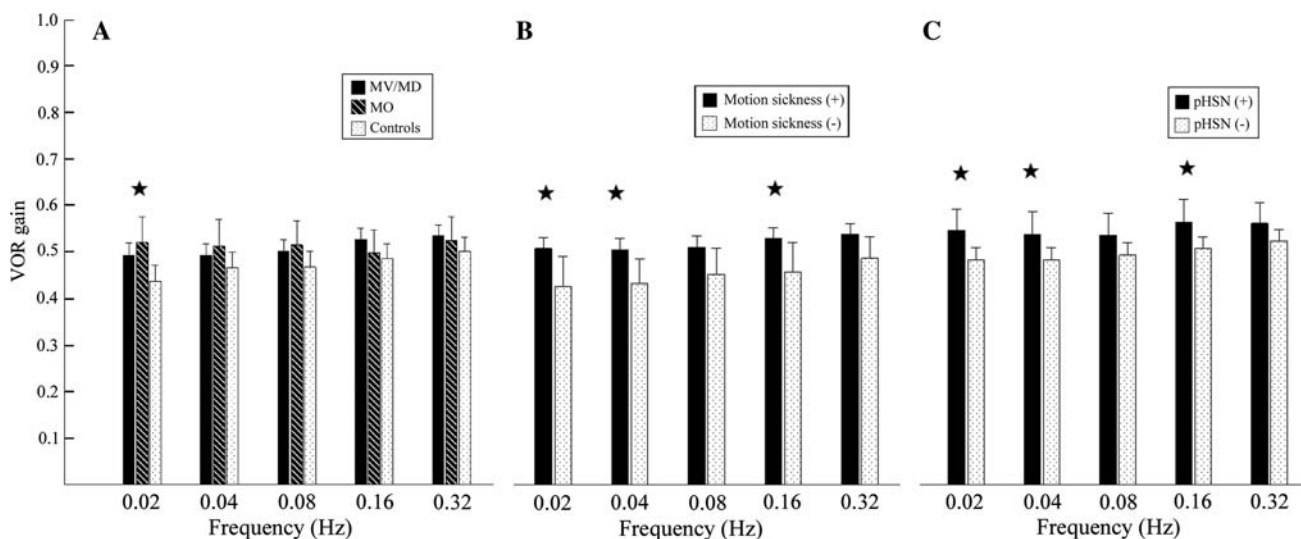


Fig. 2 a Compared with normal controls, the migraineurs (MV, MD, and MO) exhibit increased gain of the vestibulo-ocular reflex (VOR) gain only at 0.02 Hz of stimulation ($p < 0.05$, one-way ANOVA with Tukey B corrections for multiple comparisons). **b** The VOR gains are higher at 0.02, 0.04, and 0.16 Hz rotations in patients with motion

sickness than those without motion sickness ($p < 0.05$, t test). **c** The patients with perverted head-shaking nystagmus (HSN) also show increased VOR gains at 0.02, 0.04, and 0.16 Hz rotations, compared with those without perverted HSN ($p < 0.05$, t test). *MV* migrainous vertigo, *MD* migrainous dizziness, *MO* migraine only. ★ $p < 0.05$

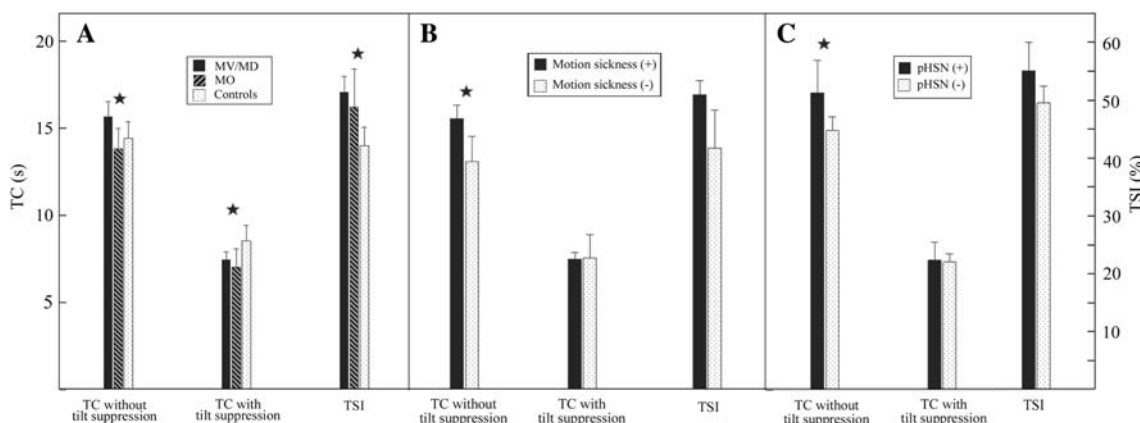


Fig. 3 a The time constants (TCs) of the vestibulo-ocular reflex (VOR) are increased in MV/MD, compared with those in MO and normal controls ($p < 0.05$, one-way ANOVA with Tukey B corrections for multiple comparisons) while tilt suppression of the VOR is greater in MV/MD ($p < 0.05$, one-way ANOVA with Tukey B corrections for multiple comparisons). **b** The VOR TC is also increased in the motion sickness group ($p = 0.024$, t test) while the

tilt suppression of the VOR does not differ between the groups ($p > 0.05$, t test). **c** The VOR TC is also greater in migraineurs with perverted head-shaking nystagmus (HSN) than in those without perverted HSN ($p = 0.019$, t test). In contrast, tilt suppression of the VOR does not differ between the groups ($p > 0.05$, t test). *MV* migrainous vertigo, *MD* migrainous dizziness, *MO* migraine only, *TSI* tilt suppression index. ★ $p < 0.05$

those without perverted HSN (Fig. 2c). TCs were also greater in migraineurs with perverted HSN than in those without perverted HSN (Fig. 3c). However, tilt suppression of the VOR did not differ between the groups (Fig. 3c).

In the patients and normal controls, multiple linear regression analyses including age, sex, and the presence of migraine, dizziness/vertigo and motion sickness as covariates, revealed that motion sickness was the only independent factor for prolongation of TCs in migraineurs (Enter method, $R^2 = 0.046$, Table 1). The presence of

migraine itself was not an independent predictor of TC prolongation (Table 1).

Discussion

Our study showed that migraineurs with dizziness/vertigo suffer from more severe motion sickness than those without dizziness/vertigo and normal controls. Also, the VOR TCs were increased in migraineurs with dizziness/vertigo while

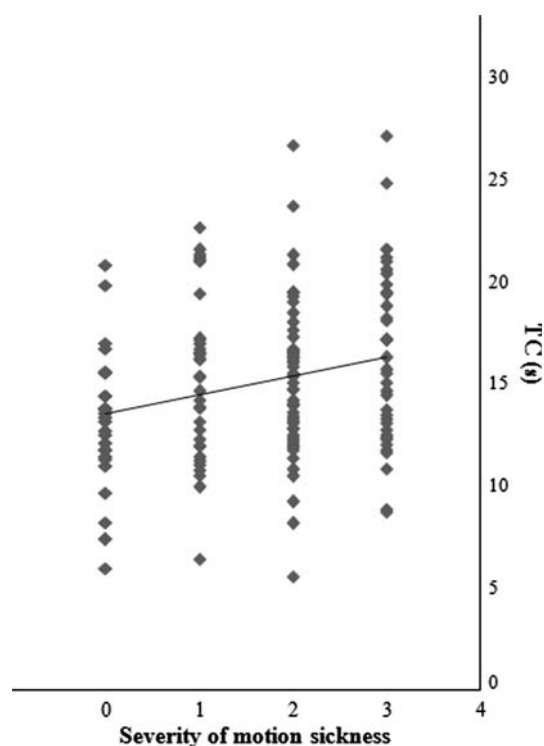


Fig. 4 A scatter plot with a regression line shows a mild increase of the time constants (TCs) of the vestibulo-ocular reflex (VOR) in proportion to the severity of motion sickness (subjective scale) in the motion sickness group (Pearson $r = 0.2$, $p = 0.004$)

Table 1 Linear regression coefficients for the time constants of the vestibulo-ocular reflex

Variable	Regression Coefficient		p value
	B	Standard Error	
Constant	12.28	1.55	
Motion sickness	2.25	0.99	0.024
Sex	-0.53	1.07	0.620
Age	0.03	0.03	0.366
Migraine	-0.72	1.28	0.575
Dizziness	-0.72	1.28	0.575

$R^2 = 0.046$

B indicates the amount of time constant increase when the specific variable rises by one unit while the other variables are kept constant. The bold part indicates the only variable with statistical significance

the tilt suppression of the VOR was greater in these patients. Furthermore, the patients with motion sickness exhibited increased VOR gains and TCs in the presence of preserved tilt suppression of the VOR.

Vestibular dysfunction in migraine

Compared with normal controls, the migraineurs exhibited increased VOR gain only at 0.02 Hz of stimulation. In

contrast, the VVOR gain was decreased only in patients with MO while the phase was similar. In a previous study, the VVOR gain was increased in migraineurs, which was ascribed to hypersensitivity of the neurological integration of visual and vestibular stimuli in migraineurs [25]. However, another study reported reduced angular VOR gain and preserved visual-vestibular interaction in patients with migraine-related dizziness [10]. The reason for those contradictory results is unclear. However, migraineurs may show different VOR or VVOR gain according to the presence of associated dizziness/vertigo and motion sickness or across the stimulation frequency ranges.

Our study showed greater TCs of the VOR and more severe motion sickness in MV/MD than in MO and normal controls. However, in view of the increased tilt suppression of the VOR in MD/MV groups, dysfunction of the nodulus/uvula may not account for the increased TCs and motion sickness in these groups. Instead, innate hypersensitivity of the vestibular system may be an underlying mechanism of motion sickness in MD/MV groups. The enhanced tilt suppression of the VOR in patients with dizziness/vertigo may be a cerebellar adaptive mechanism to suppress the hypersensitive vestibular system in these patients.

In our study, 28 (21.4%) of the migraineurs showed perverted HSN. Previously, perverted HSN has been reported in diffuse cerebellar degeneration and focal medullary [26] or cerebellar lesions [27]. Perverted HSN may occur from inappropriate storage of vestibular signals in the vertical plane by charging through the pathways from the horizontal canal (cross-coupling) [28]. These cross-coupled responses are usually attributed to lesions in central vestibular pathways including the vestibulocerebellum [27], but the precise localization remains to be elucidated. To the best of our knowledge, no study has explored HSN in migraineurs. In our study, the migraineurs with perverted HSN showed increased VOR gains and TCs in the presence of normal tilt suppression. In view of the preserved tilt suppression of the VOR, dysfunction of the nodulus/uvula may not account for the increased VOR gains and TCs in the perverted HSN group. Instead, central vestibular pathways in the brainstem or other parts of the cerebellum may be involved in the generation of perverted HSN in migraineurs.

Patients with migrainous vertigo may show spontaneous, gaze-evoked, and positional nystagmus, impaired smooth pursuit, or disturbed stance and gait during the symptomatic phase [29]. Some of our patients also showed spontaneous and positional nystagmus, and HSN even during the interictal phase, which all suggest imbalances in the peripheral or central vestibular system.

A previous study showed enhanced caloric responses only in two of 80 patients with migraine [1]. However, the caloric responses were increased in 11.4% of our patients,

which also indicates increased vestibular responses in migraineurs [6]. Since the caloric tests were performed in only 53.4% of the patients, the possible selection bias might have resulted in the discrepancy. However, the selection was mainly based on the availability of the caloric tests during the evaluation in our laboratory, and was not affected by the severity of the patients' symptoms. Indeed, there were no differences in the sex ratio, severity of motion sickness, and proportion of MV/MD/MO groups between the patients with and without caloric tests. Accordingly, we do not believe that the result was skewed by the selection.

Vestibular dysfunction in motion sickness

The vestibular system plays a critical role in producing motion sickness since subjects without peripheral vestibular function are immune to stimuli that cause motion sickness [30–32]. The cerebellar nodulus and uvula are responsible for the orientation properties of the velocity storage [19, 33, 34] and destruction of the nodulus and uvula substantially reduces or abolishes motion sickness susceptibility in dogs [35, 36]. Previously, some individuals with motion sickness also showed markedly increased VOR TC during step velocity rotation [13]. Recently, another study demonstrated an inverse relationship between the VOR TC and the number of head movements enough to develop motion sickness [16]. Furthermore, the severity of motion sickness was associated with the extent, strength, and duration of eye velocity deviation from the gravity during the rotation. These findings support that the spatiotemporal properties of the velocity storage, which are processed through the nodulus/uvula and vestibular nuclei, are likely to represent the source of conflict responsible for motion sickness [16].

In the motion sickness group, TCs of the peri- and post-rotatory nystagmus were also increased during step velocity rotation in this study and the increase was in proportion to the severity of motion sickness even though the correlation coefficient was small. These results also suggest that the individuals with increased post-rotatory TCs would be more susceptible to motion sickness. Furthermore, the VOR gains were increased in the migraineurs with motion sickness. These findings are consistent with the results of previous studies which showed that the persons susceptible to motion sickness had higher than normal angular VOR gains in response to angular acceleration of $90^\circ/\text{s}^2$ [15], and to the rotations in the low frequency ranges (<0.08 Hz) [12, 37]. However, previous studies debated on the increase of VOR gain during high frequency stimulation in those with motion sickness [12, 16, 38, 39].

In a recent study of patients with suspected bilateral vestibulopathy, the patients with low VOR gain and

relatively preserved TCs showed a strong inverse relationship between the TCs and susceptibility to motion sickness, but none between angular VOR gains and motion sickness susceptibility [40]. These findings suggest that motion sickness may develop through the velocity storage mechanism rather than the direct pathway, and reduced VOR TCs may decrease the susceptibility to motion sickness [40].

Previously, subjects highly susceptible to sea sickness showed VOR responses with significantly lower phase leads at 0.01–0.08 Hz than the non-susceptible ones [12]. However, the phase of VOR did not differ between the groups in our study.

Migraine and motion sickness

Motion sickness is also more frequent in adult migraineurs than in normal population. According to a previous study, 50.7% of 200 unselected migrainous subjects suffered from motion sickness [6]. In our study, the mean MSSQ score and subjective motion sickness scale were highest in MV, followed by MD, MO, and the control group. Furthermore, regression analysis showed that only the motion sickness was an independent factor for the prolongation of VOR TCs. This result indicates that the increased TCs in MV/MD are related to motion sickness susceptibility of the migraineurs, and not to migraine itself or associated dizziness/vertigo.

In our study, the control group included more men (46.0%) than the patients group (9.2%). Such a large difference in the sex distribution may have affected the differences observed in the VOR parameters and severity of motion sickness between the patients and the controls. However, the gain, phase, TC, and tilt suppression of the VOR did not differ between women and men in the controls. In view of the previous studies that reported higher rate of motion sickness in women [35], inclusion of more women in the patient group may have resulted in a higher incidence of motion sickness in the patients. However, no difference in the motion sickness susceptibility between the MO group and the controls does not support this presumption. Furthermore, statistical analysis showed no difference in the subjective scale and MSSQ between women and men in our controls without history of migraine and vertigo/dizziness. Indeed, sex or presence of migraine itself was not an independent predictor of TC prolongation in the logistic regression analyses.

Our study disclosed increased VOR TCs and motion sickness susceptibility in MD/MV groups, compared with MO and normal control groups. In view of the increased tilt suppression of the VOR, we speculate that dysfunction of the nodulus and uvula may not account for prolonged TCs in MD/MV. Instead, innate hypersensitivity of the vestibular

system may be an underlying mechanism of motion sickness and increased TCs in MD/MV. The increased tilt suppression in these patients may be an adaptive cerebellar mechanism to suppress the hypersensitive VOR system.

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Conflict of interest statement The authors report no conflicts of interest.

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