Convergence Vestibulo-ocular Reflex in Unilateral Vestibular Hypofunction: Behavioral Evidence in Support of a Novel Gaze Stability Exercise

Tzu-Pu Chang, MD, and Michael C. Schubert, PT, PhD, FAPTA

Background and Purpose: Convergence of the eyes during head rotation increases the gain (eye velocity/head velocity) of the vestibulo-ocular reflex (VOR). We sought to know whether convergence would increase the VOR gain (mean + SD) in unilateral vestibular hypofunction (UVH).

Methods: Vestibulo-ocular reflex gain during ipsi- and contralesional horizontal head rotation at near (15 cm) and far (150 cm) targets was measured in 22 subjects with UVH and 12 healthy controls. Retinal slip was estimated (retinal slip index [RSI]) as the difference between ideal VOR gain (no retinal slip) and the actual VOR gain.

Results: Convergence did not significantly enhance VOR gain for ipsilesional rotation (mean difference, 0.04; 95% confidence interval [CI], −0.01 to 0.09), near viewing (0.77 ± 0.34) versus far viewing (0.72 ± 0.29), yet the VOR gain during contralesional rotation was greater for near viewing (1.20 ± 0.23) than for far viewing (0.97 ± 0.21; mean difference, 0.23; 95% CI, 0.13-0.32). In the 36% of subjects with recovery of their ipsilesional VOR gain, the vergence effect trended to recover (far VOR gain: 1.06 ± 0.17 vs near VOR gain 1.16 ± 0.21; mean difference, 0.10; 95% CI, −0.02 to 0.22). Ipsilesional head rotation induced greater retinal slip for near (RSI = 0.90 ± 0.34) targets than for far targets (RSI = 0.35 ± 0.29; mean difference, 0.56; 95% CI, 0.51-0.61).

Discussion and Conclusions: The convergence-mediated VOR gain enhancement is preserved during contralesional but impaired during ipsilesional head rotation. Recovery of ipsilesional passive VOR gain does not equate to restored convergence enhancement, although it did increase ∼10%. These data suggest head motion viewing near targets will increase retinal slip, which warrants consideration as a gaze stability exercise for subjects with UVH.

Video Abstract available for more insights from the authors (see Video, Supplemental Digital Content 1, available at: http://links.lww.com/JNPT/A325).

Key words: *convergence, gaze stability, vestibular hypofunction, video head impulse test, VOR*

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INTRODUCTION

Dizziness is a common health care problem with a prevalence in the general population of 15.8%.[1] Peripheral vestibulopathy is one of the most common etiologies of dizziness.[2,3] In addition to causing vertigo and imbalance, it impairs dynamic visual acuity, resulting in blurred vision or oscillopsia during head rotation—all of which further increase fall risk.[4]

Vestibular rehabilitation is an effective means to improve the imbalance, vertigo, fall risk, and gaze stability in subjects with vestibular pathology.[5-7] The unique contributions of vestibular rehabilitation for treating vestibular hypofunction are the gaze stability exercises (or vestibulo-ocular reflex [VOR] exercise), which improve dynamic visual acuity and reduce fall risk.[7-9] The putative mechanism for the beneficial effect of gaze stability exercises is retinal slip. Retinal slip occurs when images move on the fovea of retina during head motion. This creates error, which mediates adaptation of the gain (eye velocity/head velocity) of the VOR and/or recruitment of a unique type of compensatory saccade.[10-12] Typically, the target viewing distance for gaze stability exercises is set at a distance that does not require much vergence (eg, to tape a card onto the wall 6-10 ft in front of the person, or hold a letter at arm's length).[13] However, it is unknown whether performing such exercises might benefit from near viewing that recruits the vergence-mediated VOR gain enhancement.

When we view a target within 15 cm and turn our heads, the rotation angle of the eyes must be greater than the rotation angle of the head to maintain focus on the target.[14] The increased rotation angle demands a greater eye velocity. Therefore, in healthy subjects, the VOR gain is enhanced when the eyes converge to view a near target, called "vergence-mediated VOR gain enhancement."[15] The vergence-mediated VOR gain enhancement is impaired in subjects with unilateral vestibular

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loss due to intratympanic gentamicin injection,[16] but few studies have examined this response in other etiologies causing unilateral vestibular hypofunction (UVH). We are interested in developing a vergence-enhanced VOR gaze stability exercise that may be useful for subjects with UVH. To determine whether this might be a useful exercise, we first desire to know the near- and far-viewing VOR gains among subjects with different, yet common causes for their UVH. If the enhancement of near-viewing VOR gain is similarly impaired in common vestibular disorders such as vestibular neuritis, then the additional retinal slip engendered through near viewing may warrant the development of a vergence VOR gaze stability exercise.

METHODS

Study Population

Twenty-two consecutive subjects with UVH and 12 healthy controls were recruited from the dizziness clinic within the Department of Neurology at Taichung Tzu Chi Hospital in 2019. Each subject underwent a comprehensive history, neurological and neuro-otological examination including videonystagmography, video head impulse test (vHIT), and brain magnetic resonance imaging. Three of the patient subjects also received caloric examination, which was abnormal. Unilateral vestibular hypofunction was defined as (i) horizontal semicircular canal VOR gain less 0.8 during vHIT testing, or (ii) unilateral weakness of 25% asymmetry or greater per the bithermal caloric examination. The patient subjects with histories of cervical spine disorders, vertebral or carotid artery dissection, disturbance of static visual acuity, peripheral or central ocular motor palsy, strabismus, or cognitive impairment were excluded. Healthy controls had normal vestibular function.

This study was performed in accordance with the guidelines of the 1964 Declaration of Helsinki and was approved by the Institutional Review Board of the Research Ethics Committee of Tzu Chi Medical Center (REC-107-22). Written consent was obtained from each person prior to the study.

Data Recording System

Head and oculomotor recordings were collected using a video-oculography (VOG) system (Middelfart, Denmark) with a 220-Hz infrared video camera set in front of right eye. The VOG method was used to collect the vHIT VOR gain as well as the eye position during clinical ocular alignment testing (cover/uncover test).

Study Protocol

Each subject underwent the cover/uncover and alternate cover tests for both near and far viewing, as well as vHIT during both near and far viewing. Initially, each subject was seated 150 cm in front of a featureless wall. The head and eye position was calibrated per manufacturer's recommendation (EyeSeeCam; Middelfart, Denmark). Next, subjects viewed a pink dot with diameter of 2 mm placed at the eye level on the wall in front of the person's right eye.

Cover Tests

The far-viewing cover tests (150 cm) were performed to evaluate heterophorias and heterotropias, identified whenever the eye was noted to move into its rest position. The near-viewing cover tests (15 cm) were performed to evaluate whether the subjects converged well when viewing the near target. First, each eye was assessed for its ability to maintain convergence, which was recorded using the VOG. Next, the cover test was applied to the right eye for 3 seconds, before removing the cover for 3 seconds. This was repeated 4 times (total 30 seconds). The examiner used the same protocol for the left eye cover test. For alternate cover testing, the examiner covered the subject's eyes alternatively using a paddle. The right eye was first covered for 3 seconds, and then the left eye was covered for 3 seconds. This cycle was repeated 4 times, each lasting 30 seconds. The eye position was recorded via VOG to identify any corrective horizontal eye movement.

Far- and Near-Viewing VOR Assessment via vHIT

For far-viewing vHIT, each subject sat 150 cm in front of the visual target positioned in the center of their field-of-view. A well-trained examiner quickly turned the subject's head in the horizontal plane with a small-amplitude rotation ($\sim 10^{\circ}$). Twenty horizontal head impulses were applied to both right and left sides with random order and interval. The VOR gains were automatically recorded using the EyeSeeCam software, which determined the regression slope between the eye and head velocity. For near-viewing vHIT, the same subject was seated such that the 15-cm near target was positioned in front of the recorded eye (ie, right eye) to ensure optimal pupil tracking from the camera. The vHIT protocol was repeated. We asked each subject about diplopia to ensure that vergence was not being lost during vHIT testing. The head impulse is a high-acceleration ($\sim$2500 d/s/s), moderate-velocity ($\sim$200 d/s), and small-amplitude ($\sim 10^{\circ}$) head rotation that excites vestibular afferents on the same side of head rotation, while inhibiting the contra-rotational afferents to zero.[17] Therefore, ipsilesional VOR gain refers to vestibular function from the affected ear, while contralesional VOR gain refers to vestibular function from the healthy ear.

Estimation of Retinal Slip

Ideally, when a subject rotates his or her head while fixating a distant target, retinal slip should be zero, which will occur only when the rotation of the eye(s) in the orbit equals the rotation of the head (Ø in Figure 1A). When fixating on a near target, however, an additional rotation of the eye (θ in Figure 1A) is required to compensate for the head rotation and thus the ideal near-viewing VOR gain is greater than 1.0 in order to keep optimal fixation.[14] This occurs because the eyes are not located at the axis of head rotation but instead positioned approximately 10 mm in front of the axis of head rotation, which demands a translational eye rotation as well.[16]

Accordingly, during the small-amplitude head rotation such as occurs with head impulse testing, the ideal far-viewing VOR gain is 1.07 at our far-target distance of 150 cm. The ideal near-viewing VOR gain at our near-target distance of 15 cm is 1.67.

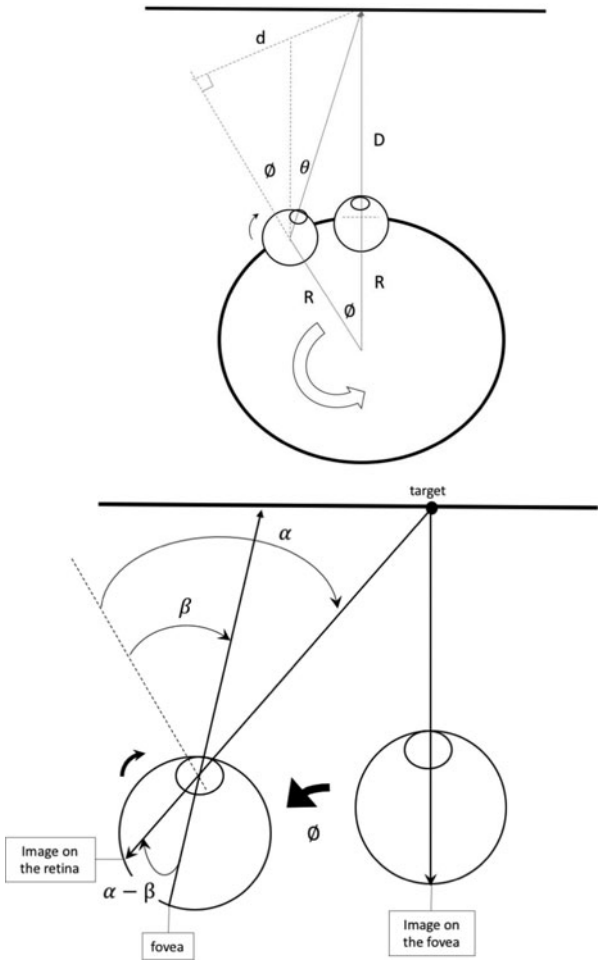

Figure 1. (A) Simplified geometry of the ideal eye movement required for an ideal near-viewing vestibulo-ocular reflex (R, the radius of head rotation; D, the distance between visual target and eye; $\emptyset$, the angle of head rotation; $\emptyset + \theta$, the ideal angle of eye rotation during head rotation). (B) Simplified geometry of the angle of retinal slip during a vestibulo-ocular reflex. When the actual eye velocity during head rotation does not reach the ideal eye velocity, the image on the retina deviates from the fovea and retinal slip develops ($\emptyset$, the angle of head rotation; α, the ideal angle of eye rotation during head rotation; β, the actual angle of eye rotation during head rotation; $\alpha - \beta$, the angle of retinal slip during head rotation).

To estimate the severity of retinal slip that occurs during head rotation at near- and far-target distances in this study, we defined a retinal slip index (RSI) as the difference between ideal VOR gain and actual VOR gain:

$$\text{Retinal slip index} = \text{Ideal VOR gain} - \text{Actual VOR gain}$$

The higher the RSI, the greater the magnitude of retinal slip. For example, an RSI = 0 implies that the actual VOR gain is equal to the ideal VOR gain and thus no retinal slip exists. There exists an inverse relationship between RSI and VOR gain in both healthy controls and patients with a pathologically reduced VOR. If the actual VOR gain does not increase as the target distance reduces, then the actual eye velocity is lower than the ideal eye velocity during the head rotation and retinal slip increases (Figure 1B).

Statistical Analysis

Descriptive statistics were used to present the demographic data and the results of cover tests. The near-viewing VOR gains were compared with the far-viewing VOR gains in the lesion side and the healthy side, respectively, via a 2-factor (distance and side of rotation) repeated-measures analysis of variance (ANOVA). Similarly, the RSI was compared for near- and far-viewing conditions using a separate 2-factor repeated-measures ANOVA. The mean of the differences between groups with 95% confidence interval (CI) was calculated. *P* value less than 0.05 was considered statistically significant. The statistical analyses were done via SPSS (Version 23).

RESULTS

Demographic Data

Table 1 shows the demographic data. Among the 22 subjects with UVH, their mean age was 59.3 ± 12.6 (mean ± SD; range: 37-77) years; 16 subjects (71.7%) were male. The clinical diagnoses include vestibular neuritis (n = 17), labyrinthine infarction (eg, magnetic resonance imaging–confirmed anterior inferior cerebellar artery stroke with unilateral vestibulopathy, n = 2), vestibular schwannoma (n = 1), and unknown vestibulopathy (n = 2).

Ocular Alignment

None of the 22 subjects noted diplopia during the test. All the subjects converged to view the near target before near-viewing vHIT was collected, confirmed by the right eye deviating in abduction (exo) when the right eye was covered and fixating back to the original position when right eye was uncovered during alternate cover test and right eye cover test. The mean angle of the exodeviation was 10.0° (3.4°-31.4°). Each person's deviation angles were the same in alternate cover test and right eye cover test except 1 subject (ID No. 17), whose deviation angle in the right eye cover test was smaller than that in the alternate cover test (4.8° vs 11.5°). This person had an exophoria when viewing the far target (150 cm) identified by a 5.9° exodeviation of the right eye in the cover test and a 6.7° exodeviation of the same right eye in the alternate cover test. Each person's right eye was still in near viewing during the left eye cover test. The degree of the right eye deviation during near-viewing cover test was not correlated to the degree of near-viewing VOR gain enhancement ($r = -0.15$, $P = 0.50$, Pearson correlation coefficient).

VOR Gain During Near and Far Viewing

The raw data of far- and near-viewing VOR gains are shown in Table 1. Figure 2 demonstrates the typical changes of VOR and compensatory saccades between far and near viewing in a person with UVH (ID No. 10).

During the vHIT while viewing the far target (150 cm), the VOR gain in the healthy side was 0.97 ± 0.21 (mean ± 1 SD) and the VOR gain in the lesion side was 0.72 ± 0.29.

Table 1. Demographic Data

ID	Age	Gender	Diagnosis	Lesion Side	From Onset to Testing	Far-Viewing VOR Gain		Near-Viewing VOR Gain	
						Lesion Side	Healthy Side	Lesion Side	Healthy Side
1	55	M	VN	R	3 mo	0.57	0.63	0.51	1.18
2	47	M	Labyrinthine infarction	L	1 mo	1.14	1.03	1.33	1.56
3	40	F	VN	R	19 d	1.02	1.2	1.01	1.21
4	67	F	VN	R	2 mo	1.02	1.07	1.18	1.34
5	71	F	Vestibular schwannoma	R	1 y	0.42	0.51	0.49	1.32
6	49	M	VN	L	5 mo	0.81	0.9	0.95	1.1
7	68	M	VN	L	12 d	0.42	0.89	0.5	0.95
8	41	M	VN	L	10 d	0.37	0.79	0.33	0.86
9	37	M	VN	R	1 y	0.62	1.28	0.74	1.59
10	70	M	VN	L	9 mo	0.55	0.97	0.56	1.26
11	58	M	VN	R	2 mo	0.53	1.12	0.38	1.17
12	55	F	VN	R	4 d	0.98	1.06	1.11	1.21
13	73	M	VN	L	10 y	0.93	1.12	1.04	1.16
14	64	M	Unknown vestibulopathy[a]	L	14 y	0.47	0.78	0.47	0.85
15	60	M	VN	R	1 y	0.53	0.89	0.65	1.1
16	77	F	Unknown vestibulopathy	L	2 y	0.6	0.96	0.51	0.9
17	40	M	VN	R	22 d	0.5	0.69	0.55	0.82
18	73	M	VN	L	9 y	1.34	1.27	1.62	1.66
19	77	M	Labyrinthine infarction	R	3 mo	1.26	1.26	1.06	1.19
20	63	M	VN	L	2 mo	0.57	0.96	0.61	1.34
21	56	F	VN	R	17 d	0.67	0.93	0.71	1.18
22	64	M	VN	R	2 mo	0.56	1.06	0.53	1.38

Abbreviations: F, female; M, male; VN, vestibular neuritis; VOR, vestibulo-ocular reflex.
[a]The left vestibulopathy was associated with cerebellar atrophy.

During near viewing, the VOR gain increased to 1.20 ± 0.23 for contralesional rotation but was similar for ipsilesional rotations, 0.77 ± 0.34. There was a significant interaction between distance (far/near) and side (ipsi-/contralesional) ($P = 0.01$, 2-way repeated-measures ANOVA). The near-viewing VOR gains for contralesional rotation were greater than far-viewing VOR gains (mean difference, 0.23; 95% CI, 0.13-0.32). In contrast, impulses to the lesion side showed similar VOR gain between near- and far-target viewing (mean difference, 0.04; 95% CI, −0.01 to 0.09) (Figure 3A and 3B).

Of the 22 subjects, 8 showed recovery of their passive VOR gain (far-viewing VOR gain returned to ≥ 0.8) during vHIT testing. Their mean VOR gain during far-target viewing was 1.11 ± 0.13 for contralesional rotation and 1.06 ± 0.17 for ipsilesional rotation. The near-viewing VOR gain increased to 1.30 ± 0.20 (17%) for contralesional rotations and 1.16 ± 0.21 (9.4%) for ipsilesional rotations. The VOR gain during ipsilesional rotation showed a tendency toward vergence-mediated enhancement (mean difference, 0.10; 95% CI, −0.02 to 0.22; $P = 0.09$), though it was significant for contralesional rotation (mean difference, 0.19; 95% CI, 0.02-0.36; Figure 3C and 3D).

In the residual 14 subjects, the VOR did not recover during the passive vHIT testing. In this unrecovered group, the far-viewing VOR gain was 0.89 ± 0.20 in the healthy side and 0.53 ± 0.08 in the lesion side, and near-viewing VOR gain was 1.14 ± 0.23 in the healthy side and 0.54 ± 0.11 in the lesion side. This change in near-viewing VOR gain was significant ($P < 0.001$) only for contralesional rotations (mean difference, 0.25; 95% CI, 0.11-0.38) and not for ipsilesional rotations (mean difference, 0.01; 95% CI, −0.03 to 0.06) (Figure 3E and 3F).

In the healthy controls, the far-viewing VOR gain was 1.07 ± 0.09 for rightward impulses and 1.04 ± 0.15 for leftward impulses. The near-viewing VOR gain increased a mean 1.24 ± 0.15 for the rightward direction and 1.21 ± 0.17 for the leftward direction. The mean difference between near viewing and far viewing was 0.17 (95% CI, 0.08-0.26) for rightward and 0.18 (95% CI, 0.11-0.25) for leftward head rotation. The vergence-mediated enhancement was not affected by the side of rotation ($P = 0.95$, 2-way repeated-measures ANOVA).

Magnitude of Retinal Slip

Table 2 shows the RSI during far and near viewing. For all UVH subjects, the RSI increased as the target distance reduced (150-15 cm) for both contra and ipsilesional rotations (contralesional: mean difference, 0.38 [95% CI, 0.28-0.47]; ipsilesional: mean difference, 0.56 [95% CI, 0.51-0.61]). This implies that head rotation (particularly toward the lesion side) would induce greater retinal slip when the visual target is closer to the eyes. In the unrecovered group, the ipsilesional head rotation conveyed an RSI increase from 0.54 ± 0.08 at far viewing to 1.13 ± 0.11 at near viewing, with a mean difference up to 0.59 (95% CI, 0.54-0.63). In the healthy subjects, the RSI during rightward impulses was 0.003 ± 0.09 at far viewing and 0.43 ± 0.15 at near viewing (mean difference: 0.43; 95% CI, 0.34-0.52). During leftward impulses, the RSI was 0.03 ± 0.15 at far viewing and 0.46 ± 0.17 at near viewing (mean difference: 0.42; 95% CI, 0.35-0.50).

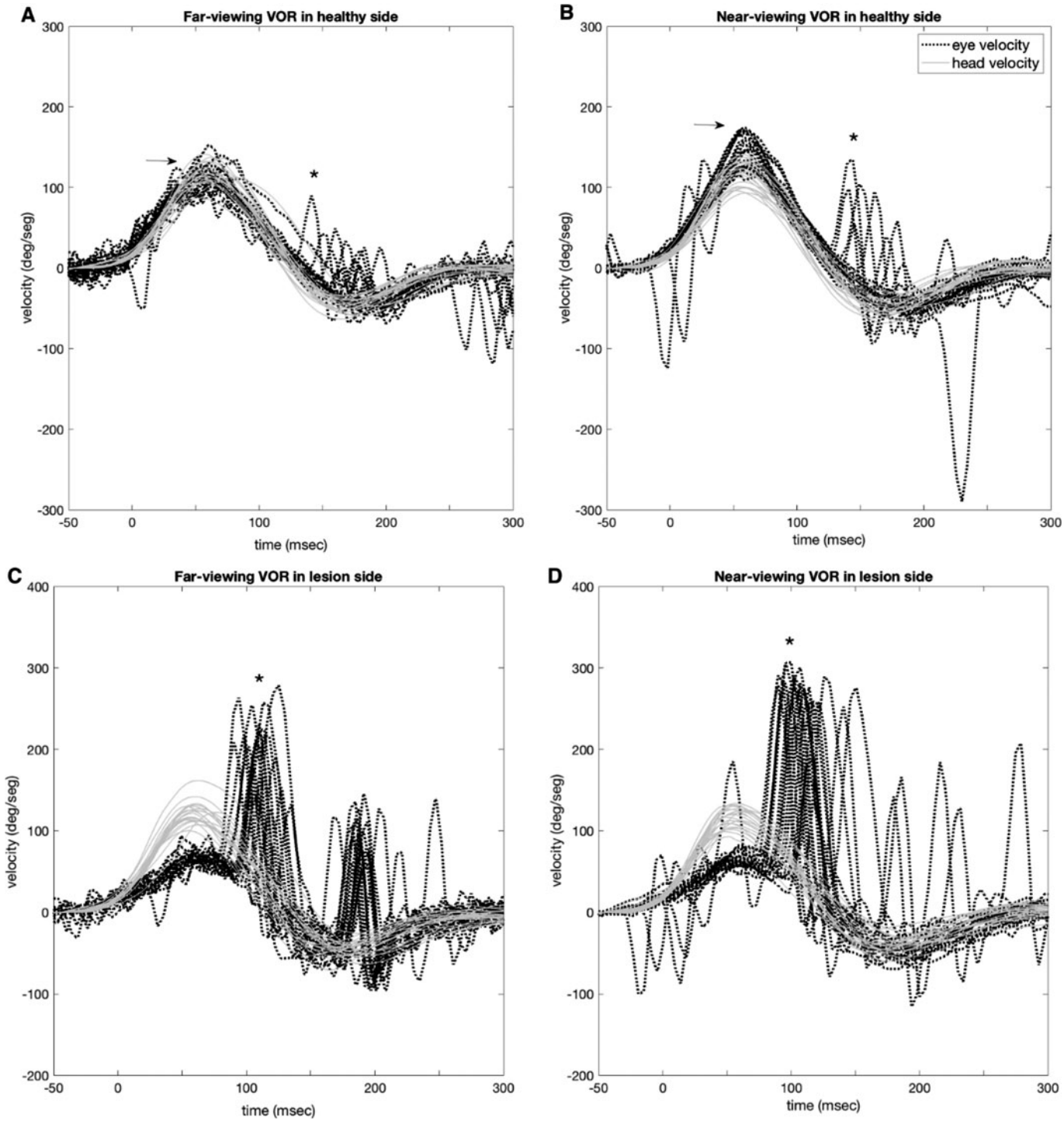

Figure 2. Video head impulse test during far and near viewing in a person with unilateral vestibular hypofunction. The peak eye velocity during head rotation to the healthy side is higher in near viewing than in far viewing (arrows in Figure 2A and 2B). Nevertheless, the VOR gains are not ideal and thus small-amplitude compensatory saccades are present (* in Figure 2A and 2B). In contrast, the peak eye velocities during ipsilesional rotation to near- and far-target viewing are similarly reduced and thus larger-amplitude compensatory saccades are required to fixate the near target (* in Figure 2C and 2D). VOR indicates vestibulo-ocular reflex.

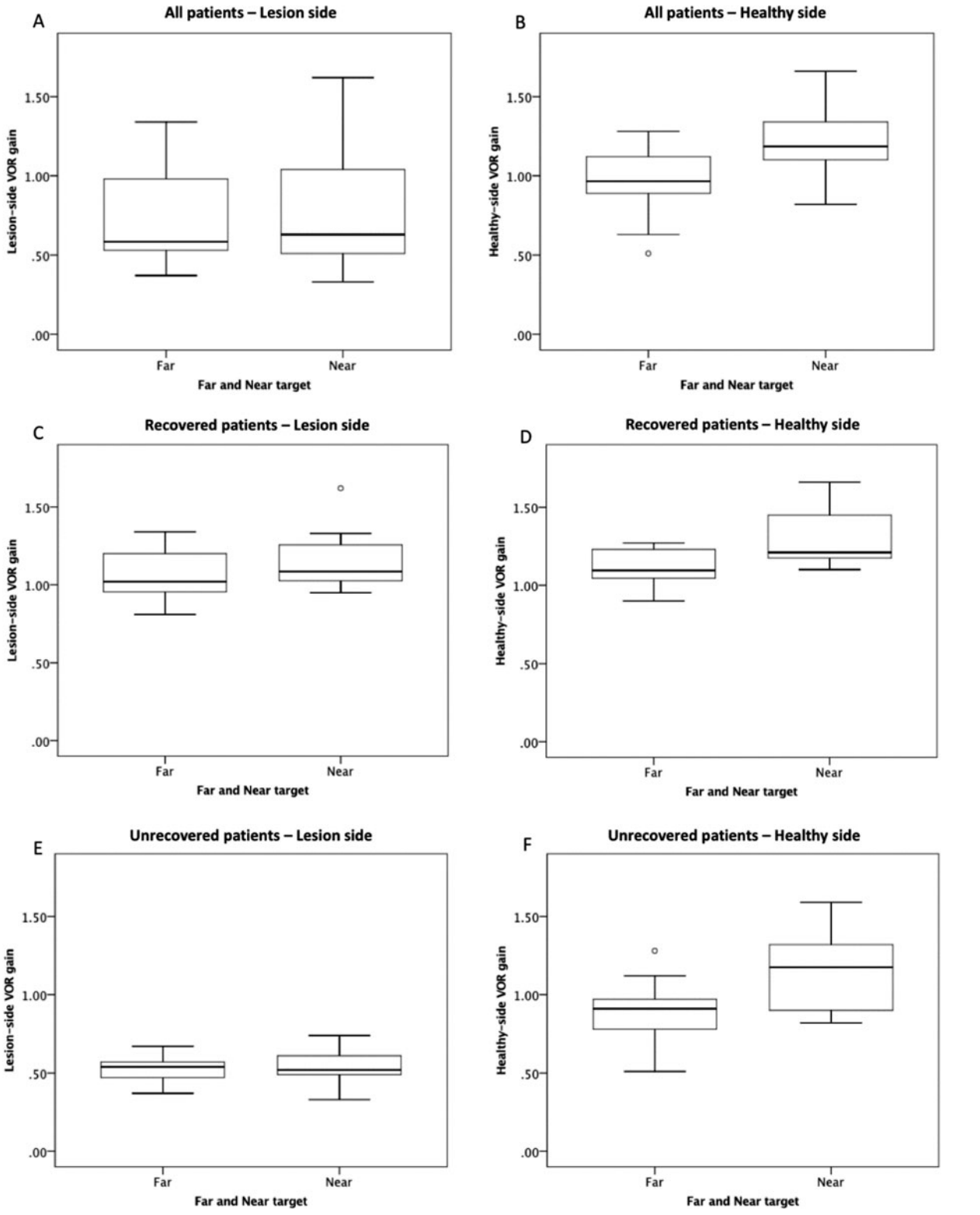

Figure 3. Box and whisker plots comparing far-viewing and near-viewing VOR gains in the lesion side (Figure 3A, 3C, and 3E) and the healthy side (Figure 3B, 3D, and 3F) of all the UVH subjects (Figure 3A and 3B), the recovered UVH subjects (Figure 3C and 3D), and the unrecovered UVH subjects (Figure 3E and 3F). Median (central line), upper and lower quartiles, upper and lower extremes, and outliers are given. UVH, unilateral vestibular hypofunction; VOR, vestibulo-ocular reflex.

Table 2. The Retinal Slip Index[a] (Mean ± SD) for Fixation at Far and Near Targets

	Far Viewing (Distance: 150 cm, Ideal VOR Gain: 1.07)		Near Viewing (Distance: 15 cm, Ideal VOR Gain: 1.67)	
Group	**RSI, Ipsilesional**	**RSI, Contralesional**	**RSI, Ipsilesional**	**RSI, Contralesional**
All UVH	0.35 ± 0.29	0.10 ± 0.21	0.90 ± 0.34	0.47 ± 0.23
Recovered UVH	0.01 ± 0.17	− 0.04 ± 0.13	0.51 ± 0.22	0.37 ± 0.20
Unrecovered UVH	0.54 ± 0.08	0.18 ± 0.20	1.13 ± 0.11	0.53 ± 0.23

Abbreviations: RSI, retinal slip index; UVH, unilateral vestibular hypofunction; VOR, vestibulo-ocular reflex.
[a]RSI = Ideal VOR gain − Actual VOR gain. An RSI of zero implies no retinal slip. The higher the RSI, the greater the magnitude of retinal slip.

DISCUSSION

This study reveals that vergence-mediated VOR gain enhancement during near viewing is preserved for contralateral rotation but impaired for ipsilesional rotation in UVH subjects, and that some subjects show a partial recovery of their vergence-enhanced VOR gain concurrent with the recovery of their passive VOR gain (far viewing). Although the magnitude of recovery of vergence VOR gain was not statistically significant, it was improved approximately 10%—which is greater than the amount of change in VOR gain that has been shown to be clinically relevant as related to fall risk is (0.06).[18] On the contrary, if there is no recovery of vestibular function, then vergence enhancement is lost for ipsilesional albeit preserved for contralesional head rotation.

The amount of VOR enhanced by viewing near targets has been studied in animal experiments for 40 years[14,19] but not confirmed in human until early this century.[15,16,20] In a search-coil study on 11 subjects with intratympanic gentamicin injection for unilateral Meniere disease, VOR gains were enhanced by viewing near target in the untreated ears but not in the treated ears.[16] This is putatively related to selective damage of the type I vestibular hair cells. In another study of subjects with semicircular canal plugging to repair a superior semicircular canal dehiscence, the vergence-mediated enhancement was normal and the near-viewing VOR gains were increased in the treated ear,[20] also supporting that vestibular hair cell lesion is the origin of the phenomenon. A recent, though smaller sample study did measure the near-viewing VOR using vHIT and showed an impaired vergence-mediated VOR enhancement; however, the authors report neither the effects of recovery nor the etiology for their subjects.[21] Our study demonstrates that the impairment of vergence-mediated VOR gain enhancement exists across a broader diagnostic group (ie, viral infection, labyrinthine infarction, and vestibular schwannoma) than those etiologies previously studied (eg, intratympanic gentamicin, canal plugging).

Mediation of the vergence-enhanced VOR is presumed to be within the cerebellum and/or vestibular nucleus (position-vestibular-pause neurons), which integrates the type I hair cell and irregular vestibular afference of the semicircular canals and otolithic organs, eye position signals from both the neural integrator (ie, nucleus prepositus hypoglossi) and the proprioceptive fibers, and likely the visual disparity signals from visual cortex.[22-27] Most of the subjects in our study have vestibular neuritis presumed to be due to a viral infection of the vestibular nerve[28] though 2 of our subjects suffered an acute vestibular syndrome with sudden deafness that was magnetic resonance imaging confirmed to be labyrinthine stroke and not vestibular labyrinthitis. These 2 subjects had no other signs of stroke. Therefore, it appears that the impaired VOR enhancement in most our subjects is caused by damage to the irregular afferents within the eighth cranial nerve. For those subjects with restored vestibular function, the damaged irregular afferents putatively self-repaired, given the partially restored vergence-mediated VOR gain enhancement.

Our study confirms that the VOR gain during rotations toward the healthy side in UVH during high-frequency near-target viewing does not generate an ideal fixation.[14] This results in some degree of retinal slip that is more severe for head rotations toward the lesion side due to the loss of vergence-mediated enhancement. Our data support that retinal slip persists during near-target viewing even in those with recovery of their passive VOR gain, albeit to a lesser amount (far-viewing RSI 0.01 vs near-viewing RSI 0.51). Therefore, it can be expected that subjects with UVH have greater gaze instability and likely more dizziness during head motion and near viewing, as occurs in common activities such as viewing a smartphone while walking.

We believe that the vergence-mediated gain phenomenon should be considered in prescribing gaze stability exercises for patients with UVH, presuming that such individuals have a normal convergence mechanism, given the phenomena are preserved for contralateral head rotation. A "vergence-mediated VOR gaze stability exercise" has the potential to improve function, given it causes retinal slip.[29] Our data show that near-target viewing during head rotation increases retinal slip and VOR gain beyond what occurs during far-target viewing (Figure 4). Furthermore, existing studies have established that vergence can be used as a unique switch (context), which the brain then uses to engage a prior learned VOR gain.[30] Additional studies are warranted to compare the benefit of the vergence VOR exercise against the current gaze stability exercises.

Our study has several limitations. First, we used monocular VOG recordings to test the subjects, therefore, precluding our ability to calculate the vergence angle. However, a recent study has confirmed that monocular VOG is a valid measure of the near-viewing VOR gain.[31] In addition, we recorded any eye position change during the cover/uncover and alternate cover tests to demonstrate that vergence was present in each person while viewing the near target before vHIT. A second limitation is our inability to directly confirm that each person maintained vergence during vHIT. However, we suspect that the reduction in vergence angle during vHIT was low, given the short

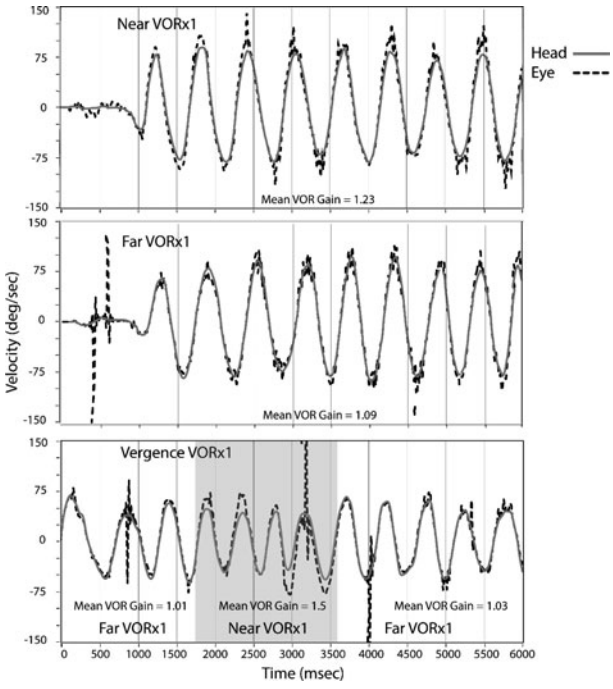

Figure 4. Vestibulo-ocular reflex gain during near, far, and vergence × 1 viewing exercises in a healthy control. During near VOR × 1 (top), the subject held a card with a single letter 15 cm in front of the eyes while moving the head in yaw and maintaining focus on the letter. Next, the subject held the card at arm's length in front of the eyes and repeated the VOR exercise (Far VOR × 1, middle). Finally, the subject moved the target from arm's length to the bridge of their nose and back while attempting to keep focus on the target (vergence VOR × 1, bottom). The peak velocity of the eye was higher than that of the head when the visual target was close to the eyes in near VOR × 1 and vergence VOR × 1 (shaded region). Eye velocity (dotted black); head velocity (gray); positive values denote rightward rotation. Note the VOR gain values. VOR indicates vestibulo-ocular reflex.

duration of each vHIT trial and that no person complained of diplopia throughout vHIT testing. Third, our sample size is not large, the intervals between onset of dizziness and data collection were heterogenous, and our healthy control subjects were not age-matched. Finally, although our study provides a theoretical basis for creating a vergence-mediated gaze stability exercise in subjects with UVH, we have not directly tested the effectiveness of this exercise.

CONCLUSIONS

In UVH, near-viewing VOR gain is significantly enhanced during contralesional but not ipsilesional rotation. The vergence-mediated gain enhancement during ipsilesional rotations partially recovers in those subjects with recovery of their passive VOR gain, though it remains impaired. This result suggests exposure to gaze stability exercises that demand vergence will increase the retinal slip when viewing a near target, which may be useful for driving VOR adaptation.

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