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Mechanism of Dynamic Visual Acuity Recovery With Vestibular Rehabilitation

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Abstract

Objective—To determine why dynamic visual acuity (DVA) improves after vestibular rehabilitation in people with vestibular hypofunction.

Design—Combined descriptive and intervention study.

Setting—Outpatient department in an academic medical institution.

Participants—Five patients (age, 42–66y) and 4 age-matched controls (age, 39–67y) were studied. Patients had vestibular hypofunction (mean duration, 177 ± 188 d) identified by clinical (positive head thrust test, abnormal DVA), physiologic (reduced angular vestibulo-ocular reflex [aVOR] gain during passive head thrust testing), and imaging examinations (absence of tumor in the internal auditory canals or cerebellopontine angle).

Intervention—Vestibular rehabilitation focused on gaze and gait stabilization (mean, 5.0 ± 1.4 visits; mean, 66 ± 24 d). The control group did not receive any intervention.

Main Outcome Measures—aVOR gain (eye velocity/head velocity) during DVA testing (active head rotation) and horizontal head thrust testing (passive head rotation) to control for spontaneous recovery.

Results—For all patients, DVA improved (mean, $51\% \pm 25\%$; range, 21%–81%). aVOR gain during the active DVA test increased in each of the patients (mean range, 0.7 ± 0.2 to 0.9 ± 0.2 [35%]). aVOR gain during passive head thrust did not improve in 3 patients and improved only partially in the other 2. For control subjects, aVOR gain during DVA was near 1.

Conclusions—Our data suggest that vestibular rehabilitation increases aVOR gain during active head rotation independent of peripheral aVOR gain recovery.

Keywords

Reflex; vestibulo-ocular; Rehabilitation; Saccades; Vestibular diseases; Visual acuity

When functioning normally, the angular vestibulo-ocular reflex (aVOR) keeps images stable on the fovea during head motion. When viewing targets at distances greater than 1m, the aVOR

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generates eye movements that are opposite in direction but equal in magnitude to the head rotation. This is necessary to maintain stability of the eyes with respect to space (termed *gaze stability*) and ensure clear vision during head motion. The fovea occupies a small region of the visual field, and image movement off the fovea by as little as 2° to 3° can cause substantial reduction of gaze stability.¹ In the case of vestibular hypofunction, eye rotational velocity is less than head rotational velocity, and gaze stability is reduced. In an attempt to stabilize gaze, people with vestibular hypofunction use different compensatory strategies to improve the ability to see clearly during head rotation.² Compensatory mechanisms include substitution or modification of a saccadic eye rotation that occurs in the direction of the deficient aVOR,^{2–6} increased gain of the cervico-ocular reflex,^{2,7} and perhaps enhancement of the smooth pursuit system.⁸ As far as we know, no study has investigated whether the aVOR is modifiable with vestibular rehabilitation and therefore a compensatory strategy of gaze stability.

Recruitment of saccades to assist a deficient aVOR during ipsilesional head rotations is well established and termed *compensatory saccades* or *vestibular catch up saccades*.^{3–6} These compensatory saccades are unique and characterized as occurring during the head rotation, in a direction opposite the head rotation, and with a latency (40 – 100ms) much shorter than that of a volitional saccade (200ms).^{5,6,9} It has been shown that during head motion, compensatory saccades reduce the amplitude of eye position errors (due to the deficient aVOR) by up to 59%.^{3,4} It is possible that these saccades stabilize gaze and assist dynamic visual acuity for people with vestibular hypofunction.

Dynamic visual acuity (DVA) refers to one's ability to see during head motion. The DVA test is a functional measure of gaze stability and incorporates head rotations that represent natural head velocities.^{10,11} For people with normal vestibular function, visual acuity during head motion and head still is similar. For patients with vestibular hypofunction, the aVOR will not keep gaze stable in space during head motion. This leads to retinal slip (image motion off the fovea of the retina) with a reduction in DVA compared with the head still. The computerized DVA test has been validated in its ability to identify the side of vestibular hypofunction for active (predictable) and passive (unpredictable) head motion.^{12–14}

Vestibular rehabilitation incorporates gaze stability exercises that mimic the adaptation experience that has been used to change the aVOR in animal and human studies.^{15,16} This is achieved by ensuring that visual images move off the fovea of the retina (retinal slip). Paired with head rotation, retinal slip is a powerful means for aVOR gain adaptation.¹⁷ Controlled studies have shown that gaze stability exercises lead to an improved DVA in subjects with unilateral (UVH) and bilateral vestibular hypofunction (BVH).^{18,19} To date, however, the mechanism of recovery of DVA is unknown but may involve enhanced aVOR gain (eye velocity/head velocity) or recruitment of a nonvestibular eye movement like the compensatory saccade.

The purpose of this study was to describe the eye responses during DVA and to determine the oculomotor mechanism responsible for recovery of DVA. Our results suggest 2 mechanisms responsible for recovery of DVA: improved aVOR gain for active head rotation and, in some subjects, an increase in the number of compensatory saccades generated per active head rotation.

METHODS

We studied 5 subjects (mean age, 54.4 ± 8.9y; range, 42–66y) with vestibular hypofunction (4 with UVH, 1 with asymmetric BVH) before and after vestibular rehabilitation. Vestibular hypofunction was based on history of imbalance, nonpositional vertigo, physical examination showing a positive head thrust test result toward the affected ear, absence of a mass-enhancing

lesion within the internal auditory canals or cerebellopontine angle, and abnormal DVA score. We also studied 4 control subjects (mean age, 54 ± 12.8 y; range, 39–67y) who had no complaints of vertigo, dizziness, or imbalance and had normal DVA for active horizontal head rotation.¹² All subjects underwent passive head thrust aVOR gain testing using the 3-dimensional scleral search coil recording technique.²⁰ Passive head thrust testing is necessary to document function and, in the case of pathology, is useful to identify spontaneous recovery of the peripheral vestibular labyrinth.⁶ When subjects showed improvement in aVOR gain during passive head thrust testing, they were classified as having recovery of the UVH (UVHr); otherwise they were classified as having a chronic lesion (UVHc). Participation in this study was voluntary, and all subjects consented to be a part of this project in accordance with a protocol approved by Johns Hopkins University School of Medicine's institutional review board.

Head Thrust Test and the Scleral Search Coil Technique

A horizontal head thrust consists of a passive, unpredictable (timing and direction) manual head rotation with peak amplitude of about 20°, peak velocity of about 250°/s, and peak acceleration of about 3000°/s.²¹ Each subject was tested while seated upright and centered within a uniform magnetic field, with the interpupillary line in the earth-horizontal plane. The head was positioned so that Reid's line (from the superior most point of the bony-cartilaginous junction of the external auditory canal to the lowest point of the cephalic edge of the infraorbital rim) was also in the earth-horizontal plane, which we term the *zero reference position*. Before the start of each head thrust, each subject's head was placed in the zero reference position. Subjects fixated a light-emitting diode (LED) target positioned 124cm directly in front of them. The room was completely dark except for this LED.

Binocular eye movements were recorded in 3 rotational dimensions using a pair of search coils embedded in a silicone annulus, which was placed directly onto the sclerae of each eye. A search coil pair embedded in a bite block was used to measure head rotation. Eye and head angular positions were sampled at 500Hz at 16-bit resolution. Analog signals (presampled) were low-pass filtered with a single-pole analog filter that had a 3-dB bandwidth of 100Hz. Digital (postsampled) signals were filtered with a 50-tap zero-phase low-pass digital finite impulse response filter with 50-Hz bandwidth.

DVA Test Protocol

Detailed descriptions of the DVA test have been reported previously.^{12,22} In brief, subjects were seated 2m (6.5ft) directly in front of a high-resolution, 18.1 viewable-inch monitor with a refresh rate of 85Hz. Subjects who normally wore glasses or contact lenses for distant viewing were instructed to wear them during all DVA testing. Static visual acuity was measured first by repeatedly displaying a single optotype (the letter E, randomly rotated each trial by 0°, 90°, 180°, or 270°) on a computer monitor. Subjects viewed 5 optotypes per acuity level, with optotype size then being decreased in steps equivalent to a visual acuity change of 0.1 logMAR ($\log_{10}x$, where x is the minimum angle resolved, in arcmin, with 1 arcmin equal to $1/60^\circ$).²³ The better one's visual acuity, the lower one's logMAR score, with logMAR equal to -0.3, 0, 0.3, 0.7, 1.0, and 1.3, corresponding to a Snellen visual acuity of 20/10, 20/20, 20/40, 20/100, 20/200, and 20/400, respectively. Static visual acuity was scored when a subject failed to correctly identify 5 optotypes on an acuity level or reached the logMAR score of .000 (Snellen equivalency of 20/20 acuity).

For the dynamic component of the test, a single-axis Watson rate sensor^a was positioned on each subject's head so that the sensor's axis of maximum sensitivity approximately aligned

^aSupplier Micromedical Technologies Inc, 10 Kemp Dr, Chatham, IL 62629.

with that of the horizontal semicircular canal.²⁴ All subjects were instructed in an initial practice trial for self-generated sinusoidal horizontal head rotations to control for practice effect and establish reliability for the normative controls.¹² During each head rotation, an optotype E randomly oriented in 1 of 4 directions was displayed on the monitor 2m in front of the subject when head velocity was between 120° and 180°/s (for right-side DVA testing) or between -180° and -120°/s (for left-side DVA testing) for more than 40ms. This enabled us to determine DVA scores for rightward and leftward head rotations separately.

To allow for blinks or transient loss of attention, subjects were allowed to view each optotype a maximum of 5 times, at which point the computer no longer displayed the letter and subjects were required to guess the orientation. Once a subject indicated a response, the next trial started. The test was terminated once a subject incorrectly identified all 5 optotype presentations at 1 acuity level (ie, 20/40₋₅) or reached the log-MAR level of .000 (Snellen 20/20_{-n}). The subscripted digit identifies the number of incorrect responses (5 possible) at the acuity level listed. Periodically, subjects were asked to stop the active head rotation for a 20-second rest period and were encouraged to keep their eyes closed or blink.

The DVA test score was calculated by subtracting the static visual acuity logMAR score from the dynamic visual acuity logMAR score. Additional information about logMAR computation has been published elsewhere.¹² Previously established control data have grouped DVA score across 6 decades of age starting at age 20 through age 79.¹² All subjects completing the DVA score were identified as having an abnormal or normal DVA score compared with these data.

aVOR Gain and Compensatory Saccade Data Analysis

Angular positions for eye and head with respect to space coordinates and eye with respect to head coordinates were represented by rotation vectors.^{25,26} Head-in-space, eye-in-space, and eye-in-head velocity vectors were calculated from the corresponding rotation vectors.²⁷ Head velocity was calculated and reported with reference to a head-fixed, right-handed coordinate frame (superior-inferior axes correspond with yaw axes), so that eye-in-head and head velocities were expressed with reference to exactly the same coordinate frame.²⁸

The onset of each head thrust was identified with curve fitting. The time at which the magnitude of the fitted curve became greater than 2% of the curve's peak magnitude (typically this threshold was $\approx 4^\circ/\text{s}$) was defined as the onset. A similar approach was used to identify the onset of the eye movement responses. Analysis of the thrust data was restricted to a period of 150ms from the head velocity onset. Horizontal aVOR gain for head thrusts were calculated by dividing horizontal eye velocity by horizontal head velocity during the 30-ms period before peak head velocity and were averaged across trials. Horizontal aVOR gain during DVA testing was calculated by dividing peak horizontal eye velocity by peak horizontal head velocity.

We define a *compensatory saccade* as a saccade occurring during the head rotation and in the direction of the vestibular slow component.^{5,6,9} Peak compensatory saccades velocity and amplitude were determined from velocity and position traces.

Gaze Stabilization Exercises

Subjects with vestibular hypofunction were asked to perform gaze and gait stability exercises 4 to 5 times a day, for a total of 20 to 30 minutes. The gaze stability exercises require subjects to focus on visual targets during head motion. Patient subjects were also instructed in static and dynamic balance exercises. This exercise protocol was progressed weekly, based on improvements documented in clinic (ie, improved dynamic balance, increased head velocity during gaze stability exercises), similar to a protocol that has previously been established to improve DVA.¹⁸

Dizziness Handicap Inventory

Dizziness Handicap Inventory (DHI) data were collected to assess subjective improvement in each participant's perceived limitations as a result of the vestibular hypofunction. Patients responded to 25 questions subgrouped into functional, emotional, and physical components. The DHI has excellent test-retest reliability ($r = .97$) and good internal consistency reliability ($r = .89$).²⁹

Statistical Analysis

Analysis of variance (ANOVA) was used to control for differences in age across patients and controls. Individual differences between aVOR gain, peak head velocity, peak slow phase eye velocity (SPEV), compensatory saccades velocity, compensatory saccades amplitude, and compensatory saccades frequency during DVA were assessed using independent t testing assuming equal variance ($P < .05$). For the controls, statistical comparisons for the compensatory saccades were performed only when the number of compensatory saccades generated was 10 or more. We described the number of compensatory saccades per head rotation as a ratio to determine change for patient subjects, before and after rehabilitation. DVA scores were compared with those of age-matched normative controls.

RESULTS

Control Subjects

DVA score and aVOR gain—Trials of head thrust data that included blinks or in which the subject did not fix on the target with both eyes at the onset of head rotation were not included in the analysis. DVA scores for control subjects were in agreement with previously established age-matched healthy control values (table 1). The aVOR gains for passive head thrust testing and during the DVA testing were normal (table 2). Figure 1A illustrates a normal passive head thrust test in the horizontal canal for a 50-year-old control subject.

Compensatory saccades—Control subjects used compensatory saccades during DVA; however, their frequency was much lower than that in patient subjects. Statistical comparison was performed on only 2 control subjects that generated 10 or more compensatory saccades. One control subject had a significant difference in compensatory saccades amplitude between rightward and leftward DVA testing ($P < .05$); no other differences were found. Mean peak compensatory saccades frequency, velocity, amplitude, and ratio per DVA test direction for each of the controls are listed in table 3.

Although the overall numbers were small, we noticed a trend between compensatory saccades frequency and DVA test side. For example, 3 of 4 control subjects made more compensatory saccades during ipsi-rotational DVA testing compared with the contra-rotational head direction—that is, during right-side DVA testing, subjects tended to generate more compensatory saccades for rightward head rotation when the optotype flashed than during leftward head rotations (blank screen). We did not notice an appreciable difference in age concerning the recruitment of these unique saccades.

Patient Subjects

DHI score, DVA score, and aVOR gain—Figure 1B illustrates a positive head thrust test result for head rotations to the left, showing a deficient horizontal aVOR. We did not find any difference in age between controls and subjects with vestibular hypofunction (ANOVA, $P = .96$). Patient subjects were seen in clinic for a mean of 5.0 ± 1.4 visits over mean 66 ± 24 days. DHI scores improved over this time period in 3 of 5 subjects (UVHr, UVHc) by a mean score

of 22 ± 13 points. DHI scores in the other 2 subjects were unchanged (mean, -3.0 ± 1.4 points) (see table 1).

Four of 5 subjects with vestibular hypofunction had an improvement in the ipsilesional DVA score from pre- to post-vestibular rehabilitation for a combined mean decrease of $51\% \pm 25\%$ (range, 21%–81%). DVA scores in 3 of 4 subjects with UVH returned to normal, and the fourth subject had a 22% improvement that was .03 logMAR units from age-matched normative control data. Although the DVA score for the fifth subject (BVH) did not return to normal, it did improve by a mean of $21\% \pm 9\%$ (see table 1).

Active aVOR gain during ipsilesional DVA testing was found to significantly improve on completion of vestibular rehabilitation for all patients ($P < .05$) (see table 2). The subject with BVH also had a significant improvement in aVOR gain during DVA testing of the less affected side (62%). Combined, subjects with vestibular hypofunction had an average of $35\% \pm 29\%$ (range, 8%–76%) aVOR gain increase during DVA testing.

Compensatory saccades—We found a combined $40\% \pm 13\%$ *increase* in the number of compensatory saccades recruited for subjects with chronic UVH after rehabilitation. The subject with BVH used a similar number of compensatory saccades before and after rehabilitation. In contrast, the subject with UVH who showed passive aVOR gain recovery (head thrust testing, UVHr) had a 47% *reduction* in the number of compensatory saccades generated during ipsilesional DVA testing.

Three of 4 subjects (1 BVH, 2 UVHc) with chronic vestibular hypofunction also had an increased ratio of compensatory saccades/head rotation (table 4). Figure 2 illustrates some different mechanisms recruited to improve DVA. Panel A illustrates active aVOR gain recovery and reduction in compensatory saccades recruitment from before to after rehabilitation in subject UVHr. Active aVOR gain recovery is also evident in panels B and C; however, the number of compensatory saccades recruited for both the UVHc and BVH subjects has increased. Note, too, the limited number of compensatory saccades in the subject with BVH in the non-DVA test direction—suggesting a correlation between compensatory saccade recruitment and desire for gaze stability.

DISCUSSION

Our data suggest that gaze stability exercises improve visual acuity during active head rotation via 2 primary mechanisms: an improvement in active aVOR gain and an increase in the number of compensatory saccades. In addition, our data suggest that gaze stability exercises reduce the perception of dizziness handicap experienced by subjects with unilateral vestibular hypofunction.

Previous studies suggest 2 possible explanations for how the gaze stability exercises improve aVOR gain during DVA testing. First, afferent physiology studies in animals with vestibular loss show that the central vestibular neurons retain their rotational sensitivity.³⁰ This suggests the aVOR can be modified although it has been damaged. Another explanation may be enhancement of the smooth pursuit system. Recently, Bockisch et al⁸ investigated smooth pursuit in subjects with BVH and reported that they had, on average, 9% greater gains than those of normative controls. Bockisch reported peak velocities reaching $40^\circ/\text{s}$ and concluded that smooth pursuit may be a useful compensatory mechanism for a deficient aVOR. Although we did not measure smooth pursuit, we do not believe this system contributed much to our result. The smooth pursuit system is typically limited to velocities less than $60^\circ/\text{s}$ and peak frequencies near 1 Hz.^{31,32} Even motivated healthy people trained in smooth pursuit have been reported to have pursuit velocities ($90^\circ/\text{s}$) less than our DVA testing parameters (120° – $180^\circ/$

s).³³ Existing data therefore suggest that if smooth pursuit is a compensatory gaze stability strategy, it would be limited to head rotational velocities below 100°/s.

As far as we know, our data represent the first mechanistic link between gaze stability exercises and improvement in the ability to see clearly during head rotation, suggesting important implications for gaze stability rehabilitation programs. Other nonvestibular motor control studies^{34,35} suggest that learning tasks that incorporate a graded exercise program, such as the gaze stability program outlined in this study, are effective in causing neural plasticity and learning.

Recruitment of Compensatory Saccades

We found that subjects with vestibular hypofunction use a unique type of saccade (compensatory saccade) to assist gaze stability during DVA testing. These saccades appear to be similar to those described in nondynamic visual acuity studies in which subjects with vestibular hypofunction underwent passive or active head rotation.^{3–6} These saccades therefore appear to arise independent of a specific type of exercise. Their correlation with vestibular hypofunction suggests that their onset is related to retinal slip or retinal position error signals, and they are useful to assist gaze stability.

Prior literature has established that people use different compensatory strategies to maintain gaze stability.² Our data support this. We found that a subject with peripheral aVOR gain recovery (UVHr) reduced the ratio of compensatory saccades/head rotation by more than half. In most subjects with chronic vestibular loss, however, the ratio increased, albeit to varying amounts. This suggests that the recruitment of compensatory saccades is inversely correlated with aVOR gain, and in some cases this relationship is reversible depending on peripheral aVOR gain recovery. Tian et al⁵ also reported an inverse correlation between aVOR gain and recruitment of compensatory saccades.

There is in theory a possibility that the compensatory saccades reported here are vestibular quick phases due to accentuating the tonic asymmetry in the central vestibular neurons, such as is manifested in post-head shaking nystagmus (HSN).³⁶ This is extremely unlikely, for 2 reasons: (1) HSN requires subjects to have blocked foveal fixation and (2) HSN causes saccades (quick phases) to always occur toward the intact side and in the same direction. As can be seen in figure 2, the type of saccades displayed occurs during both head rotational directions and in room light (DVA test).

It is interesting that the compensatory saccades velocity and amplitude were significantly reduced at the final DVA measure. This may be related to the active aVOR gain recovery. That is, the slow component eye velocity increased with a corresponding reduction in the retinal slip error signal. Although the number of saccades/head rotation (ratio) increased, their velocity and amplitude may have decreased due to the reduced retinal error signal.

Finally, there appears to be a preference to recruit more compensatory saccades for head rotations in the DVA test direction. That is, when DVA was being tested (optotype flashing) for head rotation to the right only, control subjects showed a trend to use more compensatory saccades for head rotations toward the right than toward the left. This suggests that the recruitment of the compensatory saccades is dependent on a desire for the brain to see clearly during active head rotation.

Residual aVOR Gain Deficiency During Passive Head Rotation

Our data also show that the passive aVOR (head thrust test) does not commonly recover in subjects with chronic unilateral vestibular hypofunction after rehabilitation. Although one of our subjects did have a large increase in passive aVOR gain, this person had vestibular neuritis,

which has been shown to be reversible in some cases.³⁷ Our data suggest that gaze stabilization exercises do not cause much improvement in aVOR gain during passive head thrust rotations. Szturm et al³⁸ and later Herdman et al¹⁹ measured aVOR gain using passive whole body rotation before and after gaze stability exercise programs to determine whether peripheral vestibular recovery could explain changes in measured outcomes variables. Each of the studies used low- (60°/s) and high-velocity (120° or 240°/s) passive rotations. For people with chronic vestibular hypofunction, Szturm et al³⁸ reported a reduction in aVOR gain asymmetry for 60°/s but not for 120°/s rotations. For rotational velocities below 100°/s, visual mechanisms (eg, smooth pursuit coupled with input from the contra-rotational peripheral end organ) can generate a normal aVOR.³⁹ For higher-velocity rotations (>100°/s), however, passive aVOR gain depends mostly on input from the ipsi-rotational peripheral end organ.⁴⁰ Therefore, recovery of vestibular asymmetry for passive low-velocity rotations without simultaneous recovery for higher-velocity rotations implies recovery of the central aVOR pathways, not the peripheral aVOR pathway.⁴¹ For people with BVH, Herdman et al¹⁹ reported no change in aVOR gain for either 60° or 240°/s whole body rotations, although DVA scores (active head rotation) improved significantly. Neither of these studies measured the aVOR gain for active head rotation.

Improvement in Subjects' Perceptions of Dizziness Handicap

We found that 3 of 5 subjects with vestibular hypofunction had an improved DHI score similar to values previously reported.⁴² However, only 2 (40%) of 5 subjects had a significant change in perception of handicap, identified by Jacobson and Newman²⁹ as a greater than 18-point difference when the DHI is used as a measure of change. Others have also reported similar magnitude of improvement in DHI scores (≈35%) after vestibular rehabilitation.⁴³

Study Limitations

Our study would have been more powerful had we incorporated a randomized and controlled crossover design. However, we believed this would have been unethical, considering that previous studies have established gaze stability exercises improve DVA.¹⁸ Another limitation of this study involved the single measurement of DVA in the controls. We believe this was justifiable having previously shown that the DVA test is stable over time.¹²

CONCLUSIONS

Our results suggest that DVA recovers as a result of improved active aVOR gain independent of peripheral vestibular recovery. In addition, we report that the number of compensatory saccades used per head rotation is variable and appears inversely correlated with passive aVOR gain. The compensatory saccades may be a useful gaze stability mechanism for some people. Together, these data suggest that vestibular rehabilitation has a mechanistic effect on recovery of gaze stability during active head rotation.

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Fig 1.

aVOR gain during passive horizontal head thrust testing in a healthy control subject and subject with left UVH. Left and right refer to direction of passive horizontal head thrust rotation. (A) Data from both eyes in a subject with normal VOR function. (B) Data from left eye only in a subject with left UVH. Head and/or eye velocity plots have been inverted for ease of comparison. Note that the quick phases in the bottom left panel are in the same direction as the slow-phase eye velocity, illustrating the compensatory saccades.

Fig 2.

(A) Patients with UVH and partial recovery; (B) patients with UVHc; and (C) patients with BVH. Improvement of aVOR gain during DVA testing. Eye velocity plots have been inverted for ease of comparison. Eye velocities are from left eye only. For head rotation in the contralesional direction, compensatory saccades occur with much less frequency, suggesting that compensatory saccades recruitment is dependent on subjects' intent for gaze stability. NOTE. The compensatory saccades (CS) ratio is the number of compensatory saccades/total number of head rotations.

Table 1

Subject Characteristics and DVA Scores

Diagnosis	Age (y)	Pre-DHI	Post-DHI	Pre-DVA Score		Post-DVA Score	
				Ipsi/L	Contra/R	Ipsi	Contra
UVHr	42	34	8	.097	.018	.018	.018
Normative	38	NA	NA	.018	.018		NA
Age-match bin	35–44	NA	NA		.094		.094
BVHc	50	60	64	.319	.273	.273	.198
Normative	50	NA	NA	.071	.071		NA
Age-match bin	45–54	NA	NA		.162		.162
UVHc	56	56	22	.271	.230	.171	.171
UVHc	58	26	28	.180	.160	.097	.076
Normative	61	NA	NA	.112	.127		NA
Age-match bin	55–64	NA	NA		.168		.168
UVHc	66	34	26	.421	.158	.180	.058
Normative	67	NA	NA	.107	.107		NA
Age-match bin	65–74	NA	NA		.216		.216

NOTE: Values in italics represent DVA scores from the age-matched healthy control subjects. Values in boldface represent mean $\pm$ 2 standard deviations (SDs) DVA scores (pooled right and left horizontal head rotation) from previously established age-matched control data for the listed age-match bin. ¹² DVA values are in logMAR units.

Abbreviations: c, chronic; Contra, contralesional head rotation; Ipsi, ipsilesional head rotation; L, left; NA, not applicable; r, recovery; R, right.

Table 2

aVOR Gain During Active DVA and Passive Head Thrust Test for Horizontal Head Rotation

Diagnosis	Ipsilesional/Left Active DVA aVOR Gain (Passive HTT aVOR Gain)		Contralesional/Right Active DVA aVOR Gain (Passive HTT aVOR Gain)	
	Prerecovery	Postrecovery	Prerecovery	Postrecovery
UVHr	0.68±0.04 (0.25±0.03)	1.02±0.13* (0.62±0.08*)	1.01±0.15 (1.10±0.08)	1.02±0.08 (1.10±0.09)
Normative	<i>1.02±0.02 (0.98±0.10)</i>	NA (NA)	<i>1.00±0.02 (0.98±0.08)</i>	NA (NA)
BVHc	0.48±0.10 (0.17±0.04)	0.85±0.06* (0.16±0.04)	0.62±0.09 (0.48±0.04)	1.01±0.05* (0.66±0.03*)
Normative	<i>1.03±0.02 (0.97±0.06)</i>	NA (NA)	<i>1.02±0.01 (0.96±0.05)</i>	NA (NA)
UVHc (age 56)	0.94±0.10 (0.43±0.06)	1.03±0.07* (0.49±0.07*)	1.10±0.12 (1.08±0.06)	1.08±0.10 (0.76±0.03*)
UVHc (age 58)	0.57±0.09 (0.36±0.12)	0.61±0.08* (0.31±0.08)	1.00±0.15 (0.61±0.11)	0.97±0.09 (0.71±0.03*)
Normative	<i>1.13±0.07 (1.01±0.1)</i>	NA (NA)	<i>1.12±0.06 (1.01±0.09)</i>	NA (NA)
UVHc (age 66)	0.86±0.06 (0.48±0.07)	1.10±0.03* (0.33±0.04*)	1.25±0.08 (1.11±0.05)	1.30±0.14 (0.82±0.07*)
Normative	<i>1.06±0.08 (1.02±0.03)</i>	NA (NA)	<i>1.04±0.07 (1.02±0.03)</i>	NA (NA)

NOTE. Values are mean ± 1 SD. Values are for horizontal head rotations. Values in italics represent scores from the age-matched healthy control subjects to be compared with the data in the row(s) above. Values in parentheses are from the passive head thrust test. Ipsilesional values are from the subject with BVH for the leftward rotations.

Abbreviation: HTT, head thrust test.

* $P < .05$ for pre and post comparisons.

Table 3

Characteristics of Compensatory Saccades for Normative Subjects

Age (y)	Compensatory Saccades Parameters During DVA Testing						VOR Parameters During DVA Testing					
	Frequency*		Amplitude (deg)		Ratio†		Peak Head Velocity (deg/s)		Peak SPEV (deg/s)			
	DVAL	DVAR	DVAL	DVAR	DVAL	DVAR	DVAL	DVAR	DVAL	DVAR	DVAL	DVAR
38	3L/1R	2R/1L	1.30±0.05	1.50±0.80	.03	.10	151±29	152±30	145±21	148±33		
50‡	12L/4R	8R/3L	4.30±1.30	3.50±1.00§	.10	.08	156±19	156±26	154±18	161±26		
61‡	40L/30R	39R/31L	2.70±1.60	3.00±1.50	.20	.13	141±17	148±18	151±19	157±18		
67	5L/7R	3R/1L	3.80±1.10	3.00±1.10	.08	.04	167±23	176±19	169±25	182±18		

NOTE. Values for amplitude, velocity, peak head velocity, and peak SPEV are mean ± 1 SD.

Abbreviations: DVAL, DVA test with head rotation to the left; DVAR, DVA test with head rotation to the right.

* Number of compensatory saccades that occurred during ipsi-rotational and contra-rotational DVA test.

† Number of compensatory saccades/number of head rotations.

‡ Statistical comparisons were applied only to those normative subjects who generated more than 10 compensatory saccades.

§ $P < .05$.

Table 4
Characteristics of Compensatory Saccades During Ipsilesional DVA Testing in Subjects With Vestibular Hypofunction

Diagnosis (Years of Age)	Compensatory Saccades Parameters During DVA Testing			VOR Parameters During DVA Testing		
	Frequency Pre (Post)	Mean Peak Compensatory Saccades Amplitude (deg) Pre (Post) [†]	Mean Peak Compensatory Saccades Velocity (deg/s) Pre (Post) [†]	Ratio Pre (Post)	Mean Peak Head Velocity (deg/s) Pre (Post)	Mean Peak SPEV (deg/s) Pre (Post) [†]
UVHc (38)	300±157 [†]	5.10±2.05 (4.30±1.80)	224±57 (138±47)	1.90±0.70	146±18 (140.2±15)	116±29 (150±23)
BVHc (50)	269±265	8.10±2.10 (5.00±2.10)	299±48 (169±36)	0.95±1.06	123±17 (124±17)	62±13 (105±16)
UVHc (56)	228±286 [†]	7.20±3.70 (6.20±2.60)	140±52 (90±31)	0.24±0.22	120±34 (129±21)	112±28 (133±21)
UVHc (58)*	248±356 [†]	15.00±7.60 (8.50±3.40)	195±42 (182±39)	0.56±0.90	183±35 (131±23) [†]	102±18 (79±10)
UVHc (66)	43±65 [†]	10.00±5.50 (8.80±3.60)	205±73 (184±68)	0.37±0.58	152±48 (136±12) [†]	133±37 (153±14)

NOTE: All subjects had reduced compensatory saccades amplitude and compensatory saccades velocity after rehabilitation.

Abbreviations: Post, postrecovery; Pre, prerecovery.

* Subject had reduced peak head velocity and SPEV from pre- to postrehabilitation.

[†] $P < .05$ for pre and post comparisons.