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Oculomotor Strategies and Their Effect on Reducing Gaze Position Error

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Objective: Vestibular adaptation exercises have been shown to improve gaze stability during active head rotation in individuals with vestibular hypofunction. Little is known, however, of the types of eye movements used during passive head rotation and their effect on gaze stability in individuals with vestibular hypofunction. The primary purpose of this study was to determine differences in oculomotor strategies and their effect on stabilizing gaze during ipsilesional passive and active head rotations in vestibular hypofunction.
Patients: Subjects with unilateral (n = 4) and bilateral (n = 3) vestibular hypofunction and healthy subjects (n = 4) based on bithermal caloric and rotational chair testing.
Intervention: Diagnostic.
Main Outcome Measure: Head and eye velocity and position data measured with scleral search coil.
Results: Subjects with unilateral and bilateral vestibular hypofunction generated 3 types of gaze-stabilizing eye movements with ipsilesional head impulses: slow vestibular ocular reflex, compensatory, and corrective saccades. The types of eye movements generated during active and passive head impulses were highly individualized. Gaze position error was reduced when compensatory saccades were recruited as part of the gaze-stabilizing strategy.
Conclusion: Rehabilitation for individuals with vestibular hypofunction should identify individuals' unique gaze stability preferences and attempt to facilitate compensatory saccades.
Key Words: Compensatory saccades—Oculomotor—Vestibular hypofunction—Vestibulo-ocular reflex.

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Uncompensated vestibular hypofunction results in visual blurring with head motion. Vestibular adaptation exercises involve head motion while maintaining gaze stability on a target and are based on studies that have shown that the vestibulo-ocular reflex (VOR) is modifiable when head motion induces retinal slip (1,2). Vestibular adaptation exercises have been shown to improve visual acuity during active head motion, reflecting improved gaze stability, in individuals with unilateral (UVH) and bilateral vestibular hypofunction (BVH) (3,4).

Gaze stability seems better for self-generated, or active, versus passive head movement in individuals with vestibular hypofunction (5,6). Enhancement of gaze stability with active head movement is thought to be due in part to compensatory eye rotations that may reflect central preprogramming. These compensatory eye movements are very quick, occur in the direction of the deficient slow phase, and have latencies shorter than volitional latencies (3,7).

The purpose of this study was to determine the type and frequency of different oculomotor response strategies associated with active and passive head rotations in individuals with vestibular hypofunction. In addition, we examined the contribution of these eye movements to reduce gaze position error. By determining the presence of specific eye movements, we hope to better understand

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how individuals with vestibular hypofunction maintain gaze stability.

METHODS

Participants

Emory University's Institutional Review Board approved the protocol, and all participants gave informed consent. Patients with vestibular hypofunction were referred from the Dizziness and Balance Center and healthy age-matched controls from a convenience sample. Vestibular function and diagnosis was determined by bithermal water caloric tests and computerized rotational chair testing in all subjects (8,9).

Research Protocol

We recorded eye and head motion using search coils in subjects during high-acceleration active and passive head impulses (3). The direction of the head impulse, either left or right, was predetermined using a randomly generated sequence. For active head impulses, subjects rotated their own head from midline. For passive head impulses, the subject's head was manually rotated with random timing and direction. Subjects were instructed to fixate a laser target, which was extinguished during head movement. Horizontal head velocity was measured online by a rate sensor (Watson Industries, Eau Claire, WI, USA) and impulses with velocities less than 120 degrees/s were discarded.

Data Capture and Processing

Data procedures have been detailed previously (10). Briefly, eye and head positions were measured using 6-ft magnetic field coils (CNC Engineering, Seattle, WA, USA). Search coils (Skalar, Delft, the Netherlands) were precalibrated against known rotations and additionally by having the subject fixate a target stepped ±20 degrees from midline. The system was 99% linear over a range of ±25 degrees. Data were stored on a desktop computer for offline analysis.

After collection, all data were filtered with a low-pass bandwidth finite impulse response at 100 Hz. Eye, gaze, and head velocities were obtained by differentiation of their respective position signals and accelerations by differentiation of their velocity signals using a 2-point central difference algorithm. Onset of head and eye movements was defined as the time at which head and eye velocities were greater than 2 SDs above baseline velocity. Distinctions between onset of head impulses and types of eye movements were confirmed using acceleration profiles (11). All data were processed using a customized interactive computer program (The Math Works, Natick, MA, USA).

Frequency of oculomotor strategies was determined for each head impulse type. Gaze position error (gaze amplitude minus initial target position) at the end of the ipsilesional head impulse was used to determine the effect of oculomotor strategy on gaze stability.

Definitions of Oculomotor Strategies

Vestibulo-Ocular Reflex

Slow component eye rotation in the opposite direction of the head impulse with latency less than 10 milliseconds after onset of head rotation (12).

Slow Vestibulo-Ocular Reflex

Slow component eye rotation during ipsilesional head impulse with latency greater than 10 milliseconds after onset of head rotation.

Compensatory Saccade

Rapid eye rotation with peak acceleration greater than 6,000 degrees/s^2 that occurred in the *opposite* direction of head *during* the head impulse.

Corrective Saccades

Rapid eye rotation with peak acceleration greater than 6,000 degrees/s^2 that occurred in the *opposite* direction of head *after* the head impulse. To distinguish these from spontaneous saccades, the latency of the corrective saccade had to occur within 170 milliseconds after head velocity reached zero (13).

Quick Phases

Rapid eye rotation with peak acceleration greater than 6,000 degrees/s^2 that occurred in the *same* direction of head *during* the head impulse.

Statistical Analysis

Tests for significant differences in age across groups were performed using analysis of variance. Tests for statistical significance for percent occurrences of types of eye movements by head impulse type (active versus passive) and diagnosis (UVH versus BVH) was performed with χ^2 goodness of fit. Independent t tests were used to compare gaze position error across types of eye movements and between UVH and BVH. Significance was set at $\alpha < 0.05$.

RESULTS

Participant Characteristics

No significant difference in age was found between the healthy subjects (n = 4; range, 34–83 yr) and subjects with UVH (n = 4; range, 35–81 yr) or BVH (n = 3; range, 53–73 yr; $F = 0.51$, $p = 0.62$). Two patients with BVH had autoimmune disorders as the underlying cause without signs of central nervous system involvement.

Oculomotor Strategies After Head Impulses

Healthy controls generated appropriate VOR eye movements after active and passive head impulses. After head impulses toward the healthy side, patients with UVH generated VOR eye rotations only, with latencies consistent with a normal VOR.

TABLE 1. *Frequency of oculomotor responses to ipsilesional head impulses by group*

	UVH (n = 4)		BVH (n = 3)	
Eye movement	Active head impulse (n = 63)	Passive head impulse (n = 71)	Active head impulse (n = 50)	Passive head impulse (n = 50)
Slow VOR only	8%	24%	6%	12%
Slow VOR with CS	25%	28%	56%	70%
Corrective saccades	40%	35%	56%	52%
QPs	30%	0	18%	4%

Percentages will not add to 100% because single trials often included multiple oculomotor response types.

BVH indicates bilateral vestibular hypofunction, head impulses in both directions; CS, compensatory saccade; UVH, unilateral vestibular hypofunction, ipsilesional head impulse.

Patients generated 3 different gaze-stabilizing eye movements with active or passive ipsilesional head impulses: slow VOR, compensatory saccades, and corrective saccades. Patients also made occasional quick phases (QP) that were noncompensatory (i.e., did not contribute to gaze stability).

Slow VOR

Although patients generated a slow VOR alone during some ipsilesional head impulses, most VOR occurred with a compensatory saccade (Table 1). There were no differences in peak velocity of slow VOR for either UVH or BVH during active (UVH 86 ± 18 degrees/s versus BVH 94 ± 26 degrees/s) or passive (UVH 67 ± 21 degrees/s versus BVH 78 ± 25 degrees/s) head impulses ($p > 0.05$).

Compensatory Saccades

Compensatory saccades occurred during both active and passive head impulses. Compensatory saccades were always associated with a slow VOR and then a corrective saccade occasionally. The occurrence of compensatory saccades after active head impulses varied considerably in subjects with UVH: 1 subject generated compensatory saccades with every active head impulse, whereas another subject never generated a compensatory saccade. In contrast, all subjects with UVH generated compensatory saccades in response to passive head impulses. Subjects with BVH used more compensatory saccades with a slow VOR than did subjects with UVH for active ($p < 0.01$) and passive head impulses ($p < 0.001$; Table 1).

Corrective Saccades

Corrective saccades were identified with equal frequency between both types of impulses for subjects with UVH (~36%, $p = 0.7$) and BVH (~56% $p = 0.8$; Table 1).

Quick Phases

Of 4 subjects with UVH, 2 exhibited QP only during active head impulses, which accounted for nearly half their eye movements. For 2 subjects with BVH, QP occurred more frequently during active than passive head impulses (Table 1).

Contribution of Eye Movements to Gaze Stability

Compensatory saccades had a strong influence on reducing gaze position error associated with active or passive head impulses. For patients with UVH, gaze position error was always lower when the VOR was accompanied by a compensatory saccade ($p = 0.007$, active; $p = 0.036$, passive; Table 2). In addition, gaze position error after a slow VOR with a compensatory saccade was lower for active versus passive impulses ($p = 0.017$). For patients with BVH, gaze position error during passive head impulses was similar whether the VOR was accompanied by compensatory saccades ($p = 0.3$). However, slow VOR with compensatory saccades did reduce gaze position error for active versus passive head impulses ($p = 0.0001$). Both UVH ($p < 0.001$) and BVH ($p < 0.001$) subjects had greater gaze position error when QP occurred during the active head impulse (Table 2). Gaze position error during active head impulses with QPs was greater for subjects with UVH than those with BVH ($p < 0.001$).

TABLE 2. *Gaze position error at the end of the ipsilesional active and passive head impulse*

	VOR with CS	VOR without CS	QPs
UVH			
Active	$3.4 \pm 1.6^{a,b}$	9.3 ± 4.1^{b}	$22.5 \pm 5.7^{a,b,c}$
Passive	$5.9 \pm 2.7^{a,c}$	9.1 ± 2.4^{c}	N/A
BVH			
Active	4.3 ± 1.9^{d}	N/A	$14.7 \pm 4.2^{d,e}$
Passive	7.1 ± 3.0^{d}	6.7 ± 1.1^{e}	$9.4 \pm 0.3^{d,e}$

[a–e]Letter combinations denote significant difference at $p < 0.05$.
N/A indicates the eye movement occurred in fewer than 2 trials.

DISCUSSION

Eye movements generated during rapid head impulses in patients with vestibular hypofunction can be categorized as compensatory (i.e., occur during the head impulse), corrective (i.e., occur after the head impulse), or noncompensatory (i.e., increase gaze position error). Although these eye movements have been previously identified, we have extended those findings by determining the distribution of these eye movements with respect to predictability of head impulse as well as describing their impact on gaze stability (7,14). Generally, we found patients used a slow VOR combined with a compensatory saccade to substitute for reduced vestibular function, although individual differences are pronounced. Our data suggest that 1) gaze position error is reduced when compensatory saccades occur and 2) recruitment of compensatory saccades seems related to the severity of the vestibular hypofunction. These findings suggest that vestibular rehabilitation should incorporate attempts to identify the patients' preferred gaze-stabilizing strategies and then develop exercises that lead to their recruitment, in particular, compensatory saccades.

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