

Recovery of Dynamic Visual Acuity in Bilateral Vestibular Hypofunction

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Objective: To determine the effect of vestibular exercises on the recovery of visual acuity during head movement in patients with bilateral vestibular hypofunction (BVH).

Design: Prospective, randomized, double-blinded study.

Setting: Outpatient clinic, academic setting.

Patients: Thirteen patients with BVH, aged 47 to 73 years.

Intervention: One group (8 patients) performed vestibular exercises designed to enhance remaining vestibular function, and the other (5 patients) performed placebo exercises.

Main Outcome Measures: Measurements of dynamic visual acuity (DVA) during predictable head movements using a computerized test; measurement of intensity of oscillopsia using a visual analog scale.

Results: As a group, patients who performed vestibular exercises showed a significant improvement in DVA ($P = .001$), whereas those performing placebo exercises did not ($P = .07$). Only type of exercise (ie, vestibular vs placebo) was significantly correlated with change in DVA. Other factors examined, including age, time from onset, initial DVA, and complaints of oscillopsia and disequilibrium, were not significantly correlated with change in DVA. Change in oscillopsia did not correlate with change in DVA.

Conclusions: Use of vestibular exercises is the main factor involved in recovery of DVA in patients with BVH. We theorize that exercises may foster the use of centrally programmed eye movements that could substitute for the vestibulo-ocular reflex.

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PATIENTS WITH VESTIBULAR HYPOFUNCTION complain of imbalance, head-movement-induced dizziness and head-movement-induced visual blurring (oscillopsia).¹⁻³ These problems are most severe in patients with bilateral hypofunction but are often significant in patients with unilateral vestibular loss as well. A number of randomized, prospective studies⁴⁻⁸ have documented that vestibular exercises improve postural stability and decrease subjective complaints of dizziness in patients with acute or chronic vestibular hypofunction. To date, there has been little research on the effect of vestibular exercises on dynamic visual acuity (DVA) during head movements or on oscillopsia. One randomized, prospective study by Herdman et al⁹ of patients with unilateral vestibular hypofunction found that vestibular exercises did im-

prove visual acuity during head movement. The relationship between DVA and oscillopsia is not clear because improvement in DVA did not correlate with improvement in oscillopsia as measured by the oscillopsia visual analog scale (oVAS). To our knowledge, the only publication that examined change in visual acuity during head movement in patients with bilateral vestibular hypofunction (BVH) was a retrospective study,¹⁰ but the authors did not specifically describe the change in DVA in the population reported. Furthermore, there have been no randomized, controlled studies that examined whether vestibular exercises improve visual acuity during head movements or decrease complaints of oscillopsia in individuals with BVH.

Decrements in visual acuity during head movement in patients with vestibular hypofunction are potentially serious prob-

lems. This deficit could contribute to decreased activity level, avoidance of driving with resultant diminished independence, and, ultimately, limited social interactions and increased isolation. Oscillopsia occurs because of inadequate vestibulo-ocular reflex (VOR) gain and suggests that compensation for the vestibular loss has not occurred. This study examined the effect of an exercise intervention on visual acuity during head movement in patients with BVH. We hypothesized that (1) patients performing vestibular exercises would have improved visual acuity during head movement compared with patients performing placebo exercises, (2) there would be no correlation between DVA and the patients' subjective complaints of oscillopsia, and (3) improvement in DVA would be reflected by changes in residual vestibular function as indicated by an increase in VOR gain.

METHODS

PATIENTS

Patients included in the study had been referred to the laboratory for assessment from the clinical practice of 2 of the investigators (S.J.H. and R.J.T.). We assessed vestibular function in these subjects by using caloric and computerized rotary chair tests.^{11,12} For rotary chair testing, we used step velocities at 60°/s and 240°/s rotations with electrooculography. The criteria for patients with BVH included refixation saccades made in response to unpredictable head thrusts to the right and left, a gain of less than 0.1 on the rotary chair step test, and a peak slow-phase eye movement of less than 5°/s during irrigation of each ear on bithermal water caloric testing.^{11,12} Vestibular function, as measured by rotary chair and by small-amplitude, high-acceleration unpredictable head thrusts, was assessed in a subset of patients before and after the course of vestibular exercises. Data on age, time from onset, degree of deficit (complete vs incomplete vestibular hypofunction), duration of exercises, and complaints of oscillopsia and of disequilibrium were collected on all subjects. Only patients with BVH and abnormal visual acuity during head movements (DVA) for their age were included. Abnormal DVA was defined as DVA greater than the mean plus 2 SDs of the DVA of healthy subjects of the same age.¹³ Exclusion criteria for participants included the presence of nystagmus in room light and visual acuity with the head stationary of worse than logMAR 0.500 (where logMAR indicates the logarithm of the minimal angle of resolution) (because decrements with head movement could potentially be subject to a ceiling effect); minors and people incapable of understanding the purpose of the study were also excluded. Informed consent was obtained in compliance with the University of Miami (Miami, Fla) or Emory University (Atlanta, Ga) institutional review board protocol from October 2000 to November 2003.

DVA TEST PROCEDURE

Details of the DVA test procedure have been reported previously.^{13,14} Briefly, the subject moved the head actively (predictable DVA). An optotype (the letter "E") was displayed on the monitor when the subject's head velocity was between 120°/s and 180°/s. A computer-generated program altered the orientation of the "E" randomly. The computer was set so the letter appeared during only the rightward or leftward portion of a horizontal head movement. The optotype size was changed decrementally such that changes in visual acuity from line to line

were equivalent to 0.1 logMAR.¹⁵ There were 5 optotypes presented for each acuity level. When the subject indicated the direction of orientation of the "E," the subject's response was recorded and the next trial begun. The trial was scored as an error if the subject incorrectly identified the direction of the orientation of the "E" or if the subject did not know the orientation after viewing the optotype 5 times. When the subject incorrectly identified the orientation of the "E" for all optotypes presented at a particular acuity level, the test was stopped. The total number of errors in identifying the orientation of the optotypes was recorded. The test-retest reliability of this computerized test has an intraclass correlation coefficient of 0.92 in patients with vestibular hypofunction.

TEST PROTOCOL

The test was performed first with the subject's head stationary (static visual acuity). The series of optotypes was displayed and scored. The rate sensor was placed on the subject's forehead and oriented to detect horizontal movement of the head. All subjects performed a practice test in which optotypes were presented during predictable head movements to the right. Previous research¹³ has demonstrated that a practice trial is necessary to familiarize the subject with the test and to minimize a learning effect before data are collected. Data were collected separately for display of the optotype during predictable rightward and leftward head movements. We calculated DVA by counting the total number of errors in identifying the orientation of the optotype and subtracting that number from the static visual acuity. Raw scores were then converted to a logMAR score. The DVA was measured prior to entry into the study and at 2-week intervals after initiation of either control or vestibular exercises.

MEASUREMENT OF OSCILLOPSIA AND DYSEQUILIBRIUM

A 10-cm visual analog scale (VAS) was used to assess the degree of oscillopsia (oVAS) (perception of visual blurring) and disequilibrium (dVAS) (perception of being off-balance) in the patients. The VAS used a 10-cm line oriented vertically with "no symptoms" corresponding to the bottom of the line and "worst possible symptoms" to the top of the line. Patients were instructed to place a mark on the 10-cm vertical line that corresponded to the intensity of their symptoms. For oscillopsia, one end of the scale was anchored with "No difficulty seeing clearly at all (normal)," and the other end was anchored with "The worst it could be." For disequilibrium, one end of the scale was anchored with "I feel perfectly steady," and the other end was anchored with "The worst it could be." Patients were asked to rate their perception first while they were sitting and then while they were walking. The difference in symptom intensity between the sitting and walking conditions was used as the oVAS or dVAS score. Thus, the expression of the oVAS and dVAS scores was similar to the expression of the DVA score, which is the difference between visual acuity with the head stationary and with the head moving. The test-retest reliability for each of the perception measurements, $r=0.65$ (intraclass correlation coefficient 1,1), was based on 25 patients with unilateral vestibular hypofunction or BVH.

EXERCISES

Patients were assigned randomly to either the vestibular exercise or placebo exercise group. The randomization schedule was generated using a computer program for 2-sample randomization. If a participant who had been enrolled in the study was dropped from the study, the next participant enrolled took that

Table 1. Progression of Vestibular Exercises*

Exercise Weekly Progression*	Duration	Frequency
<i>Adaptation:</i> X1 with target held in hand and also with target at distance, horizontal and vertical head movements	1 min each exercise	4 Times daily; total time, 20 min
<i>Adaptation:</i> X1 with target held in hand and also with target at distance, horizontal and vertical head movements <i>Substitution:</i> Eye-head movements between 2 targets with emphasis on seeing clearly, horizontal and vertical eye-head movements	1-2 min each exercise	5 Times daily; total time, 30-40 min
<i>Adaptation:</i> X1 with target held in hand and also with target at distance, horizontal and vertical head movements; X1 with checkerboard with target placed in center held in hand, horizontal head movements <i>Substitution:</i> Eye-head movements between 2 targets; imaginary target paradigm	1 min each exercise	4 Times daily; total time, 28 min
<i>Adaptation:</i> X1 with checkerboard with target placed in center held in hand; X2 with target held in hand; both horizontal and vertical head movements <i>Substitution:</i> Eye-head movements between 2 targets; imaginary target paradigm	1 min each exercise	4 Times daily; total time, 28 min
<i>Adaptation:</i> X1 with target held in hand; horizontal and vertical head movements; X1 with checkerboard with target placed in center held in hand, horizontal and vertical head movements; X2 with target held in hand, horizontal and vertical head movements <i>Substitution:</i> Eye-head movements between 2 targets; imaginary target paradigm	1 min each exercise	4 Times daily; total time, 36 min

*X1 and X2 are the names given to the exercises.

group assignment. The sequence was not concealed from the investigator who obtained consent from the subjects and supervised the exercises (S.J.H.). The sequence was concealed from the participant and from the investigator who performed the outcome measures. The group assignment (vestibular exercise or placebo exercise) was concealed from the participants and from the investigator who performed the outcome measures.

The vestibular exercise group practiced exercises that consisted of adaptation exercises and eye-head exercises to targets (**Table 1**), which were designed to improve gaze stability.¹⁶ They also performed gait and balance exercises. The placebo exercise group performed exercises designed to be "vestibular-neutral." These placebo exercises consisted of saccadic eye movements with the head stationary while viewing a Ganzfeld (a large featureless surface). The placebo exercise group also performed gait and balance exercises, although exercises that specifically incorporated head movements were avoided. Each group had the same contact time with the investigators. All patients were seen in the clinic on a weekly basis to review and update their exercise program. During the clinic visits, they also were taught an exercise program that they were to follow at home. All patients were asked to perform the exercises 4 to 5 times daily for a total of 20 to 40 min/d plus 20 min/d of balance and gait exercises.

The progression of exercises was developed based on clinical experience with patients who were in the acute and subacute stages following onset of vestibular dysfunction. These patients had considerable symptomatic complaints associated with head movement. Thus, the change in exercises from week to week represents a conservative progression. Patients followed the program unless they reported difficulties related to an increase in their complaints of dizziness. If the exercises exacerbated the patient's complaints of dizziness, the patient was instructed to work through the dizziness if possible. If this was not possible, the first step was to modify the exercises by decreasing the head (or eye) velocity to a more tolerable level. If that did not relieve symptoms, the frequency of performing the exercises was decreased from 5 times daily to 3 times daily for 3 days, then increased to 4 times daily for next 3 days. Patients were given a calendar to record exercise compliance and were

instructed to bring the calendar with them each week. An individual was considered compliant if he or she performed more than 50% of the exercises. At the end of 6 weeks, subjects in the placebo exercise group were started on a program of vestibular exercises.

DATA ANALYSIS

Baseline differences between groups for age, initial DVA score, and initial complaint of oscillopsia and of disequilibrium while walking were determined using analysis of variance.

To determine if vestibular rehabilitation improved DVA, repeated-measures univariate analyses of variance were performed with time (pretherapy and posttherapy) as the repeated factor and DVA score as the variable of interest. Appropriate post hoc statistics were performed if a significant main effect or interaction was found ($P < .05$). We defined individual improvement in DVA as a change in DVA greater than the mean plus 2 SDs of the test-retest variability determined from a separate, representative group of subjects with vestibular hypofunction. In addition, we compared final DVA with reference range values of DVA by age for each subject.

To identify factors associated with rehabilitation outcome (ie, change in DVA score), we calculated Pearson correlation coefficients between change in DVA score and potential predictor variables, including exercise group, age, duration of therapy, oVAS, dVAS, and initial DVA score. We did not include time from onset as a variable because of the difficulty identifying exact time of onset for all subjects, which resulted in a loss of data. We performed statistical analysis to identify outliers based on residuals being 2 SDs outside the mean.

In an attempt to identify mechanisms underlying a change in DVA score, we examined the change in VOR gain from before and after therapy. We defined a significant improvement as an increase in VOR gain in response to 240°/s step rotation. The change in gain had to be greater than the mean plus 2 SDs of the test-retest variability determined for a separate population of patients with vestibular hypofunction. Because of limited data, we did not perform formal statistical analysis on this variable.

Table 2. Experimental and Control Groups

Age, y/Sex	Time From Onset, mo	oVAS Pretherapy, cm	dVAS Pretherapy, cm	DVA Pretherapy, logMAR	DVA Posttherapy, logMAR
Experimental Group					
70/M	48	1.1	4	0.279	0.120
62/M	NA	7.6	1.1	0.260	0.210
73/F	NA	0.0	3.5	0.418	0.212
53/F	84	0.2	0.0	0.194	0.139
47/F	NA	3.2	9.2	0.320	0.083
66/M	54	3.2	8.6	0.231	0.160
73/M	10	0.0	0.1	0.343	0.262
65/M	8.5	0.0	0.1	0.450	0.301
Control Group					
62/M	NA	7.6	1.1	0.296	0.260
73/F	NA	0	3.9	0.428	0.419
46/M	0.5	NA	NA	0.328	0.168
72/F	3	NA	NA	0.678	0.629
65/M	7	0	4	0.463	0.450

Abbreviations: DVA, dynamic visual acuity; dVAS, disequilibrium visual analog scale; logMAR, logarithm of the minimal angle of resolution; NA, data were not obtained from patient; oVAS, oscillopsia visual analog scale.

Table 3. Subject Characteristics*

Characteristic	Experimental Group (n = 8)	Control Group (n = 5)	P Value†
Age, y	63.6 (9.4)	63.6 (10.8)	>.99
Time from onset, mo	40.6 (32.3)	3.5 (3.3)	.10
Initial DVA; predictable, logMAR	0.312 (0.089)	0.438 (0.151)	.08
Initial complaint of oVAS while walking	1.0 (1.4)	2.5 (4.4)	.36
Initial complaint of dVAS while walking	3.3 (3.9)	3.0 (1.6)	.92
Duration of exercises, wk	5.1 (1.5)	6.0 (1.6)	.35

Abbreviations: DVA, dynamic visual acuity; MAR, minimal angle of resolution.

*Data are given as mean (SD).

†Level of significance of between-group differences.

RESULTS

SUBJECT CHARACTERISTICS

Data were collected from October 2000 to November 2003. Fourteen patients with BVH initially consented to the study and were randomly assigned to either the experimental group or the control group. One control patient was dropped from the study because she was moving her head during the exercises. The remaining subjects were in the experimental (n=8) or control group (n=5) (**Table 2**). None of the patients in either group were receiving treatment with vestibular suppressant medications during the study. There were no adverse events or effects in the intervention group.

Within the limits of the small sample size and statistical power of the design, there were no differences between groups for age, initial DVA scores, initial complaints of oscillopsia or disequilibrium, or duration of

exercises ($P>.05$, **Table 3**). There was no difference in exercise compliance between the 2 groups (range, 50%-100%) based on weekly calendars.

Outlier analysis revealed that the change in DVA score for 1 control subject (control subject 3) was greater than 2 SDs outside the mean; thus, all further analyses were performed without this subject.

EFFICACY OF VESTIBULAR REHABILITATION ON DVA SCORE

With the outlier removed, there was a significant interaction of group and time (Wilks $\Lambda=0.576$; $F_{1,10}=7.371$; $P=.02$) in addition to significant main effects of time ($F_{1,10}=17.372$; $P=.002$) and group ($F_{1,10}=10.238$; $P=.009$). Post hoc analysis revealed significant group differences in pretherapy DVA scores ($F_{1,10}=4.856$; $P=.052$) and posttherapy DVA scores ($F_{1,10}=16.425$; $P=.002$). Repeated-measures univariate analysis of variance revealed that the control group did not change significantly from pretherapy (mean [SD], 0.466 [0.158]) to posttherapy DVA score (0.439 [0.151]; $F_{1,3}=8.122$; $P=.07$) (**Figure 1**). The experimental group improved significantly from pretherapy (0.312 [0.089]) to posttherapy DVA score (0.185 [0.072]; $F_{1,7}=25.622$; $P=.001$). As individuals, 7 of 8 subjects in the experimental group had an improvement in DVA. The DVA of 5 of the 8 subjects also returned to reference range for age. As individuals, only 1 of the 5 control subjects had an improved DVA score, although not to within reference range for age. The improvement in DVA in the experimental group occurred within 5.1 (1.5) weeks.

FACTORS CORRELATED WITH CHANGE IN DVA SCORE

To explore the factors that contributed to rehabilitation outcome, we calculated Pearson correlation coefficients. The correlations between change in DVA (pretherapy DVA vs posttherapy DVA) and group, age, oVAS,

dVAS, and pretherapy DVA were examined. Only type of exercise (ie, group) was significantly correlated ($r=0.65$; $P=.02$) with change in DVA score. No other factor measured (age, complaints of oscillopsia or disequilibrium, or initial DVA) was significantly correlated with change in DVA ($r=-0.03$ to 0.36 ; $P>.05$).

CHANGE IN VESTIBULAR FUNCTION WITH VESTIBULAR REHABILITATION

None of the participants had a significant change in VOR gain, based on our criteria, as measured using step rotary chair test at either 60°/s or 240°/s rotation in the dark (**Figure 2**). All changes in VOR gain were within 2 SDs of the test-retest variability determined on a separate group of patients.

COMMENT

The results of this study show that, as a group, individuals with BVH who performed vestibular exercises had a significant improvement in gaze stability as measured by the computerized DVA test. As clinicians, we are concerned about the potential of individual patients to improve; therefore, it is important to note that most patients (7 of 8) who performed the exercises showed significant improvement. In fact, DVA scores in 5 of 8 participants had values that returned to reference range for age. In contrast, the DVA score in only 1 control subject improved, and the score did not return to reference range for age. Furthermore, the analysis showed that only the vestibular exercises were significantly correlated with the improvement in DVA. It is possible that some other factors that we did not measure may have contributed to recovery. For instance, we were unable to determine the exact date of onset of the BVH in several patients. Therefore, it is not known whether time from onset until the initiation of exercises is an important factor in response to the exercises. In addition, we did not examine the usual physical activity levels of the subjects in this study, which may also contribute to recovery. It is probable that combinations of factors or patient characteristics may alter the potential for recovery. Studies with larger numbers of subjects are needed to resolve these issues.

Of interest are the factors that were not correlated with recovery of DVA, especially age. There are conflicting reports on the effect of age on recovery following unilateral vestibular deficits. One study¹⁷ suggested that older individuals are less likely to show recovery with rehabilitation, whereas others indicated that age is not a factor.^{9,18} Our data suggest that vestibular exercises are effective in improving DVA regardless of the subject's age. It is interesting to note that of the 10 subjects in the study who were older than 60 years, only those performing the vestibular exercises showed an improvement in DVA. Of the 3 subjects who were younger than 60 years, 1 was in the control group and was the only person in the control group who showed an improvement in DVA. This subject, however, was also observed during the acute stage after onset (<2 weeks), and it is possible that younger age plus a more acute onset may be a factor in natural

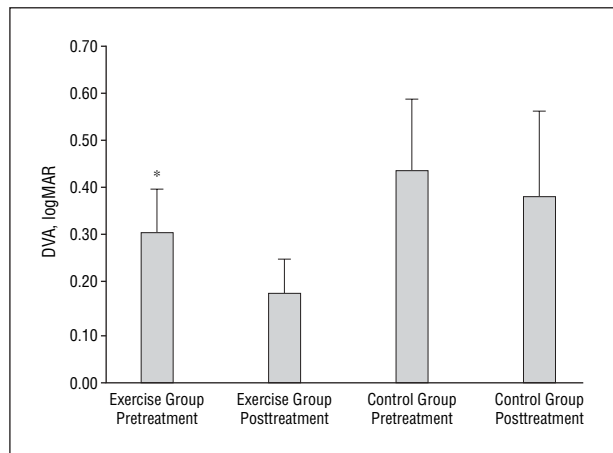


Figure 1. Note the significant improvement in dynamic visual acuity (DVA) in the vestibular exercise group but not in the control group (includes the outlier). LogMAR indicates logarithm of the minimal angle of resolution. Asterisk indicates $P=.001$. The bar height is the mean, and the line is the SD.

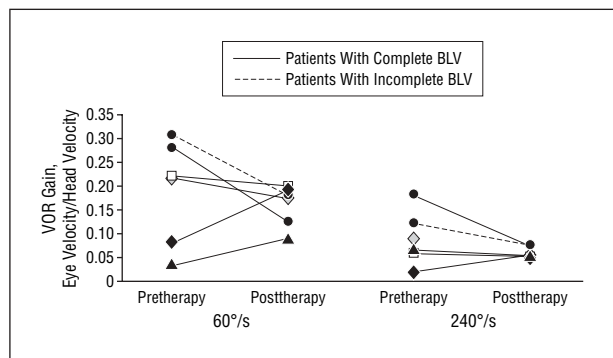


Figure 2. Change in slow-phase eye velocity (SPEV) gain with exercises in individual patients with bilateral vestibular hypofunction. In most patients, there was no change in SPEV gain following a course of vestibular exercises as measured using 60°/s or 240°/s rotations in the dark. One person seemed to have an increase in gain of 0.1 during 60°/s rotation. However, the same person had no change in gain during 240°/s rotation. Solid lines indicate individual patients with complete bilateral vestibular loss (BVL) as indicated by no response to ice water irrigation of the horizontal canals; dotted lines indicate individual patients with incomplete BVL; and VOR, vestibulo-ocular reflex.

recovery. More studies are needed because there were too few subjects in our study to determine this.

We do not think that this improvement in DVA reflected a change in vestibular function. We found no evidence of a change in VOR gain as measured by rotary chair testing. The rotary chair test of vestibular function is somewhat limited, however, in that it assesses only the function of the horizontal canals and only at a narrow velocity range, so we cannot be fully confident in this conclusion. A second mechanism for improvement in DVA may be the use of central preprogramming of other types of eye movements to improve gaze stability.¹⁹⁻²¹ We have previously demonstrated that subjects with unilateral vestibular hypofunction have better visual acuity during self-generated head rotation than during unpredictable head movements.¹⁴ This suggests that central programming of eye movements may contribute to gaze stability during predictable head movements. Centrally programmed eye movements have been described in patients with peripheral vestibular hypofunction and in

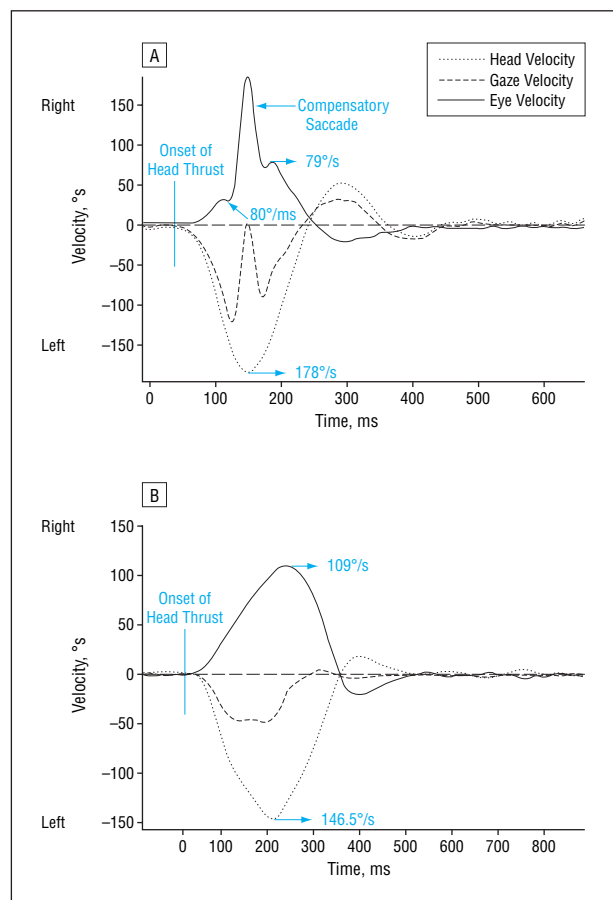


Figure 3. Eye movements that may substitute for the loss of vestibular function during self-generated horizontal head movements (head thrust) include preprogrammed or compensatory saccades (A) and high-velocity, slow-phase eye movements (B). Preprogrammed saccades occur during the head movement itself and are in the opposite direction of the head thrust. The latency of onset of preprogrammed saccades (80 ms in this example) indicates that these are not voluntary eye movements. High-velocity, slow-phase eye movements (A) are also made in the direction that is opposite the direction of the head thrust. The velocity of these eye movements (108°/s in this example) is higher than that which would be expected if these were visually induced smooth-pursuit eye movements. In both A and B, the direction of head movement is to the left with the compensatory eye movement to the right.

clude preprogrammed saccades that occur during the head movement as well as high-velocity, slow-phase eye movements (velocities of 80°/s to 120°/s)²⁰ (**Figure 3**).

The relationships between DVA and complaints of oscillopsia are not clear.^{22,23} We found no relationship between improvement in DVA and improvement in the patients' perception of oscillopsia while walking. This is similar to the results of our earlier study, which demonstrated no relationship between DVA and subjective complaints of oscillopsia in subjects with unilateral vestibular hypofunction.^{9,22} There are several possible explanations for these results. First, DVA was measured during horizontal head movements. In contrast, oscillopsia may reflect the inability to stabilize eyes during the vertical head movements occurring while walking. Second, the subjective complaint of oscillopsia may be more related to the patient's tolerance for retinal slip and to the patient's perception of the amount of control he or she has over the vestibular disorder than to actual clar-

ity of vision. This was suggested by Grunfeld et al,²³ who examined patients with bilateral vestibular loss. Thus, patients may demonstrate improvement in the objective measure of visual acuity during head movements but still have significant complaints of oscillopsia. Finally, the complaint of visual blurring during head movement may reflect the patients' experiences during unpredictable head movements more than during predictable head movements.

In conclusion, the results of this study suggest that the use of specific vestibular rehabilitation exercises facilitates the recovery of gaze stability during head movement in patients with BVH. The results are remarkably similar to our previously reported findings⁹ in patients with unilateral vestibular hypofunction. The recovery of DVA is relatively rapid, occurring in approximately 5 weeks of exercises. That age was not a factor in this recovery suggests that older patients will benefit from the use of vestibular rehabilitation. Our study also suggests that recovery of DVA is due to mechanisms other than improvement in residual vestibular function.

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Author Contributions: Drs Herdman, Hall, Schubert, and Tusa had full access to all data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. **Study concept and design:** Herdman and Tusa. **Acquisition of data:** Herdman, Hall, Schubert, and Das. **Analysis and interpretation of data:** Herdman, Hall, Schubert, and Tusa. **Drafting of the manuscript:** Herdman. **Critical revision of the manuscript for important intellectual content:** Herdman, Hall, Schubert, Das, and Tusa. **Statistical analysis:** Schubert. **Obtained funding:** Herdman. **Administrative, technical, and material support:** Hall, Das, and Tusa.

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