

Role of Central Preprogramming in Dynamic Visual Acuity With Vestibular Loss

Susan J. Herdman, PT, PhD; Michael C. Schubert, PT, MS; Ronald J. Tusa, MD, PhD

Objective: To determine the contribution of central preprogramming of eye movements to dynamic visual acuity (DVA) during head movement in patients with vestibular hypofunction.

Study Design: Prospective, clinical study.

Setting: Tertiary care, academic hospitals.

Participants: Twenty-six healthy subjects and 20 patients with unilateral (UVL) and 7 with bilateral vestibular loss (BVL) (age range, 20-86 years).

Interventions: Diagnostic interventions, including caloric and rotational chair testing.

Main Outcome Measure: Measurements of DVA during predictable (DVA-predictable) and unpredictable (DVA-unpredictable) head movements using a computerized test.

Results: There was a difference between DVA-predictable and DVA-unpredictable scores in all groups ($P < .02$). The difference between DVA-predictable and

DVA-unpredictable scores for the BVL group was significantly greater than that for the other groups ($P < .005$). Age was a significant factor in DVA-unpredictable scores for the healthy subjects ($P < .001$) and UVL group ($P < .02$). Comparisons of DVA between groups were significant ($P < .03$), with the following exceptions: UVL group for head movements toward the unaffected side for DVA-predictable and DVA-unpredictable scores, compared with healthy subjects, and UVL group for head movements toward the affected side for DVA-predictable scores, compared with the BVL group.

Conclusions: Unpredictable head movements cause a greater decrement in visual acuity than do predictable head movements. This suggests that central programming of eye movements and/or efference copy contributes to gaze stability during predictable head movements in healthy subjects and patients with vestibular hypofunction. Patients with BVL use central programming of eye movements to maintain gaze stability more than do healthy subjects or patients with UVL.

Arch Otolaryngol Head Neck Surg. 2001;127:1205-1210

From the the Department of Rehabilitation Medicine (Dr Herdman and Mr Schubert), the Department of Neurology (Dr Tusa), and Yerkes Research Center (Dr Tusa), Emory University, Atlanta, Ga.

MOVEMENT of the head can cause significant retinal slip. When retinal slip exceeds $2^\circ/\text{s}$, degradation of visual acuity occurs.¹⁻³ Various mechanisms that may augment the vestibulo-ocular reflex (VOR) include the pursuit/optokinetic system, the cervico-ocular reflex (COR), efference copy, central programming of eye movements, and anticipatory intent.⁴⁻¹¹ The contribution of these mechanisms to gaze stability was studied primarily by comparing the gain of the compensatory eye movements during active and passive head movements in which the direction, amplitude, and temporal quality of head movement were predictable.^{3,6,12-14} Other studies have shown that there is little difference of the gain of the VOR in the light (vi-

sual-VOR) under conditions in which the active and passive head movements are predictable.^{3,15}

We chose to examine the role of central programming of eye movements and efference copy in maintaining gaze stability using a functional measure, ie, dynamic visual acuity (DVA). We examined the ability of these compensatory mechanisms to augment the VOR by comparing visual acuity during predictable (active) and unpredictable (passive) head rotation. We hypothesized the following: (1) visual acuity during head rotations in which the direction and timing of the head movement was unpredictable (DVA-unpredictable) would be significantly worse than visual acuity when the head movement was predictable (DVA-predictable) for healthy subjects, patients with unilateral vestibular

SUBJECTS AND METHODS

SUBJECTS

Healthy subjects were recruited from among laboratory personnel and family members of patients at 2 tertiary care academic hospitals. Informed consent was obtained in compliance with the institutional review board protocols of the University of Miami, Miami, Fla, and Emory University, Atlanta, Ga. 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.). Vestibular function was assessed in these subjects using caloric and vertical-axis rotational chair tests.^{17,18} Subjects were excluded from the healthy group if they had abnormal results of vestibular function tests or a history of vertigo. Bilateral and unilateral vestibular deficits were identified based on the clinical evidence of an abnormal vestibular response (positive findings of the head-thrust test)¹⁹ and the results of vestibular testing (rotational chair or caloric tests).^{17,18} For the head-thrust test, the patient's head was first pitched forward approximately 30°, and the patient was asked to fixate on a stationary target. The patient's head was moved through a small amplitude, first slowly and then rapidly, in the yaw plane. The direction of the rapid head impulses was randomized to be unpredictable. Patients underwent testing using a near and a far target with appropriate visual correction. When the head thrust resulted in a corrective saccade to refixate the target, the test result was considered positive for the side of the head thrust (indicating vestibular hypofunction). We used step-velocity rotational chair testing at rotations of 60°/s and 240°/s with electronystagmography. Unilateral vestibular deficits were defined by at least a 25% difference in slow-phase eye velocity between right and left sides during the caloric or rotational chair test (at a chair speed of 240°/s). Bilateral vestibular loss was defined as less than 5° of slow-phase eye velocity in response to bithermal caloric tests, including ice water, and a gain (peak slow-phase eye velocity/chair velocity) of less than 0.2 on results of rotational chair testing. We defined no response to ice water irrigation unilaterally or bilaterally as a complete loss of vestibular function unilaterally or bilaterally, respectively, recognizing that this represents no function in the horizontal canals, as it is not possible to measure function of the remainder of the labyrinth by using caloric irrigation.

INSTRUMENTATION

An optotype (the letter *E*) is displayed on the monitor when the subject's head velocity ranges from 120°/s to 180°/s. A computer-generated program alters the orientation of the *E* randomly. The computer can be set so the letter appears during only the rightward or leftward portion of a horizontal head movement. There are 5 trials at each acuity level. The optotype size is changed decrementally so changes in

visual acuity from line to line are equivalent to 0.1 logarithm of the minimal angle of resolution (LogMAR).²⁰ When the subject indicated the direction of orientation of the *E*, the subject's response was recorded, and the next trial was 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. Data are the number of errors in identifying the orientation of the optotype. Details of the test procedure have been reported previously.¹⁶

TEST PROTOCOL

The test was performed first with the subject's head stationary. The series of optotypes was displayed and scored. The rate sensor was then placed on the subject's forehead and oriented to detect horizontal movement of the head. All subjects then performed a practice trial in which optotypes were presented during active head movements to the right, to familiarize the subjects with the test and to minimize a learning effect before data were collected. Data were then collected separately for display of the optotype during active rightward and leftward head movements (DVA-predictable). For DVA during unpredictable head movements, subjects then performed a practice trial in which their heads were moved by one of the investigators (M.C.S.) to the right and left in a random order. The optotype was displayed only when the subject's head velocity ranged from 120°/s to 180°/s. After the practice trial, data were collected separately for display of the optotype during rightward (leftward head movements did not result in display of the optotype) and leftward (rightward head movements did not result in display of the optotype) head movements (DVA-unpredictable). Dynamic visual acuity was calculated by counting the total number of errors in identifying the orientation of the optotype and subtracting that value from the static visual acuity. Raw scores were then converted to a LogMAR score.

STATISTICAL ANALYSES

Comparison of DVA-predictable and DVA-unpredictable scores within each group was examined using the *t* test. The relationship of age to DVA scores in healthy subjects and in subjects with vestibular deficits and the relationship of time from onset to DVA scores in the patient groups were determined using regression analysis. Correlation of degree of deficit to DVA scores in patients with UVL was determined using point-biserial correlation. Between-group comparisons were performed using analysis of covariance with least squares difference post hoc testing. Level of significance for all analyses was $P < .05$. Data for age and time from onset are presented as mean \pm 1 SD; for DVA, mean \pm 2 SD.

loss (UVL), and patients with bilateral vestibular loss (BVL); (2) patients with BVL would have a significantly greater decrement in DVA-unpredictable scores compared with all other groups; and (3) age would be a factor in DVA-unpredictable scores as it was for DVA-predictable scores.¹⁶

RESULTS

HEALTHY SUBJECTS

Twenty-six healthy subjects (mean age, 39.6 \pm 15.5 years; range, 20-69 years) were studied. Mean DVA-

predictable score was 0.030 ± 0.027 LogMAR; mean DVA-unpredictable score, 0.045 ± 0.044 LogMAR. The difference between DVA-predictable and DVA-unpredictable scores was significant at $P = .02$.

Regression analysis showed a significant relationship between age and DVA-unpredictable score ($F = 15.18$; $P < .001$) (**Figure 1**). Results indicated that 40% of the variance of DVA-unpredictable score could be accounted for by age. The relationship of age to DVA-predictable score approached significance ($F = 3.59$; $P = .07$).

PATIENTS WITH VESTIBULAR DEFICITS

UVL Group

Twenty patients with UVL were studied (mean age, 66.7 ± 13.1 years; range, 33-86 years). **Table 1** gives the characteristics of the subjects with UVL. Mean time from onset was 7.3 ± 8.0 months. Only 3 of the patients were less than 1.5 months from onset at the time of the study. In 1 patient, caloric testing could not be performed because of a tympanic membrane perforation. Time from onset was not a significant factor for DVA-predictable or DVA-unpredictable scores (Table 1). There was no correlation between degree of deficit and DVA scores (Table 1). There was a significant difference between DVA-predictable and DVA-unpredictable scores for head movements toward the affected and unaffected sides (**Table 2**).

BVL Group

Seven patients with BVL were studied (mean age, 63.4 ± 12.7 years; range, 43-75 years) (Table 1). Mean time from onset was 30.6 ± 38.5 months. Time from onset and degree of deficit were not significant factors in DVA in this small group. There was a significant difference between DVA-predictable and DVA-unpredictable scores (t test, $P = .004$) for patients with BVL (Table 2).

Effect of Age on DVA Score

We examined the possible relationship between DVA score and age in patients with UVL and BVL separately (Table 2). Regression analysis showed a significant relationship between age and DVA-predictable and DVA-unpredictable scores for patients with UVL for head movements toward the unaffected and affected sides (**Figure 2**). The relationship between age and DVA score was not significant for patients with BVL.

Differentiation Among Groups

Because age was a factor in DVA for the healthy subjects and for patients with UVL, an analysis of covariance was used to compare DVA-predictable and DVA-unpredictable scores across groups. For DVA-predictable scores, all pairwise comparisons were significant ($P < .03$) except for healthy subjects compared with the UVL group on movements toward the unaffected side and the BVL group compared with the UVL group on movements toward the af-

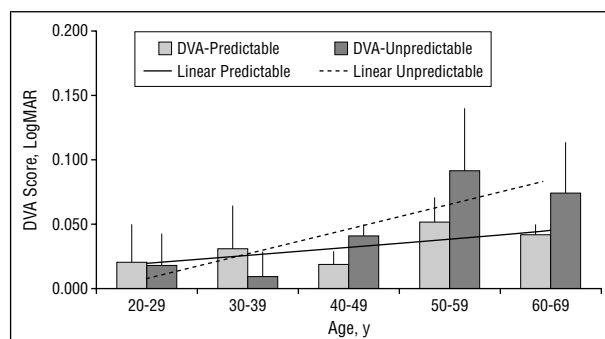


Figure 1. There was a significant relationship between age and scores for dynamic visual acuity (DVA) during predictable (DVA-predictable) and unpredictable (DVA-unpredictable) head movements, shown here as binned data. Data are given as mean \pm 2 SD; trend lines are shown. LogMAR indicates logarithm of the minimal angle of resolution.

ected side. For DVA-unpredictable scores, all pairwise comparisons were significant ($P < .01$) except for healthy subjects compared with the UVL group on movements toward the unaffected side. Comparison of the difference between DVA-predictable and DVA-unpredictable scores across groups showed that the BVL group differed from all other groups ($P < .005$) (**Figure 3**).

COMMENT

Several studies have shown that there is little difference in visual-VOR gain during active and passive head rotations in healthy subjects.^{3,15} In contrast, our results show a significant difference between DVA-predictable and DVA-unpredictable scores in healthy subjects and in patients with UVL and BVL, with poorer acuity during DVA-unpredictable movements. The most likely explanation for the differences between our findings on DVA and those of other studies on visual-VOR gain is that the active and the passive head rotations used in earlier studies were predictable in direction and tempo.^{3,15} This means that the subjects could augment the VOR using other mechanisms for gaze stability. We compared DVA during predictable head movements with DVA during unpredictable head movements. This is a crucial consideration because in normal daily activities, head movements do not always occur in a predictable manner but are random.^{1,14,21,22} It is important, therefore, to examine gaze stability using unpredictable passive head movements to establish the functional degradation of visual acuity that may happen in a natural environment.

MECHANISM UNDERLYING DIFFERENCE IN DVA-PREDICTABLE AND DVA-UNPREDICTABLE SCORES ACROSS GROUPS

Although the VOR is the primary reflex that stabilizes the eyes during head movement, several other mechanisms have the potential to contribute to gaze stability. These mechanisms may also contribute to gaze stability when there is loss of vestibular function. When the head moves slowly or at low frequencies, the pursuit/optokinetic system may be sufficient to maintain gaze stability.^{4,5} Another mechanism that may contribute to gaze stability dur-

Table 1. Subject Characteristics*

Deficit	Age, y	Degree of Deficit	Cause	Time From Onset, mo	DVA Score, LogMAR			
					Affected Side		Unaffected Side	
					Predictable	Unpredictable	Predictable	Unpredictable
UVL	33	1	Neuronitis	0.75	0.127	0.159	0.056	0.056
UVL	48	0	Neuronitis	5	0.112	0.127	0.076	0.076
UVL	49	1	Neuronitis	10	0.097	0.159	0.112	0.097
UVL	58	0	s/p AN	6	0.301	0.484	0.247	0.273
UVL	59	1	Neuronitis	6	0.127	0.143	0.097	0.143
UVL	59	0	s/p AN	4.25	0.398	0.439	0.112	0.143
UVL	62	0	s/p AN	0.5	0.176	0.337	0.067	0.247
UVL	63	0	Neuronitis	5	0.056	0.076	0.036	0.056
UVL	66	0	Neuronitis	5.75	0.247	0.357	0.127	0.112
UVL	67	0	Neuronitis	4	0.112	0.319	0.097	0.127
UVL	72	1	Neuronitis	5	0.159	0.198	0.159	0.143
UVL	72	0	Neuronitis	3	0.398	0.337	0.301	0.319
UVL	73	1	Neuronitis	12	0.198	0.159	0.112	0.159
UVL	75	0	Neuronitis	1.75	0.247	0.301	0.159	0.273
UVL	77	1	Neuronitis	36	0.273	0.418	0.301	0.461
UVL	78	1	Neuronitis	2	0.562	0.678	0.418	0.377
UVL	78	1	AN	12	0.461	0.438	0.301	0.247
UVL	79	1	Neuronitis	12	0.337	0.357	0.377	0.484
UVL	80	0	Neuronitis	12	0.301	0.544	0.159	0.461
UVL	86	0	Neuronitis	1	0.273	0.357	0.377	0.377

					DVA Score, LogMAR			
					Predictable		Unpredictable	
BVL	43	1	Neurosarcoidosis	12	0.143		0.328	
BVL	49	1	Aminoglycoside	2.25	0.296		0.419	
BVL	62	1	Aminoglycoside	1	0.450		0.411	
BVL	70	0	Radiation	108	0.198		0.390	
BVL	72	0	Aminoglycoside	7	0.429		0.678	
BVL	75	1	Unknown	36	0.368		0.451	
BVL	73	0	Unknown	48	0.279		0.500	

*DVA indicates dynamic visual acuity; LogMAR, logarithm of the minimal angle of resolution; UVL, unilateral vestibular loss; BVL, bilateral vestibular loss; 1, incomplete; 0, complete loss; and s/p AN, status post acoustic neuroma. DVA-predictable and -unpredictable are explained in the introductory section of the text.

Table 2. Factors Affecting DVA*

Group	Score, LogMAR		Difference Between DVA-Predictable and -Unpredictable, P Value	Relationship of Age, P Value (%)†	
	DVA-Predictable	DVA-Unpredictable		DVA-Predictable	DVA-Unpredictable
Healthy subjects, all ages	0.030 ± 0.027	0.045 ± 0.044	.02	.07 (13)	<.001 (40)
UVL, toward unaffected side	0.185 ± 0.120	0.232 ± 0.146	.01	.001 (46)	<.001 (54)
UVL, toward affected side	0.248 ± 0.135	0.319 ± 0.157	<.001	.01 (29)	.02 (28)
BVL	0.309 ± 0.115	0.454 ± 0.112	.004	.29	.16

*For DVA-predictable and DVA-unpredictable, data are given as mean ± 2 SD. Definitions and terms are explained in the footnote to Table 1.

†Percentages represent percentage of DVA score attributable to age.

ing low-frequency head movements is the COR. The COR is a compensatory eye movement that parallels the VOR but is generated by inputs from receptors in ligaments and joints in the upper cervical region.²³ In healthy individuals, the COR may not be present. Even when present, COR gain is unremarkable, ranging from 0.07 to 0.20 at 0.1 Hz.⁷⁻⁹ We do not believe that the pursuit/optokinetic system or the COR contributed to gaze stability in our patients during predictable or unpredictable head rotations. In the paradigm we use, the velocity of head movement when the tar-

get optotype is displayed (120°/s-180°/s) exceeds the ability of these mechanisms to contribute to gaze stability.

During active head movements, efference copy, in which the motor commands that produce 1 movement, eg, movement of the head, would also produce compensatory eye movement, can contribute to gaze stability.¹¹ During active or passive head movements, in which the direction and temporal qualities of the head movement are predictable, central programming of appropriate compensatory eye movement can contribute to gaze stabil-

ity.⁶ Mechanisms such as efference copy and the central programming of eye movements may have contributed to gaze stability during active head rotations (which are also predictable) but would not have contributed to gaze stability during the unpredictable passive head rotations. We think these later 2 mechanisms account for the difference between DVA-predictable and DVA-unpredictable scores that we found in all groups.

RELATIVE CONTRIBUTION OF CENTRAL PROGRAMMING TO DVA ACROSS GROUPS

We found that for healthy subjects and patients with UVL, head movements toward the affected and unaffected sides had the same difference between DVA-predictable and DVA-unpredictable scores (Figure 3). For patients with BVL, however, the difference between DVA-predictable and DVA-unpredictable scores was significantly larger compared with all other groups. This may be due to the superior use of central programming of eye movements and efference copy by the patients with BVL compared with the other groups. Another possibility is that patients with BVL differed from the other groups because the deficit in the patients with BVL was so much greater.

DISTINGUISHING AMONG GROUPS

Our results differ from those of a study by Tian et al²⁴ that failed to demonstrate significant differences in DVA between healthy subjects and patients with UVL or a difference between DVA for head movements toward the affected and unaffected sides in UVL. One explanation for the disparity between the studies is the velocity of head rotation used. We required head velocities to range from 120°/s to 180°/s before the target letter would appear. At those velocities, the eye movement generated by inhibition of the intact labyrinth during head movements toward the affected labyrinth would not be sufficient to produce gaze stability. In contrast, Tian et al²⁴ displayed the target at head velocities ranging from 50°/s to 75°/s, which would be within the effective range of the intact labyrinth to produce appropriate compensatory eye movements during head rotations toward the affected side. Using higher velocities of head movement is clearly necessary to reveal the difference in DVA between healthy subjects and patients with UVL as well as the difference between affected and unaffected sides in patients with UVL.

Unlike a previous report from our laboratory,¹⁶ in the present study we found no difference in DVA-predictable scores between healthy subjects and patients with UVL during head movements toward the unaffected side. One possibility is that the UVL group in the present study had compensated more for the effect of the vestibular loss than did the patients with UVL in the previous study. Another possibility is that there is a difference in the degree of vestibular loss between the 2 groups. Finally, patients with UVL in the present study were older (mean age, 66.7 ± 13.1 years) than those in the previous study group (mean age, 51.7 ± 14.6 years), and this difference may have been a factor.

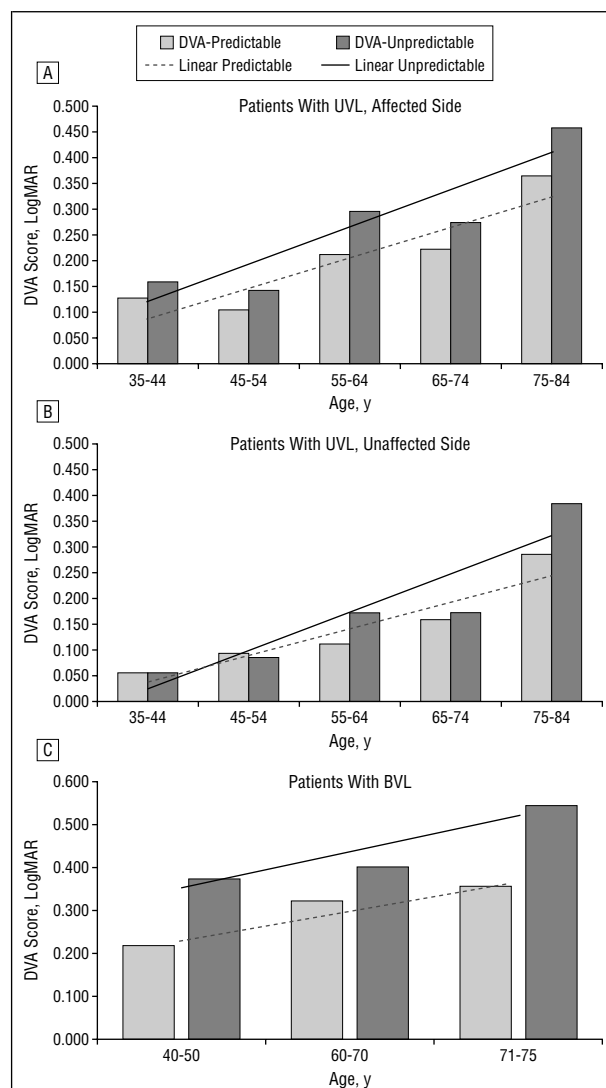


Figure 2. There was a significant relationship between age and scores for dynamic visual acuity (DVA) during predictable (DVA-predictable) and unpredictable (DVA-unpredictable) head movements toward (A) and away from (B) the affected side in patients with unilateral vestibular loss (UVL). No relationship was found between age and DVA in patients with bilateral vestibular loss (BVL) (C). Trend lines are shown. LogMAR indicates logarithm of the minimal angle of resolution.

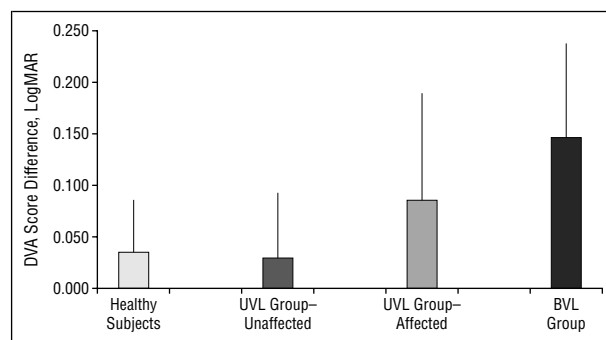


Figure 3. Difference between scores for dynamic visual acuity (DVA) during predictable (DVA-predictable) and unpredictable (DVA-unpredictable) head movements for healthy subjects, for patients with unilateral vestibular loss (UVL) for head movements toward the unaffected and affected sides, and for patients with bilateral vestibular loss (BVL). The BVL group differed significantly from all other groups ($P < .005$). Data are given as mean ± 2 SD. LogMAR indicates logarithm of the minimal angle of resolution.

In general, older subjects had poorer visual acuity during head movement than did younger subjects. This was true for DVA-predictable and DVA-unpredictable scores across all groups. This general trend is similar to earlier results from our laboratory with DVA-predictable scores.¹⁶ In part, this may be related to changes in the vestibular system with increasing age.²⁵ Baloh et al²⁵ found that at high-velocity head movements (in the range used in our study), older subjects (aged >75 years) had lower visual-VOR gain compared with younger subjects (aged 19-39 years). This decrease in visual-VOR gain with age would result in greater retinal slip and therefore in a decrement in dynamic visual acuity.

We found that, for healthy subjects and for the UVL group for head movements toward the unaffected side, the difference between DVA-predictable and DVA-unpredictable scores increased with increasing age (differences in the slopes of the trend lines for DVA-predictable and DVA-unpredictable scores are seen in Figures 1 and 2B). This suggests that age has a greater effect on DVA in subjects who are older compared with younger subjects. This was not true for patients with UVL for head movements toward the affected side, or for patients with BVL, suggesting that the loss of vestibular function, with the resultant increase in retinal slip during head movement, has a far greater impact on DVA than has age.

CONCLUSIONS

The difference between DVA-predictable and DVA-unpredictable scores suggests the degree to which subjects are able to use mechanisms such as the central programming of eye movements and efference copy to enhance gaze stability. Patients with BVL appear to use these compensatory mechanisms to a greater extent than do patients with UVL or healthy subjects. Although visual acuity during unpredictable head movements is poorer than during active (predictable) head movements, both tests distinguish patients with UVL and BVL from healthy subjects. The presence of a vestibular deficit has a greater effect on DVA than has age.

Accepted for publication May 17, 2001.

Supported by grant 03196 from the National Institute on Deafness and Other Communication Disorders, Bethesda, Md (Drs Herdman and Tusa), and by the Foundation for Physical Therapy, Alexandria, Va (Mr Schubert).

Corresponding author and reprints: Susan J. Herdman, PT, PhD, Department of Rehabilitation Medicine, Emory University, 1441 Clifton Rd NE, Atlanta, GA 30322 (e-mail: sherdma@emory.edu).

- Grossman GE, Leigh RJ, Bruce EN, Huebner WP, Lanska DJ. Performance of the human vestibuloocular reflex during locomotion. *J Neurophysiol.* 1989;62:264-272.
- Hess K, Gresty M, Leech J. Clinical and theoretical aspects of head movement dependent oscillopsia (HMDO): a review. *J Neurol.* 1978;219:151-157.
- Demer JL, Oas JG, Baloh RW. Visual-vestibular interaction in humans during active and passive, vertical head movement. *J Vestib Res.* 1993;3:101-114.
- Leigh RJ, Sawyer RN, Grant MP, Seidman SH. High-frequency vestibuloocular reflex as a diagnostic tool. *Ann N Y Acad Sci.* 1992;656:305-314.
- Leigh RJ, Brandt T. A reevaluation of the vestibulo-ocular reflex: new ideas of its purpose, properties, neural substrate, and disorders. *Neurology.* 1993;43:1288-1295.
- Kasai T, Zee DS. Eye-head coordination in labyrinthine-defective human beings. *Brain Res.* 1978;144:123-141.
- Barlow D, Freedman W. Cervico-ocular reflex in the normal adult. *Acta Otolaryngol.* 1980;89:487-496.
- Bronstein AM, Hood JD. The cervico-ocular reflex in normal subjects and patients with absent vestibular function. *Brain Res.* 1986;373:399-408.
- Huygen PLM, Verhagen WIM, Nicoalsen MGM. Cervico-ocular reflex enhancement in labyrinthine-defective and normal subjects. *Exp Brain Res.* 1991;87:457-464.
- Sawyer RN, Thurston SE, Becker KR, Ackley CV, Seidman SH, Leigh RJ. The cervico-ocular reflex of normal human subjects in response to transient and sinusoidal trunk rotations. *J Vestib Res.* 1994;4:245-249.
- Tomlinson RD, Saunders GE, Schwartz DWF. Analysis of human vestibulo-ocular reflex during active head movements. *Acta Otolaryngol.* 1980;90:184-190.
- Barnes GR. Head-eye coordination in normals and patients with vestibular disorders. *Adv Otorhinolaryngol.* 1979;25:197-201.
- Bronstein AM, Hood JD. The cervico-ocular reflex in normal subjects and patients with absent vestibular function. *Brain Res.* 1986;373:399-408.
- Takahashi M, Akiyama I, Tsujita N. Failure of gaze stabilization under high-frequency head oscillation. *Acta Otolaryngol.* 1989;107:166-170.
- Collewijn H, Martins AJ, Steinman RM. Compensatory eye movements during active and passive head movements: fast adaptation to changes in visual magnification. *J Physiol.* 1983;340:259-286.
- Herdman SJ, Tusa RJ, Blatt PJ, Suzuki A, Venuto PJ, Roberts D. Computerized Dynamic Visual Acuity test in the assessment of vestibular deficits. *Am J Otol.* 1998;19:790-796.
- Hess K, Baloh RW, Honrubia V, Yee RD. Rotational testing in patients with bilateral peripheral vestibular disease. *Laryngoscope.* 1985;95:85-88.
- Honrubia V, Marco J, Andrews J, Minser K, Yee RD, Baloh RW. Vestibulo-ocular reflexes in peripheral labyrinthine lesions, III: bilateral dysfunction. *Am J Otolaryngol.* 1985;6:342-352.
- Halmagyi GM, Curthoys IS, Cremer PD, et al. The human horizontal vestibulo-ocular reflex in response to high-acceleration stimulation before and after unilateral vestibular neurectomy. *Exp Brain Res.* 1990;81:479-490.
- Ferris FL, Kassoff A, Bresnick GH, Bailey I. New visual acuity charts for clinical research. *Am J Ophthalmol.* 1982;94:91-96.
- Grossman GE, Leigh RJ, Abel LA, Lanska DJ, Thurston SE. Frequency and velocity of rotational head perturbations during locomotion. *Exp Brain Res.* 1988;70:470-476.
- Demer JL, Viirre ES. Visual-vestibular interaction during standing, walking, and running. *J Vestib Res.* 1996;6:295-313.
- Hikosaka O, Maeda M. Cervical effects on abducens motoneurons and their interaction with vestibulo-ocular reflex. *Exp Brain Res.* 1973;18:512-530.
- Tian J, Shubayev I, Demer JL. Dynamic visual acuity during passive and active head thrusts in normal and unilaterally vestibulopathic humans [abstract]. *Soc Neurosci Abstr.* 2000;26(pt 2):1991.
- Baloh RW, Jacobson KM, Socotch TM. The effect of aging on visual-vestibuloocular responses. *Exp Brain Res.* 1993;95:509-516.