

A New Dynamic Visual Acuity Test to Assess Peripheral Vestibular Function

Domenic Vital, MD; Stefan C. A. Hegemann, MD; Dominik Straumann, MD; Oliver Bergamin, MD; Christopher J. Bockisch, PhD; Dominik Angehrn, Dip Ing; Kai-Uwe Schmitt, PhD; Rudolf Probst, MD

Objective: To evaluate a novel test for dynamic visual acuity (DVA) that uses an adaptive algorithm for changing the size of Landolt rings presented during active or passive head impulses, and to compare the results with search-coil head impulse testing.

Design: Prospective study in healthy individuals and patients with peripheral vestibular deficits.

Setting: Tertiary academic center.

Participants: One hundred neuro-otologically healthy individuals (age range, 19-80 years) and 15 patients with bilateral (n=5) or unilateral (n=10) peripheral vestibular loss (age range, 27-72 years).

Interventions: Testing of static visual acuity (SVA), DVA during active and passive horizontal head rotations (optotype presentation at head velocities >100°/s and >150°/s), and quantitative horizontal head impulse testing with scleral search coils.

Main Outcome Measure: Difference between SVA and DVA, that is, visual acuity loss (VA loss), gain of the high-acceleration vestibulo-ocular reflex.

Results: Passive head impulses and higher velocities were more effective than active impulses and lower velocities. Using passive head impulses and velocities higher than 150°/s, the DVA test discriminated significantly ($P < .001$) among patients with bilateral vestibulopathy, those with unilateral vestibulopathy, and normal individuals. The DVA test sensitivity was 100%, specificity was 94%, and accuracy was 95%, with search-coil head impulse testing used as a reference. In healthy individuals, VA loss increased significantly with age ($P < .001$; $R^2 = 0.04$).

Conclusion: Dynamic visual acuity testing with Landolt rings that are adaptively changed in size enables detection of peripheral vestibular dysfunction in a fast and simple way.

Arch Otolaryngol Head Neck Surg. 2010;136(7):686-691

Author Affiliations: Departments of Otorhinolaryngology–Head and Neck Surgery (Drs Vital, Hegemann, Bockisch, and Probst), Neurology (Drs Straumann and Bockisch), and Ophthalmology (Drs Bergamin and Bockisch), Zurich University Hospital, Zurich, Switzerland; and Institute for Biomedical Engineering, Swiss Federal Institute of Technology and University of Zurich (Mr Angehrn and Dr Schmitt).

GAZE STABILIZATION DURING high-velocity head movements is enabled by the vestibulo-ocular reflex (VOR), which produces compensatory eye movements to stabilize images on the retina with a latency of about 10 milliseconds.^{1,2} The gaze stabilization by the VOR can be evaluated qualitatively by the head impulse test at the bedside.³ The head impulse can also be used to quantitatively measure the gain of the high-acceleration VOR (eye velocity divided by head velocity) when recording eye and head movements with high temporal and spatial resolution. Traditionally, these recordings are performed with the semi-invasive magnetic search-coil technique,^{4,5} which presently serves as the standard,⁶ and recently the recordings have been performed with video-oculography.⁷

The measurement of visual acuity (VA) during head impulses, called dynamic visual acuity (DVA) testing, offers a relatively simple alternative. This technique is based on the fact that peripheral vestibular lesions decrease the gain of the VOR and consequently increase retinal image slip^{2,8-10} during head movements. If retinal image slip velocity exceeds 2°/s to 4°/s, then VA is reduced.^{11,12} Therefore, measurement of DVA can give indirect information about the VOR performance and semicircular canal function, provided that nonvestibular ocular motor disorders have been excluded.

Several DVA testing methods have been described.^{2,8-10,12-15} What most of these methods have in common is that VA was tested with a transiently appearing Snellen optotype E with 4 possible orientations during head movements of different velocities. In general, the tests required a

rather high number of head movements because the algorithms of VA testing were stepwise. To shorten the duration of DVA testing, Schubert et al² introduced an adaptive algorithm that starts near the middle acuity level and continues in accordance to the patient's performance. The proposed algorithm still needed about 100 head impulses before arriving at the final DVA value. Peters and Bloomberg¹⁶ made use of Landolt rings as visual targets assessing DVA while patients walked on a treadmill. They also used only 4 of the 8 possible orientations.

Our aim was to design an improved DVA test, which can be used efficiently in a clinical routine and is applicable as an office procedure. With a low number of head impulses and within a short time period, this DVA test should be able to screen VOR function with a high sensitivity and specificity.

We describe the newly developed DVA testing algorithm and report test results in a large number of otologically healthy individuals with a wide age range and in patients with unilateral and bilateral vestibular loss. All results were compared with those of search-coil head impulse testing, which was used as a reference.

METHODS

PARTICIPANTS

One hundred individuals (mean [SD] age, 45 [16] years; range, 19-80 years) without otological and neurological disorders were included in the study. They were recruited from among hospital personnel, students, people who attended public lectures at the university, and family members of these persons. Normal peripheral vestibular function was verified by a normal VOR gain in quantitative head impulse testing. Fifteen patients (age, 54 [13] years; range, 27-72 years) with unilateral or bilateral peripheral vestibular loss were recruited by otologists and neuro-otologists of the ear, nose, and throat and neurology departments. Criteria for a complete unilateral vestibular loss were a history of labyrinthectomy or vestibular neurectomy and/or a VOR gain of less than 0.30 on the affected side as determined by search-coil head impulse testing; criteria for a complete bilateral vestibular loss were VOR gains of less than 0.20 on both sides. All individuals were investigated on a volunteer basis and gave written informed consent to participate in a protocol approved by the local ethics committee.

DVA INSTRUMENTATION

The equipment of our DVA testing system consisted of a personal computer with an external keyboard, a 19-inch liquid crystal display monitor (1280 × 1024 pixels, 75 Hz) and a Sparkfun velocity sensor (Sparkfun Electronics, Boulder, Colorado), which was fixed on a headset to the individual's head. The monitor was placed at a distance of 5 m in front of the patient, who was sitting on a chair.

Both static visual acuity (SVA) and DVA were measured. Visual acuity assessment was performed using the standard optotype "Landolt ring" as a visual target with 8 different orientations instead of the Snellen optotype E with only 4 possible orientations,¹⁷ reducing the chance performance level by a factor of 2. Visual acuity was expressed as the decadic logarithm of the minimum angle of resolution (logMAR), which represents the scientific term of the VA.¹⁷ The individuals were asked to recognize the orientation of a Landolt ring, which is displayed randomly on the monitor, and to type in the correct an-

swer on an external keyboard representing the 8 possible orientations. If the individual did not recognize the orientation, then a forced choice paradigm was required: individuals were told to always give their best answer, even when they had very low confidence in their answer. A series of 5 Landolt rings was presented at a given acuity level. The acuity level was passed if the orientation of at least 3 of 5 Landolt rings was recognized correctly. Only 3 optotypes were displayed if the first 3 were recognized correctly.

During DVA testing, Landolt rings were displayed for a time period of 100 milliseconds if head velocity exceeded a preset limit. To improve fixation during the head impulse, a small dot was placed in the center of the monitor. This dot was extinguished immediately before the Landolt ring occurred. In the SVA test, the next optotype was displayed automatically after the patient made his or her choice. Static visual acuity testing started at a level of 0.4 logMAR, DVA testing at a level of 0.4 logMAR above SVA. With each incorrect series of Landolt rings, acuity level increased by 0.4 logMAR. If a series was correct, then the size of the optotypes decreased by 0.1 logMAR until the series was no longer recognized correctly (incorrect detection of 3 or more Landolt rings), and the test was stopped. Visual acuity was determined by the value of the next to be last (correctly identified) series of Landolt rings minus 0.02 or 0.04 logMAR, respectively, if 1 or 2 answers on the last (incorrect) series were correct. By subtracting SVA from DVA, the term "VA loss" was calculated, which is a measure of the decrement of VA during motion.

DVA TEST PROTOCOL

The SVA was determined first using our system. Because VA loss was calculated as the difference of DVA and SVA, DVA testing was independent of the patient's vision. Individuals were allowed to wear their own eyeglasses or contact lenses during both SVA and DVA testing. Visual acuity was measured binocularly.

Dynamic visual acuity testing consisted of an active part and a passive part. In the active part, the individual generated horizontal head rotations by active movements. In the passive part, head impulses with random timing were delivered manually by the examiner standing behind the individual and holding the head laterally on both sides without interfering with the visual field. The head impulses consisted of brisk rotations toward the tested labyrinth and the center head position with a starting position of a 20° to 30° turn to the contralateral side. This kind of impulse has the advantage of being easy to perform for both the patient and the examiner, and patients are able to view the monitor continuously through the lenses of their eyeglasses. The important aspects of this procedure include achieving a correct starting position and having an experienced examiner to deliver impulses with the correct magnitude. Two different blocks of preset velocity limits were tested, 1 with 150°/s and 1 with 100°/s.

Before starting the actual DVA test, individuals were familiarized with it, and they were provided with ample opportunity to practice active and passive DVA testing to both sides.

QUANTITATIVE HEAD IMPULSE TESTING

Quantitative head impulse testing (qHIT) with search coils was performed as previously described by members of the vestibulo-oculomotor laboratory of our institution.^{6,18} Briefly, eye and head movements were analyzed during head impulse testing in a magnetic coil frame using a search coil around the cornea of the right eye applied following anesthesia with oxybuprocaine, 0.4%, and a second coil fixed to the forehead with adhesive tape. Digi-

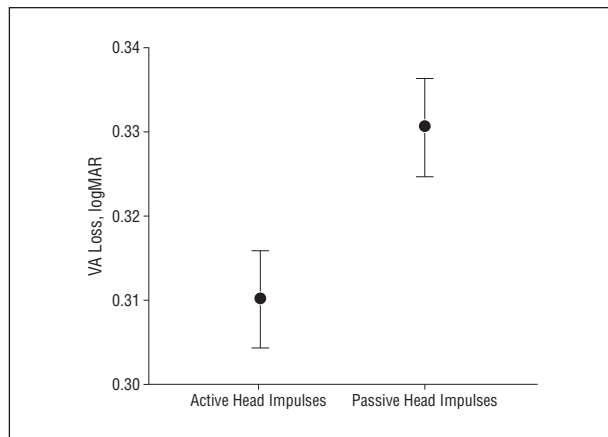


Figure 1. Comparison between active and passive head impulses. The difference of visual acuity (VA) loss is significant ($P=.006$). MAR indicates minimum angle of resolution. T-bars indicate standard errors; dots, mean values.

tized signals were computed and the VOR gain defined by $VOR = 1 - (\Delta \text{gaze} / \Delta \text{head})$, where gaze (eye-in-space) and head were evaluated when the head had turned from 3° to 7° . If the median gain was less than the mean minus 2 standard deviations of results from a reference population ($n=37$; mean [SD] age, 47 [16] years) of the vestibulo-oculomotor laboratory of our hospital, then head impulses were graded as pathologic.^{6,18}

DATA ANALYSIS

Data were analyzed by analysis of variance. Post hoc statistics were performed using Tukey test if a statistically significant main effect or interaction was found ($P<.05$). In addition, age effects on the VA loss and the correlation of VA loss and VOR gain were studied by regression analysis. Discrimination of patients from healthy individuals was examined by analysis of the z scores for the different test parameters. The z scores indicate how many standard deviations the VA loss of the patients differs from the overall mean. Sensitivity was calculated as the ratio of true-positive and the sum of true-positive and false-negative test results; specificity was calculated as the ratio of true-negative and the sum of true-negative and false-positive test results. Accuracy was calculated by the sum of true-positive and true-negative results, divided by the sum of true-positive, true-negative, false-positive, and false-negative results. Results were divided into the groups of true-positive, true-negative, false-positive, and false-negative on the basis of search-coil head impulse testing, which was used as a reference.

RESULTS

DVA OF NORMAL INDIVIDUALS

Significant effects on the VA loss were attributed to the type and the velocity of the head rotations: Active head impulses led to a lower VA loss than passive impulses ($F=7.48$; $P=.006$, **Figure 1**), just as the VA loss was lower using a velocity limit of $100^\circ/\text{s}$ than one of $150^\circ/\text{s}$ ($F=126.46$; $P<.001$). The VA loss was significantly higher with increasing age ($F=15.37$; $P<.001$). However, the linear correlation in the regression analysis was low, with only 4% of the variance of VA loss accounted by age (**Figure 2**). Even though the VA loss in normal individuals during rightward head rotations showed a sig-

nificantly poorer value for the overall effect than during leftward rotations ($F=7.02$; $P=.008$), the difference corresponded to a single optotype missed for head rotations to the right compared with the rotations to the left.

COMPARISON WITH DVA OF PATIENTS WITH PERIPHERAL VESTIBULAR LOSS

Passive head rotations ($z=2.27$) showed clearer discrimination of patients from normal individuals than active movements ($z=1.24$). Furthermore, discrimination was better during head impulses higher than $150^\circ/\text{s}$ ($z=2.08$) than during those higher than $100^\circ/\text{s}$ ($z=1.43$). Thus, the highest z score ($z=2.72$) was yielded with passive head rotations of a velocity higher than $150^\circ/\text{s}$. Using these parameters, the patient groups of bilateral vestibulopathy and of unilateral vestibulopathy on both ipsilateral and contralateral side differed significantly from healthy individuals and from each other ($P<.001$) (**Figure 3**).

Healthy individuals had a mean (SD) decrement of VA of 0.38 (0.10) logMAR under dynamic conditions. This VA loss was 1.40 (0.29) logMAR in individuals with bilateral vestibulopathy. Data of rightward and leftward head rotation were pooled in normal individuals and in patients with bilateral vestibulopathy because there was no significant difference for the test parameters of passive rotation and velocity limits of $150^\circ/\text{s}$ ($P=.11$ and $P=.98$, respectively). Persons with unilateral vestibulopathy had a mean (SD) VA loss of 1.07 (0.19) logMAR and 0.59 (0.15) logMAR during ipsilesional and contralesional head rotation, respectively. No significant difference was present between the numbers of correct answers for any of the 8 possible directions of the Landolt rings, neither for normal nor for patients with vestibulopathy ($F=2.03$, $P=.12$, and $F=0.98$, $P=.47$, respectively).

The test performance of the patients was analyzed using VOR gain as measured by quantitative head impulse testing as a reference, age-matched normal values (mean + 2 SDs; 20-40 years: ≤ 0.56 logMAR; 41-60 years: ≤ 0.58 logMAR; 61-80 years: ≤ 0.60 logMAR), and test parameters of passive rotation and velocity limits of $150^\circ/\text{s}$. Using search-coil head impulse testing as a reference, sensitivity of the DVA test was 100% for both unilateral and bilateral vestibular loss. Specificity was calculated to be 94%. The accuracy of the DVA test was 95%.

NUMBER OF HEAD ROTATIONS

The mean (SD) number of head rotations needed to test both horizontal semicircular canals was 39 (15) for the parameters of passive rotation and velocity limits of $150^\circ/\text{s}$. Individuals without peripheral vestibulopathy needed fewer head impulses (34 [13]) than patients with peripheral vestibular loss (50 [14]).

CORRELATION OF VA LOSS AND VOR GAIN

The correlation of the VA loss and the VOR gain, as measured by qHIT, was significant ($P<.001$). Regression analysis showed a linear correlation with an R^2 of 0.72 (**Figure 4**).

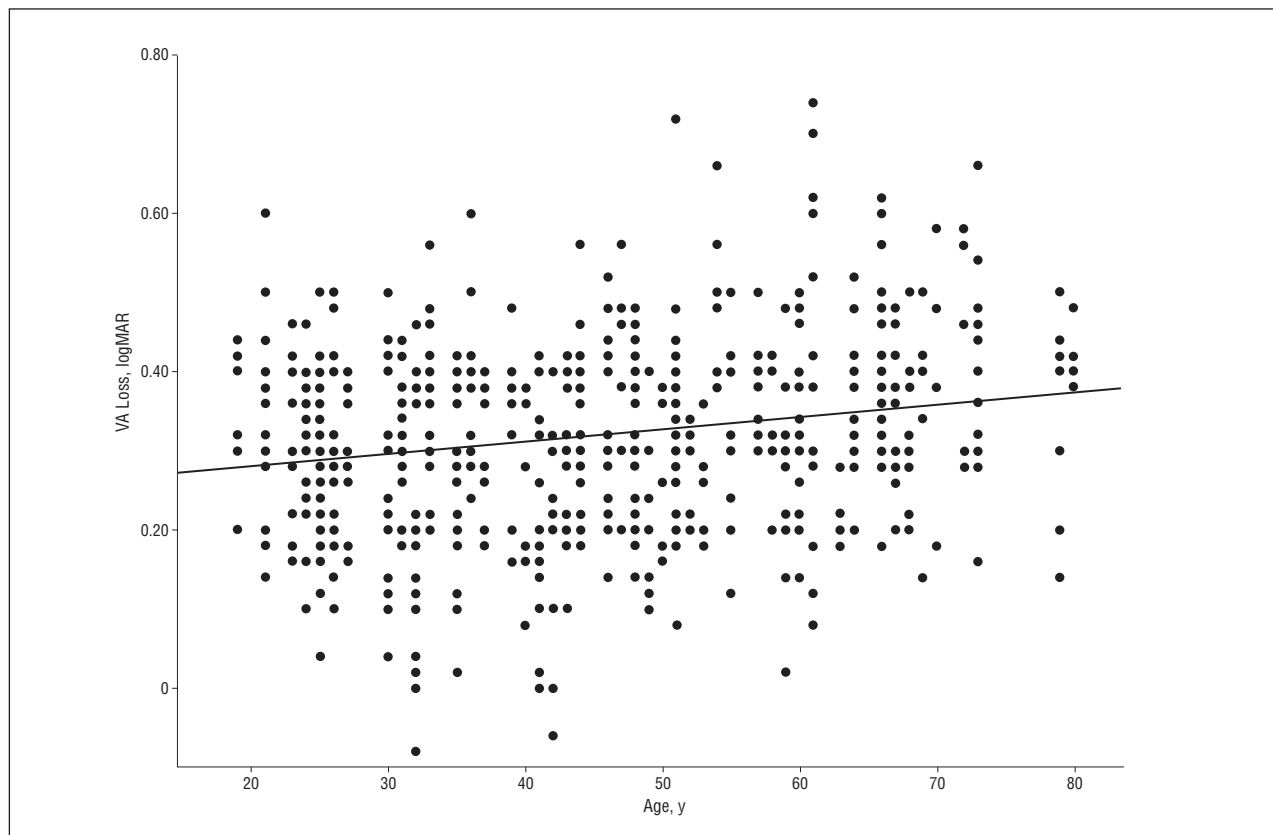


Figure 2. Dependency of the visual acuity (VA) loss on age. The dependency was significant ($P < .005$). The overall variance of VA loss was high and linear regression analysis determined that age accounted for only 4%. MAR indicates minimum angle of resolution.

COMMENT

We developed a new test procedure to measure DVA by optimizing several test parameters, in particular, the use of the optotype Landolt ring with 8 orientations instead of the optotype E with only 4, passive instead of active head rotation, and velocity limits of 150°/s leads to a DVA test with high efficiency compared with other DVA tests described in the literature. A test algorithm was designed with the aim of reducing the number of head impulses compared with previous studies.^{2,10,14} A peripheral vestibular loss was detected in a fast and simple way with high sensitivity, specificity, and accuracy. Based on the findings of this study, DVA testing may represent an easily applied office procedure in the assessment of semicircular canal function in the high-frequency range. This seems to be more important for gaze stabilization by the VOR than the low-frequency range measured with calorics.

The reduction of VA under dynamic conditions was age dependent. An age-related decrement of the VOR gain is well known, having been observed in sinusoidal rotation¹⁹ and head impulse testing.²⁰ Because participants were allowed to wear their habitual glasses or contact lenses, and the VA loss was calculated as the difference of DVA and SVA, the age-related changes in VA loss are unlikely to be related to a reduction of the SVA with increasing age. Moreover, Herdman et al,^{10,14} who tested fewer individuals, also observed an even larger age dependency in the results of their DVA tests.

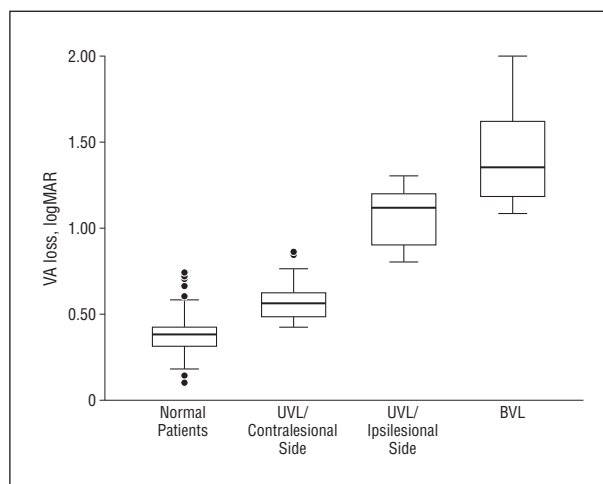


Figure 3. Boxplots of visual acuity (VA) loss for the normal individuals, of the contralateral and ipsilesional side of patients with unilateral vestibular loss (UVL), and of patients with bilateral vestibular loss (BVL). MAR indicates minimum angle of resolution. T-bars indicate 1.5 interquartile distances; dots, outliers.

Besides reducing test time, DVA testing during passive (unpredictable) head impulses with a velocity higher than 150°/s enabled the best discrimination of healthy and vestibulopathic individuals compared with our other parameters. Sensitivity and specificity of DVA were high with search-coil head impulse testing used as a reference.

During predictable active head rotations, nonvestibular oculomotor mechanisms may augment the VOR gain.

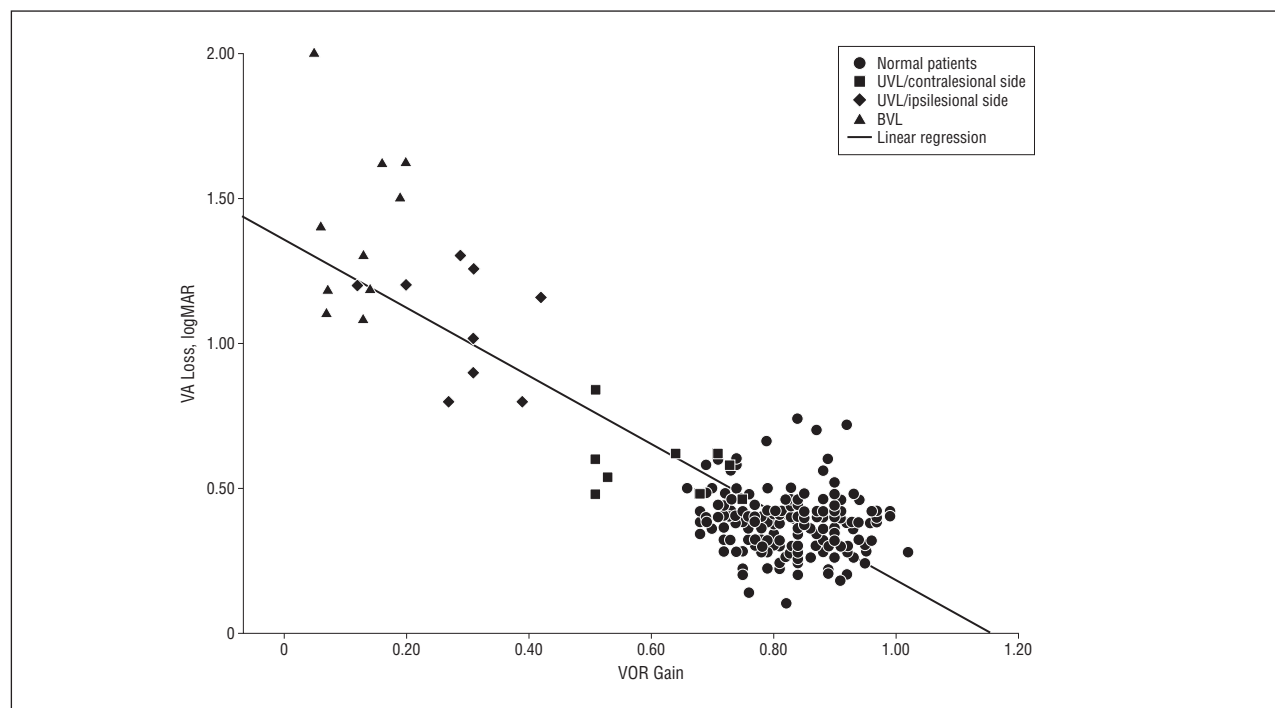


Figure 4. Correlation of visual acuity (VA) loss as measured with passive head impulses $>150^\circ/\text{s}$ and vestibulo-ocular reflex gain as measured by quantitative head impulse testing (qHIT) for normal individuals and patients with unilateral (UVL) or bilateral vestibular loss (BVL). MAR indicates minimum angle of resolution. The linear regression was significant ($P < .001$; $R^2 = 0.72$).

Such nonvestibular contributions to gaze stabilization mainly consist of anticipatory slow eye movements and preprogrammed catch-up saccades with short latencies.^{8,9,14} Catch-up saccades during nonanticipated head impulses have latencies of 100 to 180 milliseconds^{9,21} and prevent the recognition of Landolt rings within the display period of 100 milliseconds in case of a deficient VOR gain. Besides higher sensitivity for DVA testing, passive head movements may also be related to the clinical deficit experienced by the patient. Such movements are ubiquitous in daily activities such as driving a car, riding a bicycle, or skiing. A total compensation of reflexive eye movements elicited by rapid passive head movements in the presence of a peripheral vestibular deficit cannot be expected, even if catch-up saccades of short latency already occur during the head movement (so-called covert saccades).²¹ These covert saccades, however, might be responsible for good adaptation. To measure whether the adaptation is then due to recovery or to faster saccades is technically not possible by DVA because eye movements are not measured.

In contrast, latencies during predictable head movements are shorter (30-100 milliseconds)⁹ and might contribute to gaze stabilization within the stimulus presentation period. Accordingly, DVA testing during active head rotations was less sensitive for screening VOR function than passive head rotations. However, DVA testing during active movements might have potential for measuring central adaptation following peripheral vestibulopathy even though central adaptation on active head movements is only a part in vestibulopathic recovery. However, if active VA loss is significantly better than passive VA loss, such adaptation might be more accessible

to training and might imply that there is a possibility to increase adaptation. This question is addressed in our ongoing study.

One reason for the lower VA loss in healthy individuals and the poorer discrimination of normal individuals and those with vestibulopathy using the velocity limit of $100^\circ/\text{s}$ may be the push-pull mechanism of the semicircular canal function. Both horizontal semicircular canals contribute to the VOR function during head rotations of lower velocities. The contralateral afferents are inhibited with increasing velocity and are gradually driven into inhibitory cutoff.²¹ Consistent with Ewald's second law, the impact of contralateral semicircular canal signals on the VOR decreases with higher accelerations.²²

Neither the detection of a peripheral vestibular hypofunction nor the possibility of measuring the adaptation on a peripheral vestibulopathy by active DVA testing has yet been studied adequately. This will be the aim of further work, together with improvements of our test algorithm and its adaptation for testing anterior and posterior semicircular canal function.

In conclusion, our new DVA test procedure that includes an adaptive algorithm for changing the size of Landolt rings enables detection of a peripheral vestibular loss with high accuracy in a fast and simple way. The sensitivity and specificity are comparable to quantitative VOR measurements with search-coil head impulse testing. The accuracy of the test critically depends on the use of dynamic test parameters. Passive head impulses with a velocity higher than $150^\circ/\text{s}$ were found to provide very high accuracy. Our new test algorithm reduced the number of head impulses and made DVA testing fast and simple for both the patient and the examiner.

Submitted for Publication: September 30, 2009; final revision received January 15, 2010; accepted January 25, 2010.

Correspondence: Stefan C. A. Hegemann, MD, Department of Otorhinolaryngology–Head and Neck Surgery, Zurich University Hospital, Frauenklinikstrasse 24, CH-8091 Zurich, Switzerland (stefan.hegemann@usz.ch).

Author Contributions: Drs Vital, Hegemann, and Probst contributed equally to this article. Drs Vital, Hegemann, Straumann, Bergamin, Bockisch, and Schmitt and Mr Angehrn had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Vital, Hegemann, Straumann, Bergamin, Schmitt, and Probst. *Acquisition of data:* Vital and Angehrn. *Analysis and interpretation of data:* Vital, Hegemann, Angehrn, and Probst. *Drafting of the manuscript:* Vital, Bockisch, Angehrn, and Probst. *Critical revision of the manuscript for important intellectual content:* Hegemann, Straumann, Bergamin, Bockisch, Schmitt, and Probst. *Statistical analysis:* Vital, Bockisch, and Angehrn. *Administrative, technical, and material support:* Vital, Hegemann, Straumann, Bockisch, Angehrn, and Schmitt. *Study supervision:* Hegemann, Schmitt, and Probst.

Financial Disclosure: None reported.

REFERENCES

1. Collewyn H, Smeets J. Early components of the human vestibulo-ocular response to head rotation: latency and gain. *J Neurophysiol*. 2000;84(1):376-389.
2. Schubert MC, Migliaccio AA, Della Santina CC. Dynamic visual acuity during passive head thrusts in canal planes. *J Assoc Res Otolaryngol*. 2006;7(4):329-338.
3. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol*. 1988;45(7):737-739.
4. Robinson DA. A method of measuring eye movement using a scleral search coil in a magnetic field. *IEEE Trans Biomed Eng*. 1963;10:137-145.
5. Schmid-Priscoveanu A, Böhmer A, Obzina H, Straumann D. Caloric and search-coil head-impulse testing in patients after vestibular neuritis. *J Assoc Res Otolaryngol*. 2001;2(1):72-78.
6. Jorns-Häderli M, Straumann D, Palla A. Accuracy of the bedside head impulse test in detecting vestibular hypofunction. *J Neurol Neurosurg Psychiatry*. 2007;78(10):1113-1118.
7. Weber KP, MacDougall H, Halmagyi G, Curthoys I. Impulsive testing of semicircular-canal function using video-oculography. *Ann N Y Acad Sci*. 2009;1164:486-491.
8. Tian JR, Shubayev I, Demer J. Dynamic visual acuity during transient and sinusoidal yaw rotation in normal and unilaterally vestibulopathic humans. *Exp Brain Res*. 2001;137(1):12-25.
9. Tian JR, Shubayev I, Demer JL. Dynamic visual acuity during passive and self-generated transient head rotation in normal and unilaterally vestibulopathic humans. *Exp Brain Res*. 2002;142(4):486-495.
10. Herdman SJ, Tusa RJ, Blatt P, Suzuki A, Venuto PJ, Roberts D. Computerized dynamic visual acuity test in the assessment of vestibular deficits. *Am J Otol*. 1998;19(6):790-796.
11. Barnes GR, Smith R. The effects of visual discrimination of image movement across the stationary retina. *Aviat Space Environ Med*. 1981;52(8):466-472.
12. Demer JL, Honrubia V, Baloh R. Dynamic visual acuity: a test for oscillopsia and vestibulo-ocular reflex function. *Am J Otol*. 1994;15(3):340-347.
13. Schubert MC, Herdman SJ, Tusa RJ. Vertical dynamic visual acuity in normal subjects and patients with vestibular hypofunction. *Otol Neurotol*. 2002;23(3):372-377.
14. Herdman SJ, Schubert MC, Tusa RJ. Role of central preprogramming in dynamic visual acuity with vestibular loss. *Arch Otolaryngol Head Neck Surg*. 2001;127(10):1205-1210.
15. Herdman SJ, Schubert MC, Das VE, Tusa RJ. Recovery of dynamic visual acuity in unilateral vestibular hypofunction. *Arch Otolaryngol Head Neck Surg*. 2003;129(8):819-824.
16. Peters B, Bloomberg J. Dynamic visual acuity using "far" and "near" targets. *Acta Otolaryngol*. 2005;125(4):353-357.
17. Gräf M. Strategies of visual acuity assessment [in German]. *Klin Monbl Augenheilkd*. 2004;221(7):557-565.
18. Palla A, Straumann D. Recovery of the high-acceleration vestibulo-ocular reflex after vestibular neuritis. *J Assoc Res Otolaryngol*. 2004;5(4):427-435.
19. Baloh RW, Jacobson K, Socotch T. The effect of aging on visual-vestibuloocular responses. *Exp Brain Res*. 1993;95(3):509-516.
20. Brzezny R, Glasauer S, Bayer O, Siebold C, Büttner U. Head impulses in three orthogonal planes of space: influence of age. In: Brandt T, Cohen B, Siebold C. *The Oculomotor and Vestibular Systems: Their Function and Disorders*. New York: Annals of New York Academy of Science; 2003:473-477.
21. Weber KP, Aw S, Todd M, McGarvie L, Curthoys I, Halmagyi G. Head impulse test in unilateral vestibular loss: vestibulo-ocular reflex and catch-up saccades. *Neurology*. 2008;70(6):454-463.
22. Minor LB, Lasker D, Backous D, Hullar T. Horizontal vestibuloocular reflex evoked by high-acceleration rotations in the squirrel monkey, I: normal responses. *J Neurophysiol*. 1999;82(3):1254-1270.