Otology & Neurotology
28:896–904 © 2007, Otology & Neurotology, Inc.

The High-Frequency/Acceleration Head Heave Test in Detecting Otolith Diseases

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Objective: To investigate whether transient, high-acceleration interaural head heaves (translational vestibulo-ocular reflex [tVOR]) could aid in the diagnosis of otolith diseases.
Study Design: Prospective cohort study.
Setting: Tertiary referral center.
Patients: Thirteen patients with symptoms suggestive of otolith diseases and 10 age-matched controls.
Interventions: Patients underwent a clinical otoneurologic examination and standard laboratory audiovestibular evaluation, including audiometry, electronystagmography with bithermal caloric, Halmagyi-Curthoys head thrust test with search coils, and vestibular-evoked myogenic potential. All subjects underwent subjective visual vertical (SVV) and tVOR testings.
Main Outcome Measures: Sensitivity (ratio of peak eye to peak head velocities) and velocity gain (ratio of actual to ideal peak eye velocities).
Results: Five of 13 patients showed no abnormality in any tests. Of the remaining 8, 3 (38%) had reduced tVOR responses, whereas 1 (13%) had abnormal SVV. Sensitivity and velocity gains were symmetrically reduced in 2 patients, who had symptoms for 8 and 24 months. A third patient, symptomatic for 7 weeks, had asymmetric reduction of tVOR responses and a deviated SVV.
Conclusion: Both head heave and SVV tests detect acute, asymmetric otolith diseases. Subjective visual vertical test relies on imbalance of utricular tone and may not detect bilateral symmetric diseases or partial diseases with central compensation. Our preliminary data in a small group of patients show that measuring the tVOR in a higher and more physiologic range of frequencies may serve as useful adjunct to detect acute and chronic otolith dysfunction and seems to be superior to the SVV in detecting bilateral symmetric or asymmetric otolith diseases. **Key Words:** Head heaves—Otolith—Utricle—Vertigo—Vestibulo-ocular reflex.
Otol Neurotol **28:**896–904, 2007.

The otolith-ocular reflex stabilizes retinal image by generating compensatory eye movements in response to linear head acceleration and head orientation with respect to gravity. Patients with otolith dysfunction often present with a variety of symptoms, including false sensations of linear or tilting motion (of the environment or of self), lateropulsion, falling sensations or drop attacks, diplopia, oscillopsia, and visual tilt perceptions (1). Despite increased understanding of otolith physiology in the past decades, detection of otolith lesions remains challenging—clinicians often make the diagnosis on the basis of clinical history and exclusion of other possible causes.

Patients with typical and recurring otolith symptoms but normal standard laboratory vestibular test results (i.e., electronystagmography [ENG] with bithermal caloric testing, Halmagyi-Curthoys test, and vestibular-evoked myogenic potentials [VEMPs]) and subjective visual vertical (SVV) might have partial lesions of the otolith system that escape detection by these conventional tests. Recently, the head heave test has been used to assess utricular dysfunction. Administered either manually or using a head sled, the head heave test delivers transient, low-amplitude, high-acceleration translational stimuli along the interaural axis to stimulate the otolith maculae (2). Using this test, abnormal translational VOR (tVOR) responses were observed in patients with gentamicin ototoxicity (2) and acute vestibular neuritis (3).

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Supported by the Freiwillige Akademische Gesellschaft and the Maragarete und Walter Lichtenstein-Stiftung, Basel (P.K.), the Canadian Institutes of Health Research (the New Investigator Award [MSH 55058] [A.W.] and grants MOP 57853 [A.W.] and MOP 152588 [D.T.]), the Hospital for Sick Children (A.W.), and the University Health Network Ophthalmology Practice Plan (A.W.).

Presented in part at the 24th Annual Meeting of the Bárány Society, Uppsala, Sweden, June 11–14, 2006.

Dr. Tomlinson died on February 12, 2005.

We (4,5) and others (6) have previously demonstrated that the sensitivity of detecting horizontal canal dysfunction can be increased by testing the rotational VOR in a higher and more physiologic range of frequencies. The aim of the present study was to assess whether testing of the tVOR during transient, high-acceleration interaural head translation using the head heave test might reveal complete or partial otolith dysfunction in patients with typical otolith symptoms.

MATERIALS AND METHODS

Study Population

Thirteen consecutive patients with symptoms suggestive of otolith diseases (women, 6; median age, 40 yr [range, 32–65 yr]) were recruited prospectively from the Multidisciplinary Neurotology Clinic of the Department of Otolaryngology, University Health Network, University of Toronto, between September 2005 and March 2006. Symptoms suggestive of otolith diseases included lateropulsion, falling or rising sensations, drop attacks, or prolonged sensations of linear body movement after cessation of actual body movement. A thorough history was obtained, and an otoneurologic examination was conducted. Bedside clinical tests of vestibular function were performed, including the high-frequency/acceleration horizontal head thrust test (6), dynamic visual acuity, assessment for post–head shake nystagmus (7), the oscillopsia test (8), Dix-Hallpike's positioning test using Frenzel's glasses, and hyperventilation test for 60 seconds. In addition to audiometry, patients also underwent standard laboratory vestibular investigations, including ENG with bithermal caloric test, high-frequency/acceleration horizontal head thrust (Halmagyi-Curthoys test) with search coils, and testing for VEMPs (9). Patients with chronic cervical pain after whiplash-type injury, a history or clinical signs of abnormality in the cervical spine or soft tissues of the neck, or a history of eye or neurologic diseases were excluded. Ten age-matched subjects without any vestibular, neurologic, or eye diseases served as controls (women, 3; median age, 38.5 yr [range, 29–59 yr]; $p = 0.41$). Subjects were instructed to abstain from alcohol, sedatives, relaxants, or medications for dizziness for 48 hours before testings. Corrective spectacles were not worn during the tests. The study was approved by the Research Ethics Board of the University Health Network, University of Toronto.

Testing of the Subjective Visual Vertical

Subjects sat in a natural upright position in the dark during binocular viewing of a dimly illuminated straight line. The line was mounted on a linear rotating potentiometer and was located 1 m away from the subject's nasion. Starting from a random nonvertical position, the examiner slowly rotated the line toward the earth vertical and stopped when the subject perceived the line as vertical, as indicated verbally to the examiner. The signal from the potentiometer was amplified, digitized at 1 kHz, and stored for later analysis. The results of 6 trials were averaged.

The Head Heave Test

Viewing Condition

Each subject fixated a target located at a distance of 15 cm while translating along the interaural axis. The viewing distance was measured from the lateral canthi of the eyes, a landmark that approximates the location of the center of rotation of the globe. The target consisted of a single letter "e" of optotype size 3/32″ (12-point font size), which remained visible throughout the trial.

The Stimuli

To administer quasi-reproducible head movement stimuli along the interaural axis, we constructed a head sled similar to the device described by Ramat et al. (2) (Fig. 1). It consists of 2 rigidly connected, padded polycarbonate plates that can be fastened to either side of the head of the subject. Each plate is connected to a linear bearing mounted on a rigid frame and is aligned with the interaural axis. It is equipped with handles and padded stopping bars to either side to restrict the maximum lateral head translation to ±3 cm and to prevent an abrupt stop at the end of the movement. Insulation is inserted between parts of the aluminum frame to prevent distortion of the magnetic field.

The *head heave* stimuli consisted of manually delivered head impulses along the interaural axis to produce a steplike change of head position. Peak accelerations ranged from 0.1*g* to 1.6*g*. The stimuli were unpredictable in direction and timing to prevent the subjects from making anticipatory eye movements. Before each trial, the head was recentered within a window of less than 5 mm. On average, each subject underwent a total of 30 trials (i.e., 15 heaves in each direction). The mean duration of head movement was 145.3 milliseconds (range, 106–191 ms), and the median amplitude within 100 milliseconds after head movement onset was 17.2 mm (range, 11.5–26.3 mm).

Recording of Eye and Head Movements

Angular eye and head positions were measured with the magnetic scleral search coil technique. A cubic phase angle rotating electromagnetic field with a side length of 1.8 m (CNC Engineering, Seattle, WA, USA) was used. The subject wore a single-lead scleral coil annulus in each eye (Skalar, Delft, The Netherlands) after instillation of anesthetic eye drops. Head position in the horizontal plane (yaw) was measured using a search coil embedded in the bite bar. The subjects sat in a chair, with the center of their interpupillary line located at 25 cm in front of the center of the magnetic field, while biting on a bite bar. This anterior displacement of the head position was required so that the head sled device could be mounted on a

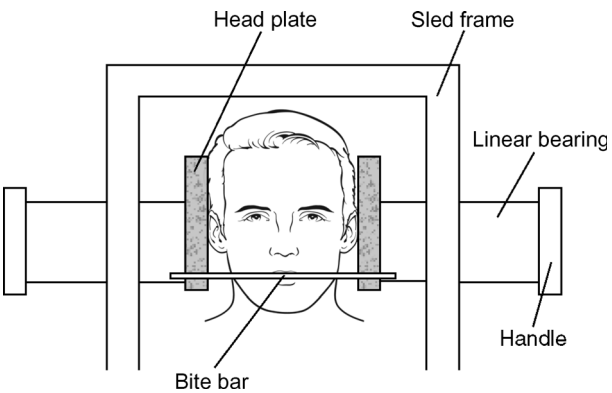

FIG. 1. Diagram showing the head sled apparatus used in the head heave test (see text for details).

permanently installed rotating chair socket. The magnetic field at this head position remained linear, as measured by a search coil mounted on a gimbal. The error in angular position measurements introduced by translational coil displacement of the eye and head coils in the magnetic field was, at most, 0.2%, as established by *in vitro* experiments. The eye and head rotation signals were amplified, digitized at 1 kHz with 12-bit resolution, and stored for later analysis. Data were analyzed using specialized software written in Matlab (MathWorks, Natick, MA, USA). Eye position signals were differentiated and filtered with a fourth-degree Savitzky-Golay filter (frequency, 0–50 Hz) to obtain velocity traces.

Translational head position along the interaural axis was measured using a linear potentiometer with an incremental sensitivity of 13×10^{-4} mm (Omega, Laval, Quebec, Canada). The potentiometer was attached to the sled frame and to 1 of the head plates. Head movement was coupled to chair movement. Yaw head rotation was prevented by fastening the head plates tightly and by filling the area between the postauricular occiput and the head plates with 2 grain-filled bags. The bags were molded to the subject's individual head anatomy and deflated to restrict any head movement relative to the side plates to which they were firmly attached. Head acceleration was recorded using a capacitive beam accelerometer (Crossbow Technology, San Jose, CA, USA) attached to the bite bar.

Data Analysis

Subjective Visual Vertical

The SVV was considered to be abnormal if the deviation from vertical was more than 2 degrees (10).

Head Heaves

Heaves that contained artifacts such as saccades or blinks before the occurrence of peak head and eye velocity were identified and excluded from further analysis. Saccades were identified by their main sequence, whereas blinks were identified by their characteristic trajectory and time course. Only heave trials with a yaw head rotation less than 0.64 degrees and peak head accelerations of 0.5*g* or greater were analyzed. These cutoff values were chosen to prevent the yaw rotational VOR from being a potential confounder and to optimize the diagnostic sensitivity by maximizing the signal-to-noise ratio. Overall, 35% of heaves were included in the final analysis.

To characterize the performance of the interaural tVOR, 2 parameters were computed: sensitivity and velocity gain. The *sensitivity*, expressed in degrees per centimeter, was calculated by dividing the peak eye velocity (in degrees per second) of the cyclopean eye by the peak head velocity (in centimeters per second) within the first 100 milliseconds after the onset of head movement (i.e., before the occurrence of visual feedback). Peak head velocity was computed by integrating head acceleration, which was measured by the accelerometer. The *velocity gain* was calculated by dividing the actual peak cyclopean eye velocity (in degrees per second) by the peak velocity of the ideal cyclopean eye (in degrees per second) within the first 100 milliseconds after onset of head movement. The ideal eye position was computed on the basis of the trigonometric requirements given by target distance, interpupillary distance, and the head position signal of the linear potentiometer. Head movement onset was determined from the accelerometer traces and was defined as the first instance when acceleration consistently exceeded baseline by 3 standard deviations (SDs).

Transducer delays relative to the accelerometer signal were measured using zero-latency *in vitro* experiments. We first determined the latency of the potentiometer by attaching the accelerometer to the same head plate as the potentiometer. Second, we performed an experiment similar to the procedure described by Crane et al. (11), in which both the accelerometer and a search coil were attached to the end of a pivoting shaft that was mounted on the chair temporarily at eye and head positions. The shaft, whose proximal end pivoted about an anchor approximating the center of the head, formed an armature that rotated the search coil around the proximal end. This rod mechanically approximated the line of sight. Care was taken to ensure that there was no slack at the pivot point so that rotatory movement imposed on the shaft would rotate the search coil and the accelerometer on the armature with zero actual time delay. Multiple trials in both directions were conducted. When the zero-latency armature data were analyzed using the 3-SDs technique (11), apparent latencies were found to be 23 milliseconds for the potentiometer and 2 milliseconds for the coil signals with respect to the accelerometer traces. These values were used to correct the measured latency values by subtraction. Noise level was 0.031 mm for the potentiometer, 0.02 degrees for coil, and 5.5 mg for accelerometer signals.

TABLE 1. *Patient characteristics and presenting symptoms*

Patient	Age (yr)	Sex	Duration (mo)	Symptoms
1	65	Male	1.5	Episodic[a] movement–associated rocking and falling sensations
2	35	Female	24	Episodes of feeling of drunkenness that worsen with head movement after a trip on an airplane
3	43	Female	32	Episodic lateropulsion to the left after a closed head injury
4	57	Male	60	Episodic anteropulsion and lateropulsion, and prolonged vertical linear movement sensation
5	36	Female	24	Episodic[a] feeling of imbalance, oscillopsia, and tilting of the floor
6	48	Male	29	Episodic[a] anteropulsion and retropulsion after a closed head injury
7	40	Male	10	Daily sudden sensations of falling forward after a boat cruise
8	40	Male	84	Imbalance, rocking sensations, and feeling of continuing movement after stopping
9	40	Male	7	Episodic anteropulsion and rocking, falling and rising sensations in supine position after a closed head injury
10	37	Female	48	Lateropulsion, anteropulsion, and retropulsion lasting seconds after a respiratory viral illness
11	57	Female	6	Drop attacks, upward motion feelings, and imbalance when walking on tilted surfaces
12	42	Female	8	Backward-falling sensations, imbalance, and oscillopsia
13	32	Male	6	Lateropulsion, falling and rising sensations

[a]Patients were symptomatic (ongoing episode) at the time the head heave tests were performed.

TABLE 2. *Results of clinical tests, audiometry, and neuroimaging*

Patient	Horizontal head thrust (Halmagyi) test	Dynamic visual acuity	Head shake nystagmus	Oscillopsia test	Dix-Hallpike maneuver	Hyperventilation test	Cerebellar signs	Audiometry	Brain MRI
1	Positive bilaterally	Reduced	Absent	Not performed	Negative	Not performed	Broad-based gait	Right high-frequency and left pan-frequency reduction	Not performed
2	Negative	Not performed	Absent	Not performed	Negative	Not performed	Absent	Normal	Normal
3	Positive left	Reduced	Absent	Positive	Negative	Positive	Absent	Normal	Not performed
4	Positive right	Not performed	Absent	Not performed	Negative	Not performed	Ataxic gait, Romberg positive to the right	Bilateral high-frequency reduction	Not performed
5	Positive bilaterally	Reduced	Absent	Positive	Negative	Not performed	Absent	Left congenital deafness, right fluctuating reduction	Nonspecific changes
6	Positive left	Reduced	Absent	Positive	Negative	Not performed	Absent	Left high-frequency reduction	Normal
7	Negative	Not performed	Absent	Not performed	Negative	Not performed	Absent	Normal	Normal
8	Negative	Not performed	Absent	Not performed	Negative	Not performed	Absent	Normal	Nonspecific changes
9	Negative	Normal	Absent	Negative	Negative	Negative	Absent	Normal	Normal
10	Negative	Not performed	Absent	Not performed	Negative	Not performed	Absent	Bilateral high-frequency reduction	Not performed
11	Negative	Not performed	Absent	Not performed	Negative	Not performed	Absent	Right pan-frequency reduction (more at low frequencies)	Normal
12	Atypical (bilateral overshoot)	Reduced	Absent	Positive	Negative	Not performed	Broad-based and ataxic gait	Normal	Not performed
13	Negative	Not performed	Absent	Not performed	Negative	Not performed	Absent	Normal	Normal

MRI, magnetic resonance imaging.

TABLE 3. *Results of standard laboratory vestibular tests*

Patient	Electronystagmography with bithermal caloric test	Horizontal head thrust test (Halmagyi-Curthoys) using search coils	Vestibular-evoked myogenic potentials	Subjective visual vertical
1	Air caloric response 5 degrees/s right and 6 degrees/s left	Bilateral pan-frequency loss	Absent bilaterally	2.8 degrees left
2	34% Right reduction	Normal	Normal	0.6 degrees left
3	Normal	Normal	Absent right	0.9 degrees right
4	25% Left reduction	Normal	Normal	0.5 degrees right
5	Severe bilateral reduction	Bilateral pan-frequency loss	Absent left	0.6 degrees right
6	29% Left reduction	Left high-frequency loss	Normal	0.2 degrees left
7	Normal	Normal	Normal	0.6 degrees right
8	Normal	Normal	Normal	0.8 degrees left
9	Normal	Normal	Normal	0.3 degrees right
10	Normal	Normal	Normal	0.7 degrees right
11	55% Right reduction	Normal	Normal	0.1 degrees left
12	39% Right, moderate bilateral reduction	Bilateral pan-frequency loss	Normal	1.8 degrees left
13	Normal	Normal	Normal	1.3 degrees left

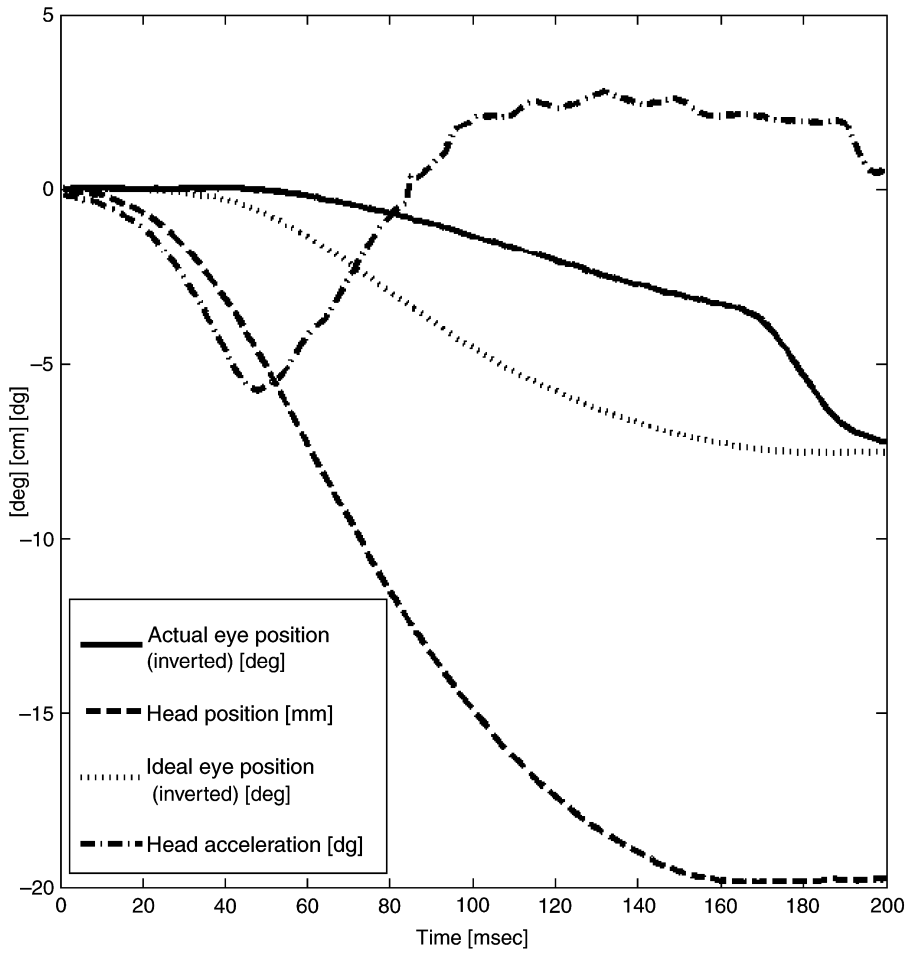

FIG. 2. Graph showing stimulus profile and response to a single head heave toward the left from a healthy control (dg indicates deci-*g*, where *g* is gravity acceleration [9.81 m/s^2]).

Statistical analyses were performed using analysis of variance for parametric data and Mann-Whitney *U* tests for nonparametric data. The sensitivity and velocity gain data of each individual patient were compared with those of the healthy group mean using the *Z* tests. Significance level was set at $p < 0.05$.

RESULTS

Patients presented with a number of symptoms suggestive of otolith diseases (Table 1). Most (10/13) reported a sensation of linear pulsion. Table 2 summarizes the results of clinical tests, audiometry, and neuroimaging. Five (38%) of 13 patients had normal standard laboratory vestibular function test results (Table 3), which included ENG with bithermal caloric, horizontal head thrust with search coils, and VEMP. In the remaining 8 patients, 4 had deficits of semicircular canal function only (i.e., reduced caloric response or positive head thrust in Patients 2, 4, 6, and 11), whereas 3 showed saccular dysfunction (i.e., absent VEMP in Patients 1, 3, and 5).

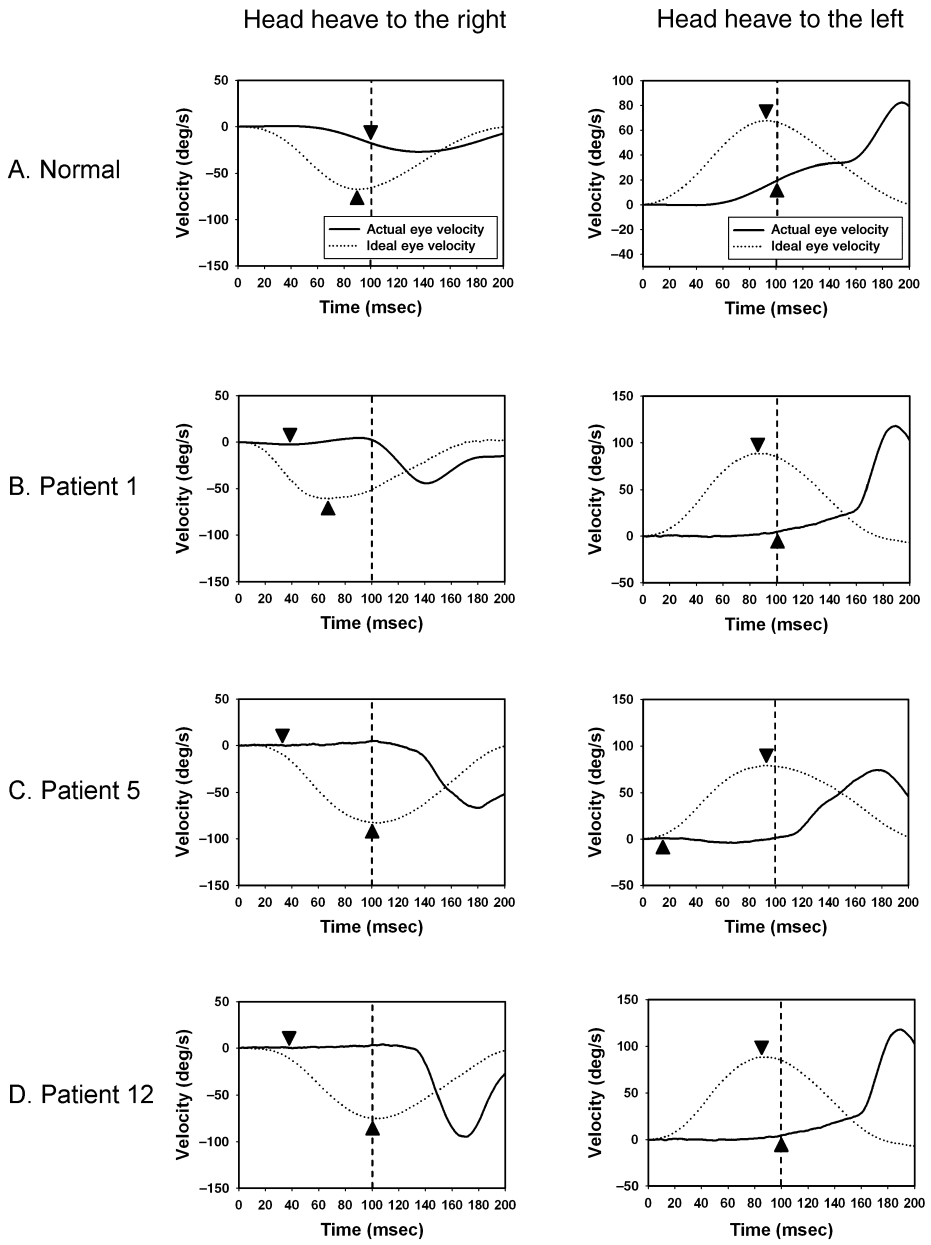

FIG. 3. Graphs showing representative velocity profiles of ideal and actual eye movements during head heave to the right and to the left during the first 200 milliseconds after head movement onset in a healthy subject (*A*) and in patients 1, 5, and 12 (*B to D*). The vertical dashed line denotes the first 100 milliseconds within which the sensitivity and velocity gains were calculated. Actual eye movements in patients that occurred after 100 milliseconds were responses after visual feedback. Arrowheads indicate the peak values of actual and ideal eye velocity in the first 100 milliseconds, which were used to calculate velocity gain.

The stimulus profile and response to a single leftward head heave from a healthy control are shown in Figure 2. Normal response to head heave consists of an open-loop initial phase, in which the tVOR response is undercompensatory with a velocity gain of about 36% (12,13), followed by a second phase, in which gaze position error generates a corrective saccade via visual feedback. Stimulus profiles did not differ significantly between patient and control groups. The median peak acceleration of the head was 0.615*g* for patients and 0.640*g* for controls (not statistically significant). The median amplitude of head movements within 100 milliseconds of their onset was 17.1 mm for patients and 17.4 mm for healthy controls (not statistically significant). The mean maximum head yaw rotation was 0.387 degree (SD, ±0.083 degree) for patients and 0.351 degree (SD, ±0.106 degree) for controls (not statistically significant).

Representative velocity profiles of actual and ideal eye movement responses during single head heaves to the right and to the left from a healthy subject and 3 patients are shown in Figure 3. In the healthy subject, responses were symmetric in both directions (Fig. 3A). The mean sensitivity in the healthy control group was 1.388 degrees/cm (SD, ±0.401 degree/cm) for rightward heaves and 1.310 degrees/cm (SD, ±0.344 degree/cm) for leftward heaves. The corresponding velocity gain values were 36.7 ± 10.6% for rightward heave and 34.9 ± 9.0% for leftward heave. These reference mean and SD values are concordant with those reported by others (12,13). One explanation for the low and variable tVOR responses in healthy subjects is that most naturally occurring head movements consist of both rotations and translations, with rotations being dominant. The pure, high-acceleration tVOR stimuli used in this experiment, however, is arbitrary, such that the brain has neither a need nor an opportunity to optimize its performance (12).

Three patients (Patients 1, 5, and 12) had significantly lower tVOR responses (i.e., mean sensitivities and velocity gains) as compared with healthy controls (*Z* test, $p < 0.05$) (Fig. 3 and Table 4). Translational VOR responses were symmetrically reduced in both directions in 2 of these 3 patients (Patients 5 and 12), who had symptoms for 8 (Patient 12) and 24 months (Patient 5). The third patient (Patient 1) also had a bilateral reduction, but the responses were more severely reduced during leftward head translation than during rightward translation. This third patient, who had symptoms for 7 weeks, was the only patient with abnormal SVV, showing a significant deviation to the left.

The mean latency of eye movement response with respect to head movement onset in healthy controls was 45.1 milliseconds (SD, ±13.8 milliseconds) for rightward heave and 46.1 milliseconds (SD, ±13.1 milliseconds) for leftward heave. This latency value is higher than those reported (11,12) previously, probably because our healthy subjects are older (14). Two of the 3 patients with decreased sensitivities and velocity gains also showed significant increase in mean latency (*Z* test, $p < 0.05$); the latency was increased bilaterally in Patient 12 and unilaterally in Patient 1 (during left heave) (Table 4).

Clinical examination (Tables 2 and 3) and routine vestibular testing in these 3 patients revealed additional associated dysfunction of the cochlea (hearing deficit in 2 patients [i.e., Patients 1 and 5]), saccules (abnormal VEMP test result in Patient 5), and semicircular canals (abnormal head impulse, oscillopsia, and post–head shake nystagmus test results, and reduced responses to caloric and horizontal head thrust stimuli in all 3 patients).

DISCUSSION

A variety of laboratory tests have been used to determine otolith function. They include oculogravic illusion with centrifugal stimulation (15), evaluation of the subjective visual vertical or horizontal (10,16), eccentric rota-

TABLE 4. *Sensitivity, velocity gain, and latency of eye responses during high-acceleration/frequency head heaves (mean ± SD)*

	Sensitivity ± SD (deg/cm)		Velocity gain ± SD (%)		Latency ± SD (msec)	
	Right head heave	Left head heave	Right head heave	Left head heave	Right head heave	Left head heave
Healthy group (n = 10)	1.388 ± 0.401	1.310 ± 0.344	36.7 ± 10.6	34.9 ± 9.0	45.1 ± 13.8	46.1 ± 13.1
Patient 1	0.303 ± 0.040[a]	0.244 ± 0.094[a]	8.4 ± 1.2[a]	6.3 ± 2.2[a]	42.0 ± 14.7	103.8 ± 64.1[a]
Patient 2	1.430 ± 0.182	1.297 ± 0.241	34.0 ± 6.5	35.4 ± 7.8	33.1 ± 6.0	34.2 ± 9.3
Patient 3	1.738 ± 0.222	1.610 ± 0.177	46.6 ± 5.8	43.4 ± 4.1	37.3 ± 7.1	43.9 ± 5.8
Patient 4	1.195 ± 0.289	1.080 ± 0.148	31.7 ± 8.2	28.3 ± 3.7	33.0 ± 2.9	48.0 ± 7.0
Patient 5	0.332 ± 0.202[a]	0.379 ± 0.070[a]	8.9 ± 5.5[a]	10.4 ± 2.3[a]	50.0 ± 20.7	71.5 ± 31.6
Patient 6	2.013 ± 0.109	1.535 ± 0.148	52.7 ± 2.6	39.9 ± 4.5	35.3 ± 3.8	49.6 ± 7.4
Patient 7	1.000 ± 0.215	1.208 ± 0.083	25.3 ± 7.2	30.8 ± 3.0	38.0 ± 3.6	38.8 ± 5.8
Patient 8	1.672 ± 0.185	1.726 ± 0.225	43.9 ± 3.9	45.5 ± 5.2	46.1 ± 10.6	37.8 ± 6.8
Patient 9	1.979 ± 0.116	1.699 ± 0.250	52.8 ± 2.5	45.5 ± 7.4	66.0 ± 4.0	51.5 ± 10.6
Patient 10	1.412 ± 0.198	1.453 ± 0.165	37.7 ± 5.3	38.2 ± 4.5	63.6 ± 16.5	66.0 ± 3.8
Patient 11	0.995 ± 0.100	1.032 ± 0.179	26.3 ± 2.5	28.3 ± 3.9	72.7 ± 42.3	73.6 ± 34.4
Patient 12	0.498 ± 0.144[a]	0.481 ± 0.089[a]	13.1 ± 3.6[a]	13.0 ± 2.3[a]	120.0 ± 5.7[a]	119.7 ± 11.7[a]
Patient 13	1.342 ± 0.137	1.079 ± 0.098	36.1 ± 3.6	28.6 ± 3.5	74.6 ± 6.7	66.7 ± 4.8

[a] $p < 0.05$ (*Z* tests).

tion (17,18), off-vertical axis rotation (19), and vestibular click- or tap-evoked myogenic potentials (9,20). Although some of these tests are tailored to assess either utricular (e.g., SVV) or saccular (e.g., VEMP) function selectively, most use head movement as a stimulus and elicit responses that are modulated by the maculae in *both* the utricle and the saccule because the maculae surfaces are curved and contain sensory cells with polarization vectors pointing to all directions in 3-dimensional space (21).

The subjective visual vertical test has been proposed to be a sensitive test for assessing utricular function. It detects erroneous, tilted perception of the true earth vertical, which might occur after a unilateral lesion to the otolith organs or to their projections to the brainstem in the early stages of disease (22). However, the abnormality of SVV often diminishes rapidly within weeks after unilateral surgical vestibular deafferentation, such that with time, only a small tilt remains (23,24). The diagnostic yield of measuring SVV is also limited because it relies on asymmetry in utricular tone. Subjective visual vertical in patients with bilateral vestibulopathy is often indistinguishable from that in healthy subjects (24). Our findings that SVV was abnormal in 1 patient who had otolith symptoms for 7 weeks but that it was normal in 2 patients who had symptoms for more than 8 months provide support that SVV may be less sensitive in detecting utricular dysfunction in patients who have central compensation of partial otolith disease over time or in patients with bilateral symmetric otolith dysfunction.

A promising approach to assess utricular function is to measure tVOR along the interaural axis during whole-body translation (25–28). In patients with unilateral vestibular nerve section, for example, tVOR response toward the operated ear was reduced acutely (27), followed by resolution of response asymmetry for 10 weeks. More recently, the head heave test has been used to stimulate the otolith maculae with lower inertia than whole-body translation by delivering transient, low-amplitude, high-acceleration, translational stimuli either manually (3) or using a head sled (2,12). The head heave test has been shown to be useful in detecting unilateral vestibular injury (2) and in prognosticating recovery in acute vestibular neuritis (3).

In this pilot study, we applied the head heave test to a small, consecutive group of patients with otolith symptoms who had no definitive diagnosis. A high proportion of this group of patients showed no abnormal findings on routine laboratory vestibular testings. This may reflect that currently available tests are inadequate to detect subtle or partially compensated otolith diseases or that some patients might have a nonorganic cause. We found that the head heave test provides additional diagnostic information about utricular function in 3 of 13 patients tested. Translational VOR responses were reduced in both directions of head heave in all 3 patients. *Symmetric* reduction was found in 2 of these 3 patients who had longer duration of symptoms, with 1 of them also having a markedly prolonged latency bilaterally. This *symmetric* reduction in responses may be caused by the central compensation of unilateral otolith disease, or it may represent bilateral symmetric otolith dysfunction. A third patient, who had a much shorter duration of symptoms, had *asymmetric* reduction in tVOR responses, *asymmetric* increase in latency, and a concordant pathologic tilt of the SVV. This *asymmetric* reduction in responses suggests asymmetric otolith disease with minimal central compensation, perhaps because of the shorter duration of symptoms. Taken together, our results indicate that the head heave test may be a useful additional laboratory tool to aid in detection of acute and chronic unilateral otolith diseases and bilateral symmetric or asymmetric otolith diseases.

The true sensitivity of the head heave test in detecting otolith disease remains to be ascertained because no test is currently considered to be the "gold standard" for the evaluation of otolith function. In addition, because of the small sample size of this pilot study, testing a much larger number of patients is needed for optimal power. We are currently recruiting a larger patient cohort and patients with specific lesions (e.g., after unilateral vestibular nerve section) to further clarify the diagnostic yield of the head heave test in the laboratory setting. The practical usefulness of the head heave maneuver, by which a head heave impulse is delivered manually, will also be investigated further in clinical settings.

Acknowledgments: The authors thank Adrian Lasker and Stefano Ramat for technical advice.

REFERENCES

1. Gresty MA, Bronstein AM, Brandt T, et al. Neurology of otolith function. Peripheral and central disorders. *Brain* 1992;115: 647–73.
2. Ramat S, Zee DS, Minor LB. Translational vestibulo-ocular reflex evoked by a "head-heave" stimulus. *Ann N Y Acad Sci* 2001; 942:95–113.
3. Nuti D, Mandalà M, Broman AT, et al. Acute vestibular neuritis: crognosis based upon bedside clinical tests (thrusts and heaves). *Ann N Y Acad Sci* 2005;1039:359–67.
4. Prepageran N, Kisilievsky V, Tomlinson RD, et al. Symptomatic high frequency/acceleration vestibular loss: consideration for a new clinical syndrome of vestibular dysfunction. *Acta Otolaryngol* 2005;125:48–54.
5. Tomlinson RD, Schwarz DW, Istl Y, et al. Quantification of the vestibulo-ocular reflex and visual-vestibular interaction for the purpose of clinical diagnosis. *Med Biol Eng Comput* 1982;20:99–107.
6. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol* 1988;45:737–9.
7. Hain TC, Fetter M, Zee DS. Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. *Am J Otolaryngol* 1987;8:36–47.
8. Burgio DL, Blakley BW, Myers SF. The high-frequency oscillopsia test. *J Vestib Res* 1992;2:221–6.
9. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry* 1994;57:190–7.
10. Böhmer A. Zur beurteilung der otolithenfunktion mit der subjektiven visuellen vertikalen. *HNO* 1997;45:533–37.
11. Crane BT, Tian J, Wiest G, et al. Initiation of the human heave linear vestibulo-ocular reflex. *Exp Brain Res* 2003;148:247–55.
12. Ramat S, Zee DS. Ocular motor responses to abrupt interaural head translation in normal humans. *J Neurophysiol* 2003;90:887–902.

13. Ramat S, Straumann D, Zee DS. Interaural translational VOR: suppression, enhancement and cognitive control. *J Neurophysiol* 2005;94:2391–402.
14. Tian JR, Crane BJ, Wiest G, et al. Effect of aging on the human initial interaural linear vestibulo-ocular reflex. *Exp Brain Res* 2002;145:142–9.
15. Curthoys IS, Dai MJ, Halmagyi GM. Human otolithic function before and after unilateral vestibular neurectomy. *J Vestib Res* 1990/91;1:199–209.
16. Clarke AH, Schonfeld U, Hamann C, Scherer H. Measuring unilateral otolith function via the otolith-ocular response and the subjective visual vertical. *Acta Otolaryngol* 2001;545:84–7.
17. Aw ST, Todd MJ, McGarvie LA, et al. Effects of unilateral vestibular deafferentation on the linear vestibulo-ocular reflex evoked by impulsive eccentric roll rotation. *J Neurophysiol* 2003;89: 969–78.
18. Clarke AH, Engelhorn A, Scherer H. Ocular counterrolling in response to asymmetric radial acceleration. *Acta Otolaryngol* 1996;116:652–6.
19. Furman JM, Schor RH, Schumann TL. Off-vertical axis rotation: a test of the otolith-ocular reflex. *Ann Otol Rhinol Laryngol* 1992; 101:643–50.
20. Halmagyi GM, Yavor RA, Colebatch JG. Tapping the head activates the vestibular system: a new use for the clinical reflex hammer. *Neurology* 1995;45:1027–9.
21. Spoendlin H. Ultrastructure of the vestibular sense organ. In: Wolfson RJ, ed. *The Vestibular System and Its Diseases*. Philadelphia, PA: University of Pennsylvania Press, 1966:39–68.
22. Halmagyi M. New clinical tests of unilateral vestibular dysfunction. *J Laryngol Otol* 2004;118:589–600.
23. Dai MJ, Curthoys IS, Halmagyi GM. Linear acceleration perception in the roll plane before and after unilateral vestibular neurectomy. *Exp Brain Res* 1989;77:315–28.
24. Tabak S, Collewjin H, Boumans LJ. Deviation of the subjective visual vertical in long-standing unilateral vestibular loss. *Acta Otolaryngol* 1997;117:1–6.
25. Bronstein AM, Gresty MA. Short latency compensatory eye movement responses to transient linear head acceleration: a specific function of the otolith-ocular reflex. *Exp Brain Res* 1988;71: 406–10.
26. Angelaki DE, Newlands SD, Dickman JD. Primate translational vestibuloocular reflexes: IV. Changes after unilateral labyrinthectomy. *J Neurophysiol* 2000;83:3005–18.
27. Lempert E, Gianna C, Brookes G, et al. Horizontal otolith-ocular responses in humans after unilateral vestibular deafferentation. *Exp Brain Res* 1998;118:533–40.
28. Crane BT, Tian JR, Ishiyama A, et al. Initiation and cancellation of the human heave linear vestibulo-ocular reflex after unilateral vestibular deafferentation. *Exp Brain Res* 2005;161: 519–26.