Comparison of Head Thrust Test With Head Autorotation Test Reveals That the Vestibulo-ocular Reflex Is Enhanced During Voluntary Head Movements

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Objectives: To compare 2 clinical tests of vestibular function, the head autorotation test (HART) and the head thrust test (HTT), and to determine why they give disparate results in patients with known unilateral vestibular deficiency (UVD) due to labyrinthectomy.

Methods: We used scleral coils to measure the horizontal (yaw) vestibulo-ocular reflex (VOR) in 5 healthy human subjects and in 11 patients who underwent labyrinthectomy. We used 2 paradigms. Using HART, subjects visually fixated a target during self-generated, swept-frequency, sinusoidal, horizontal head rotations. Using HTT, patients fixated the target during horizontal head thrusts delivered randomly in direction and time.

Results: In subjects without UVD, eye movements were almost perfectly compensatory for both paradigms. In subjects with UVD, VOR gain for ipsilesional head thrusts was low for both paradigms, but significantly ($P < .001$) higher (less abnormal) for HART ($0.60 \pm 0.13$) than for HTT ($0.14 \pm 0.13$). Contralesional gain was reduced for both, to $0.64 \pm 0.20$ for HART and to $0.57 \pm 0.17$ for HTT. Because ipsilesional and contralesional gains were not statistically different for HART ($P = .69$), comparison of VOR gains for half-cycle responses to the HART stimulus could not reliably identify the side of the known lesion. In contrast, HTT consistently identified the side of the lesion for all subjects with UVD. To investigate whether preprogramming contributes to the boost in VOR as measured by HART, we compared the gain and response delay of eye movements during actively self-generated and passively received head thrusts. For subjects without UVD, response delays were shorter for active ($6 \pm 1$ milliseconds) than for passive ($12 \pm 1$ milliseconds) HTT. For ipsilesional rotations of subjects with UVD, active HTT yielded a significantly higher gain ($0.44 \pm 0.20$) ($P < .001$) and a shorter delay ($15 \pm 6$ milliseconds) ($P < .001$) than did passive HTT ($0.14 \pm 0.13$ and $37 \pm 15$ milliseconds, respectively). Contralesional test results revealed a similar performance boost for active head movements. Data are given as mean $\pm$ SD.

Conclusion: When comparison of half-cycle gains is used to identify the lesion side, self-generated predictable head movement paradigms, such as HART and active HTT, are less accurate than passive HTT in the characterization of UVD, in part because preprogramming can augment the VOR during voluntary head movements.


Dizziness is the ninth most common reason adults visit a primary care physician, and it affects approximately 90 million Americans. Identification of the side, affected semicircular canals, and degree of unilateral vestibular deficiency (UVD) is an important goal in the examination of patients experiencing dizziness.

Two high-frequency rotational stimulus paradigms—the head thrust test (HTT) and the head autorotation test (HART)—are in wide use for assessing angular vestibulo-ocular reflex (VOR) function, yet have not been directly compared.

Researchers have advocated HTT, in which high-acceleration impulsive head rotations are delivered in the plane of one pair of semicircular canals while a subject attempts to maintain visual fixation on a distant target. Implied by the second law of Ewald, such head rotations elicit an asymmetric response in patients with UVD by maximally stimulating the neural pathway arising from one semicircular canal crista while maximally inhibiting or silencing the pathway from the coplanar canal. The approach has proved sensitive for the detection of vestibular dysfunction in subjects with known UVD. Researchers have advocated HTT, in which high-acceleration impulsive head rotations are delivered in the plane of one pair of semicircular canals while a subject attempts to maintain visual fixation on a distant target. Implied by the second law of Ewald, such head rotations elicit an asymmetric response in patients with UVD by maximally stimulating the neural pathway arising from one semicircular canal crista while maximally inhibiting or silencing the pathway from the coplanar canal. The approach has proved sensitive for the detection of vestibular dysfunction in subjects with known UVD.
A second paradigm, HART, uses self-generated, swept-frequency, sinusoidal head rotations performed while the subject visually fixates a distant earth-fixed target. Fourier analysis of system gain and phase, along with formulaic measures of response asymmetry, are applied to the measured head and eye movements. Researchers have reported extensively on this approach, and several commercially available vestibular testing systems are based on this paradigm.

We sought to compare the ability of HTT with that of HART to identify dysfunction in subjects with known UVD after surgical labyrinthectomy. There are 3 differences between these 2 stimulus paradigms that could contribute to a difference in measured VOR for a given patient. First, HTT stimuli are passive (ie, delivered by the examiner), while during HART, the head movement is active (ie, self-generated). Second, HTT stimuli are presented unpredictably in time and direction, while the sinusoidal frequency–sweep head movement during HART is predictable cycle by cycle. Third, HTT stimuli are often of higher acceleration and wider spectral bandwidth than are HART stimuli, because of some subjects’ inability to make rapid, self-generated, sinusoidal head movements.

We hypothesized that during active predictable head movements, subjects with UVD can augment apparent VOR performance by using information about the intended/expected head movement to complement deficient vestibular function and guide compensatory eye movements. Such an effect would be expected to reduce the sensitivity of tests like HART that rely on self-generated head movement stimuli for detecting vestibular hypofunction.

We tested these predictions by comparing VOR gains measured for subjects with and without UVD during HART and HTT, and by comparing subjects’ responses to active and passive impulsive head rotations.

**METHODS**

### SUBJECTS

We studied 5 human subjects without UVD and 11 with UVD due to labyrinthectomy. Subjects without UVD ranged in age from 32 to 55 years and were free of vestibular and ocular disease, except for wearing corrective lenses. Subjects with UVD ranged in age from 48 to 72 years, and the time from labyrinthectomy ranged from 3 to 120 months. All had undergone vestibular rehabilitation therapy postoperatively. The indication for labyrinthectomy was vestibular schwannoma or meningioma in 6 subjects and unilateral Meniere disease in 5. The side of labyrinthectomy was the left for 6 subjects with UVD and the right for 5. All subjects gave written informed consent, and the experimental procedures were approved by the Joint Committee on Clinical Investigation, The Johns Hopkins University School of Medicine, Baltimore, Md.

### EXPERIMENTAL TECHNIQUE

Head and eye movements were recorded in 3 dimensions using magnetic search coils embedded in contact lenses and in a bite block. The instrumentation and technique have been described in detail elsewhere. Eye and bite block angular positions were sampled at 500 Hz. The resulting signals were low-pass filtered with a single-pole analog filter with a 3-dB bandwidth of 100 Hz. Each subject was tested while seated upright and centered within a uniform magnetic field, with the interpupillary line parallel to the horizon and the Frankfort line (from the top of the tragus to the infraorbital foramen) in the plane of head rotation. All rotational stimuli were in the horizontal (yaw) plane. During each trial, the room was completely dark except for a target light-emitting diode positioned 1.24 m anterior to the center of the head, at the same elevation as the pupils. All subjects were tested more than 20 minutes after removal of eyeglasses.

Each experiment began with the subject’s head held rigidly at the starting position via connection of the bite block to a bar, while the subject performed calibration tasks. The bar was then removed, and all subsequent trials were performed with the head freely to move during a stimulus, then returning to the starting position.

For HART, subjects attempted to visually fixate the target while sinusoidally shaking their heads horizontally (ie, rotating about an earth-vertical axis through the center of the cervical spine) at maximum tolerated velocity in time with a metronome sweep logarithmically in frequency from 1 to 6 Hz for 20 seconds. Results from 3 trials were averaged.

For HTT, subjects fixated the same target during brief (100–200–millisecond) rotations in the earth-horizontal plane, at high acceleration (3000°–5000°/s²) starting from a complete stop. Head velocity reached 25° to 75°/s at 40 millisecond into the movement and 150° to 300°/s peak velocity by 100 to 120 milliseconds. The final position was 10° to 15° from center. For passive HTT (pHTT), stimuli were delivered manually at an unpredictable onset time and in a randomly varied direction, starting from center. The subject received no cues about the direction or time of an impending passive head thrust. For active HTT (aHTT), the subject generated the head thrust voluntarily. Only head movements reaching greater than 100°/s within the first 60 milliseconds were included in the analysis. A typical trial lasted about 3 minutes and included about 20 to 40 head thrusts to each side.

### RESULTS

**HEAD AUTOROTATION TESTING**

Figure 1 shows HART results for a healthy 33-year-old man without
UVD. In the trial shown, head velocity ranged from 40°/s at 1 Hz to approximately 180°/s at 6 to 8 Hz (Figure 1A). Eye velocity traces (inverted for comparison with head velocity) are nearly identical to head velocity traces, to within less than the 4°/s of retinal image slip for which visual acuity begins to degrade (Figure 1B). When plotted as eye velocity vs head velocity, these data lie almost exactly along a y = −x line, consistent with a nearly perfect VOR (Figure 1C). This subject without UVD has rightward and leftward VOR velocity gains of 1.0±0.05 and 1.0±0.02, respectively.

Figure 2 shows HART results for a symptomatically well-compensated 63-year-old man who had undergone right translabyrinthine vestibular schwannoma resection and postoperative vestibular rehabilitation 5 months before testing. For this subject, the response during head turns toward the contralesional left side (Figure 2A and B) is somewhat degraded, but not significantly ($P=.07$) different from normal (gain, 0.87±0.13). During rightward head turns, there is a significantly ($P<.001$) abnormal response, with a velocity gain of 0.43±0.19 and retinal slip errors of up to 100°/s. Retinal slip not only degrades foveal visual acuity but also accrues to cause the subject to lose target fixation, eliciting large saccadlike corrective eye movements to reacquire the visual target. For this subject, HART results clearly reveal an asymmetry.

Such asymmetries between responses to ipsilesional and contralesional head rotations were not always apparent. Figure 3 presents the HART responses from a 68-year-old man tested 38 months after undergoing surgery resulting in left UVD. At maximum effort, this patient achieved slower peak head velocity than the subject shown in Figure 2. The slow-phase components of the response to ipsilesional rotations were not markedly different from those observed in the contralesional half cycles. However, a gaze-correcting saccadic eye movement was often observed in the ipsilesional responses.

Figure 4A and B shows a summary of VOR gains measured using HART in 5 healthy subjects and in 11 subjects with UVD, respectively. The VOR gains for subjects without UVD are segregated into rightward and leftward head movements and illustrated separately as a check of test reliability. The VOR gains for subjects without UVD are 0.95±0.13 and 1.03±0.11 for rightward and leftward head rotations, respectively. There was no significant difference between right and left VOR gains.
(P = .13), and the combined data set was insignificantly different from 1.0 (P = .64) (95% confidence interval, 0.93-1.05). The HART gains for subjects with UVD were segregated into ipsilesional and contralesional directions. Four findings were notable. First, the ipsilesional gain of 0.60 ± 0.13 was significantly lower than normal (P < .001). Second, there was a wide distribution of ipsilesional gains for this sample population, ranging from 0.2 to 1.0. Third, the contralesional gain of 0.64 ± 0.20 was also significantly decreased from normal (P < .001). Finally, HART gains were not significantly different for ipsilesional and contralesional rotations in these subjects with UVD and, therefore, could not reliably indicate the side of the known lesion (P = .69).

**PASSIVE HTT**

Passive HTT was performed on 4 of the subjects without UVD and on all 11 subjects with UVD who had been studied using HART. **Figure 5A** shows head velocity and concurrent eye velocity traces for 2 representative head thrusts from the same subject without UVD who was described in Figure 1. The high-acceleration head rotation transients reach a peak velocity of greater than 200°/s within 80 to
never complained of losing sight of respectively. Subjects without UVDject were 1.00±0.04 and 0.99±0.06, ward and leftward gains for this sub-

nearly perfect performance. Right-
to a velocity reveals that all traces lie close
throughout most of each trace. In Fig-

tency, retinal slip is kept below 5

Figure 4C and D shows a summary of pHTT VOR gains for 4 subjects without UVD and 11 subjects with UVD, respectively. (The same subjects were used for pHHT and HART, except for 1 subject with-

out UVD who was tested only with HART.) Right and left pHTT re-
sponses of subjects without UVD are shown separately in Figure 4C. Rightward (0.94±0.04) and left-
ward (0.90±0.05) pHHT gains were not significantly different (P=.27) for subjects without UVD. For sub-
jects with UVD (Figure 4D), gains were decreased for ipsilesional and contralesional head rotations. In con-
trast to the HART VOR gains for the same population of subjects, there was a marked asymmetry be-
tween ipsilesional (0.14±0.13) and contralesional (0.58±0.17) gains. For every patient with UVD tested, ipsilateral gains were significantly lower than contralesional gains (P<.001). Ipsilesional and contra-
lesional gains were each significantly different from normal (P<.001).

Comparison of HART and pHHT results reveals that although the ipsilateral VOR gains are re-
duced for the HART paradigm ap-
plied to subjects with UVD, a simi-
lar reduction in contralesional gains was such that HART could not re-
liably distinguish the side of the

known lesion. In contrast, there was a marked and consistent asymme-
try in the VOR gains of subjects with UVD measured using the pHHT paradigm, with the ipsilateral gains close to 0 and the contralesional gains close to gains measured using

HART.

Figure 5B shows multiple head thrust stimuli and the corresponding eye movement responses for the first 80 milliseconds of each. Peak head ac-
celerations ranged from approximately 3000° to 5000°/s². Although there is a slight early mismatch of eye and head traces because of VOR la-

Figure 6A, a rightward head thrust is tracked fairly well by the eye, al-
though the VOR gain is less than nor-
mal and a small catch-up saccade-
like movement is required to correct the final eye position after the head movement ends. For a leftward head thrust, the VOR response decrease is more pronounced, as are the promi-

ent catch-up eye movements. The second and third catch-up move-
ments occur after the head stops mov-
ing; these are the corrective eye move-
ments an observer can detect when using HTT as part of the physical ex-
amination. Figure 6B shows the first 80 milliseconds of multiple stimuli for this subject. For rightward head rotations, the eye tracks the head fairly well; however, the initial VOR-
mediated eye movement response for leftward head thrusts is of low amplitu-

d. Figure 6C reveals an asymmetry in the eye movement responses to contralesional and ipsi-

lesional head thrusts, correspond-
ing to the measured VOR gains of 0.50±0.15 and 0.06±0.09, re-

spectively. Figure 4C and D shows a summary of pHHT VOR gains for 4 subjects without UVD and 11 subjects with UVD, respectively. (The same subjects were used for pHHT and HART, except for 1 subject with-

Whereas the ipslesional pHHT re-
sponse was essentially nonexistent for the first 80 milliseconds after stimu-
ulus onset (Figure 6B), the aHTT response VOR gain is enhanced, to 0.34±0.06, from 0.06±0.09 (Figure 7A). The contralesional VOR gain is also increased, to 0.74±0.05, from 0.50±0.15. Similarly, the time from stimulus onset to threshold re-
sponse is shorter for aHTT than for pHHT.

Figure 8 shows pHHT and aHTT VOR gains for 3 subjects with-

out UVD and 10 subjects with UVD (the same subjects used for HART except for those who did not gen-

erate aHTT head movements simi-
lar to those of pHHT). For subjects without UVD (Figure 8A), re-
sponses to aHTT and pHHT were not significantly different from each other or from 1.00 (P>.05 for all). For subjects with UVD, there was a significant (P<.001) increase in ap-

parent ipsilesional VOR gain when measured using the aHTT para-
digm, to 0.44±0.20 (from 0.14±0.13 for pHHT) (Figure 8B). The con-
tralesional VOR gain also increased significantly (P=.004) under aHTT conditions, to 0.81±0.14 (from

0.58±0.17 for pHHT) (Figure 8C). This level was not significantly different from the VOR gains mea-
sured in subjects without UVD using pHHT (P=.08).

Response Delay

To better identify why the VOR is ap-

pearedly enhanced when measured by the HART paradigm, we com-
pared VOR gains measured in subjects with and without UVD using head thrusts that were either pas-
vively and unpredictably received by the subjects as in the usual ap-
lication of the test (pHTT) or ac-
tively generated by the subjects (aHTT). Analysis was limited to ac-
mulated VOR responses of subjects without UVD are

ipsilesional (0.58±0.17) gains. (the same subjects used for HART et al⁸ to define stimulus and re-
sponse onset and to compute a re-
sponse delay. Figure 9 shows pHHT and aHTT VOR response delays for 4 subjects without UVD and 11 subjects with UVD. For subjects with-

out UVD (Figure 9A), the response delay was 11.0±3.3 milliseconds for pHTT and slightly, but signifi-
cantly lower, at 5.0±1.8 milliseconds for aHTT (P=.006). For sub-
jects with UVD, the response delay for ipsilesional head thrusts was sig-
ificantly shorter for aHTT (19±16 milliseconds) than for pHHT (42±17 milliseconds) (P=.006) (Figure 9B). The response delay in subjects with UVD for contralesional head thrusts was also shorter for aHTT (11.0±5.1 milliseconds) than for pHHT
Figure 5. Passive head thrust test results for a healthy subject without a unilateral vestibular deficiency (UVD) (same subject as in Figure 1). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject has nearly perfect overlap of head and eye traces. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Traces nearly overlie each other for this subject without UVD. C, Eye velocity vs head velocity. Data lie closely along a $y = -x$ line, consistent with a near-perfect vestibulo-ocular reflex.

Figure 6. Passive head thrust test results in a subject with a unilateral vestibular deficiency (UVD) (same subject as in Figure 3). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject with a left UVD follows rightward head movement moderately well, but tracks leftward head thrusts poorly. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Response is delayed and of decreased magnitude, mildly for rightward head movements and dramatically for head movements toward the lesion side. C, Eye velocity vs head velocity for the first 80 milliseconds of responses.
(15.0±4.5 milliseconds), but the difference was not significant ($P = .07$) (Figure 9C).

**COMMENT**

**CLINICAL TESTS FOR IDENTIFICATION OF UVD**

Traditional caloric and rotary chair tests for the identification of UVD fail to test the vestibular system using physiologically appropriate stimuli over the range of frequencies and accelerations for which the system apparently evolved. Caloric testing has the advantage of being strictly unilateral, but uses an unnatural stimulus (a thermal gradient) that, as measured by the eye movements it elicits, is the equivalent of a low frequency (0.025 Hz) and low amplitude (approximately 50°/s) head rotation. The caloric response can be altered by anatomic changes in the external ear canal and by individual variation in temporal bone anatomic features, and it only evaluates 1 of the 5 vestibular receptors (the lateral semicircular canal). Rotary chair vestibular testing is more physiologic; however, at the lower frequencies and velocities typically used in clinical vestibular laboratories, rotary chair tests are insensitive in the identification of chronic total unilateral vestibular hypofunction. The sensitivity of rotary chair testing can be improved by comparison of responses to steps of acceleration with the head up and head down.

In contrast, high-frequency and high-acceleration rotational stimuli unmask the inherent asymmetry in the vestibular system, as described by the second law of Ewald—that the excitatory responses of vestibular pathway neurons encode motion over a larger dynamic range than do inhibitory responses. The eye movements elicited by such stimuli are principally due to excitation of the vestibular pathway arising from the canal ipsilateral to the direction of head acceleration and are, therefore, sensitive to a unilateral peripheral vestibular loss in that canal.

There are 2 high-frequency rotational stimuli in clinical use, pHTT and HART. The main objective of this study was to compare these 2 stimuli for the identification of unilateral vestibular hypofunction. The pHTT is a single, passive (operator delivered), unpredictable, high-acceleration (2000°-4000°/s²), low-amplitude (15°-30°) head rotation in the direction of a single semicircular canal. The HART is a continuous, active (self-generated), sinusoidal head rotation that begins slowly and becomes faster, covering a frequency range of about 1 to 6 Hz. During each test, subjects are required to fixate a visual target in front of them, and their ability to maintain visual fixation is accepted as a measure of VOR function. However, because HART is actively generated, non-VOR processes, such as predictive eye movements driven by an “efference copy” neural representation of the intended head movement, could contribute to maintaining gaze stability. As a result, VOR function could seem artificially enhanced, making HART a less sensitive and less accurate test of UVD.

We tested 11 patients who had undergone surgical labyrinthectomy and compared their responses with those of 5 subjects without UVD. The VOR gains for subjects without UVD using pHTT were insignificantly different from an ideal gain of 1.0, consistent with findings from previous studies. For subjects with UVD, the VOR gains of 0.14±0.13 for ipsilesional head
thrusts and $0.58 \pm 0.17$ for contralateral head thrusts were significantly different, and the side with the lesion was clearly identified by pHTT in each patient, consistent with previous studies.6,24 For HART, the VOR gain in subjects without UVD was essentially perfect, as one would expect. Surprisingly, however, although some subjects with UVD showed obvious asymmetry of VOR half-cycle gains on HART, there was no significant difference between the ipsilesional and contralesional VOR gains over the population with UVD ($P = .69$).

Other features of the response to the actively generated HART stimulus did provide an indication of the side of unilateral vestibular hypofunction. The occurrence of rapid, gaze-correcting, saccadic eye movements during head movements toward the side of the lesion, when they occur, is a reliable indication of the side of UVD. A bias velocity in the eye movement response during the HART stimulus (a direct current shift in the eye velocity trace toward the side of the lesion) pro-

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**Figure 8.** The vestibulo-ocular reflex (VOR) gains for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For healthy subjects without UVD, responses to active and passive HTT were not significantly different from each other or from 1.00 ($P > .05$ for all). In B, active HTT ipsilesional VOR gain is significantly ($P < .001$) higher than for passive HTT. In C, contralesional UVD VOR gain is also significantly ($P = .004$) closer to normal for active than for passive HTT. Bars represent mean±SD.

**Figure 9.** The response delays for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For subjects without UVD, the response delay was shorter for active than for passive HTT ($P = .006$. In B, for ipsilesional stimuli, response delay was significantly shorter for active HTT than for passive HTT ($P = .006$). In C, the response delay for contralesional stimuli was not significantly different ($P = .07$) between passive and active HTT. Bars represent mean±SD.
vides another such indication. Head autorotation is, therefore, a useful paradigm, but comparison of VOR gains from half-cycle analysis of HART responses is not reliable for the identification of the side of UVD.

MECHANISMS FOR IMPROVED RESPONSES TO ACTIVE HEAD ROTATIONS

There are 2 main differences between HART and pHTT stimuli that might explain the improvement in VOR gain of patients with UVD for ipsilesional head movements during HART. First, the peak velocities and accelerations generated by the examiner in pHTT are greater than those that some patients are able to generate during HART. This constraint on stimulus intensity may limit HART’s ability to discern unilateral weakness in such patients. In contrast, the stimulus used for pHTT is generated by the examiner and reaches a peak acceleration of 3000°/s² and a peak velocity of 250°/s. Although these head movements have low amplitude in displacement (10°-15°) and are well tolerated by patients, they are sufficient to elicit excitation-inhibition asymmetry and, thus, selectively probe the function of the excited canal.

A second difference is that the pHTT is passive, transient, and unpredictable, whereas the HART is a self-generated, sinusoidal, predictable stimulus. Self-generated, sinusoidal, steady-state rotations could allow for nonvestibular eye movement systems (eg, predictive eye movements, efference copy, and visual-following mechanisms) to contribute to the response. The existence of predictive mechanisms in augmenting vestibular responses is suggested by measures of visual acuity during head movement. Visual acuity is improved during active compared with passive head movements. To further investigate these potential effects of self-generated stimuli, we asked patients to perform the HTT themselves, aiming to mimic the speed and amplitude of the operator-delivered head thrusts. Although patients had some difficulty reaching the top speed of operator-delivered stimuli (about 300°/s), they did reach speeds of approximately 200°/s, and we compared responses for active and passive head thrusts of similar peak velocity and acceleration. In subjects without UVD, the VOR gain was already insignificantly different from 1.00 for pHTT, so no significant change was observed for aHTT. For subjects with UVD, however, there was a marked improvement in VOR gain during active head movements, and the boost in VOR gain during active thrusts occurred from the onset of the head thrust, during the initial 20 to 40 milliseconds. The only 2 oculomotor systems with a latency of less than 40 milliseconds are the direct VOR and predictive eye movements. Saccades, the cervico-ocular reflex, and visual-following mechanisms, such as smooth pursuit, all have latencies that are 70 to 150 milliseconds. From previous studies, it has been shown that the 3-neuron VOR arc generates the response to pHTT, so the boost in gain during aHTT is, therefore, likely to be a result of predictive eye movements.

To study the effect of prediction, we measured the time between the onset of head rotation and the onset of eye rotation for active and passive head thrusts. We used manually applied passive head thrusts to approximate the usual clinical application of pHTT and the movements our subjects made during aHTT. Manual thrusts do not have an onset sharp enough or an acceleration constant enough to precisely measure VOR latency (estimated at 8.6 milliseconds in subjects without UVD by Collewijn and co-workers using a torque-applying helmet in an HTT-type paradigm). To obtain a measure of response timing, we used the method of Tabak et al to define stimulus and response onset times (at which head and eye velocities cross set thresholds) and compute a response delay. This response delay probably does not precisely equal the true synaptic and axonal conduction delay of the VOR, because the finite rate of change of acceleration for manually applied stimuli and responses reduces the precision with which onset times for head and eye movements can be measured. However, this response delay provides a useful measure for comparison of responses to passive and active head thrusts.

For subjects without UVD, the response delay was shorter for active than for passive head thrusts. For subjects with UVD, the response delay during ipsilesional head thrusts was significantly longer for passive thrusts than for active thrusts. A similarly prolonged response delay for passive ipsilesional head rotations has been reported by Tabak et al. The apparent reduction in the response delay to aHTT might be further evidence of preprogrammed eye rotation. Alternatively, some of the apparent difference in response delay may be due to a difference in VOR gain for the first 40 milliseconds of the response. The initial low velocity eye rotation makes it difficult to precisely define the onset of the eye rotation, and could give the appearance of a delayed onset.

TIMING OF CORRECTIVE EYE MOVEMENTS

Further evidence for preprogramming of eye rotations during active head thrusts was the reduced latency of rapid corrective eye movements during active compared with passive head thrusts. To compensate for a deficient VOR, patients with UVD used rapid eye movements to correct accrued gaze error and reacquire visual fixation of the target. During active head thrusts, these corrective movements occurred as early as 60 milliseconds after the onset of the head rotation, significantly earlier than during passive head thrusts (Figure 10). Under normal circumstances, the latency of voluntary true saccadic eye movements is 200 milliseconds. This latency can be reduced to 100 milliseconds if the subject is trained in a specific task (so-called express saccades). Tian et al have shown that under pHTT conditions, these rapid eye movements have latencies shorter than those of express saccades. Our data indicate that these latencies can be reduced even further under conditions of active head movement.

CONCLUSIONS

Gain asymmetries on the pHTT reliably detected the presence of
known unilateral vestibular lesions in all patients with UVD tested, whereas the half-cycle gains measured from HART did not. Relative to pHTT, HART gains overestimated ipsilesional vestibular function and were, therefore, a less accurate indicator of unilateral hypofunction.

For subjects with UVD who are unable to generate high-acceleration sinusoidal head movements, HART may underestimate hypofunction simply because it relies on stimulus accelerations inadequate to silence inhibitory pathways arising in the intact ear. In contrast, the high-acceleration movements of pHTT reliably identify response asymmetry by more selectively probing the function of the excited canal. However, even for head movements of similar acceleration and time course, the measured VOR during self-generated head rotations (aHTT) has significantly higher gain and shorter response delay than does the VOR to passive unpredictable stimuli (pHTT).

Subjects with UVD may use a variety of strategies for improving visual fixation, including preprogrammed eye movements designed to augment the deficient VOR and to compensate for a planned or anticipated head movement. Tests using passive high-acceleration head thrusts delivered unpredictably in time and direction should, therefore, be more sensitive for discerning VOR hypofunction than tests using active and/or predictable stimuli.

Accepted for publication February 18, 2002.

This study was supported by the Garnett Passe and Rodney Williams Memorial Foundation, Parkville, Australia (Dr Cremer); the Royal Australasian College of Physicians, Sydney, New South Wales (Dr Cremer); grants K23 DC00196 (Dr Carey) and R01 DC02390 (Dr Minor) from the National Institutes of Health, Bethesda, Md; and a Johns Hopkins Clinician-Scientist Award (Dr Carey).

We thank Grace C. Y. Peng, PhD, David S. Zee, MD, Adrian G. Lasker, MS, and Dale C. Roberts, MS, for their assistance with data acquisition; and John K. Niparko, MD, and Howard W. Francis, MD, for their referral of subjects.

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