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Compensatory saccades benefit from prediction during head impulse testing in early recovery from vestibular deafferentation

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Abstract The head impulse test (HIT) can identify a deficient vestibulo-ocular reflex (VOR) by the compensatory saccade (CS) generated once the head stops moving. The inward HIT is considered safer than the outward HIT, yet might have an oculomotor advantage given that the subject would presumably know the direction of head rotation. Here, we compare CS latencies following inward (presumed predictable) and outward (more unpredictable) HITs after acute unilateral vestibular nerve deafferentation. Seven patients received inward and outward HITs delivered at six consecutive postoperative days (POD) and again at POD 30. All head impulses were recorded by portable video-oculography. CS included those occurring during (covert) or after (overt) head rotation. Inward HITs included mean CS latencies (183.48 ms ± 4.47 SE) that were consistently shorter than those generated during outward HITs in the first 6 POD ($p = 0.0033$). Inward HITs induced more covert saccades compared to outward HITs, acutely. However, by POD 30 there were no longer any differences in latencies or proportions of CS and direction of head rotation. Patients with acute unilateral vestibular loss likely use predictive cues of head direction to elicit early CS to keep the image centered on the fovea. In acute vestibular hypofunction, inwardly applied HITs may risk a preponderance of covert saccades, yet this difference largely disappears within 30 days. Advantages of inwardly applied HITs are discussed and must be balanced against the risk of a false-negative HIT interpretation.

Keywords Compensatory saccades · Vestibular · Deafferentation · Latency · Prediction

Introduction

The head impulse test (HIT), first described by Halmagyi and Curthoys in 1988 [1], assesses the function of the vestibulo-ocular reflex (VOR). This reflex helps to stabilize gaze during rapid head movements. During the test, the patient is asked to fixate a target in front of him

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and the examiner rotates the head rapidly (~ 300°/s) and unpredictably in the plane of the semicircular canal being tested [2]. If the VOR is intact and properly calibrated by the brain, the eyes rotate in perfect opposition to the head motion (i.e., with equal amplitude and in the same plane, but in the opposite direction) enabling a VOR gain ratio of eye to head velocity of 1.0. When the VOR is reduced as occurs in vestibular deafferentation surgery, the eyes are pulled by inertial forces in the same direction as the head, causing the patient to lose fixation on the target. Typically, the patient will make a corrective saccade (rapid resetting eye movement) back to the fixation target [3]. It is this "compensatory saccade" that is detected qualitatively by expert examiners at the bedside as an indirect marker of VOR hypofunction. The HIT is therefore an important bedside test for the diagnosis of vestibular hypofunction and is suggested as an important aid differentiating benign peripheral vestibular deficits from dangerous vestibular strokes [4, 5].

There are two accepted methods for performing the HIT (Fig. 1). As originally described, the head was first displaced laterally then rapidly rotated back to the center [1], a technique that we refer to as an 'inward' HIT. The alternative is to begin with the head in the neutral position and rapidly rotate the head toward either side [6], which we will refer to as an 'outward' HIT.

There are theoretical reasons to favor one or the other technique and clinical interpretation often requires the examiner to distinguish compensatory saccades from nystagmus beats, which can be aided by comparison of rightward vs. leftward responses. For example, inward impulses may have greater ease of interpretation in patients with acute unilateral peripheral vestibulopathy, where spontaneous nystagmus will vary in intensity depending on orbital eye position (Fig. 1) [7]. During an inward head impulse to the right or left, the eyes should be end positioned in the center of the orbit, which may reduce any confounding introduced by the gaze position-dependent nystagmus (i.e., Alexander's law) [8]. By contrast, outward impulses offer the possible benefit of being less predictable by the patient, since the neck position offers no clue as to the direction of the next impulse. This in theory should reduce the risk of a covert (hidden) compensatory saccade occurring during the head impulse, which typically is invisible to the examiner and can lead to erroneous interpretation.

The impact of choosing inward vs. outward HIT in acute unilateral vestibulopathy is unknown. We sought to investigate these effects on compensatory saccade latencies in acute vestibulopathy by studying patients immediately following elective vestibular deafferentation surgery. We hypothesized that inward HIT would increase predictability

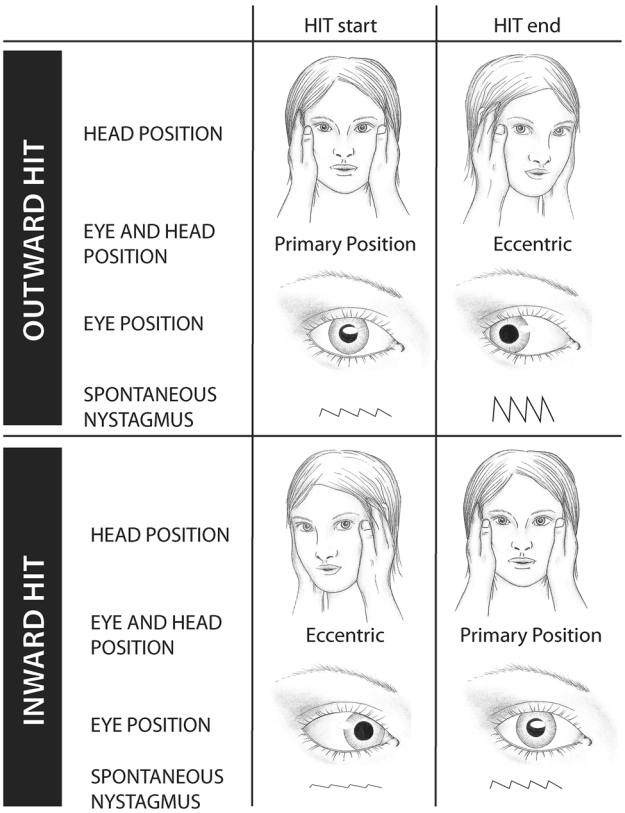

Fig. 1 Two head impulse techniques are shown in a patient with left unilateral vestibular deficit: inward HITs toward the deficient labyrinth with an eccentric head and eye position before the HIT, and outward HITs to the left, where the head and eye start moving from a centered position. The final resting head and eye position, which is eccentric in outward HITs, is important. Such an eccentric eye position would enhance an underlying spontaneous nystagmus according to Alexander's law and, thus, potentially confound test interpretation

of the head rotation, leading to a decrease in the latency of the compensatory saccades.

Materials and methods

Study population

We enrolled seven patients (Table 1) who were scheduled to undergo labyrinthectomy, neurectomy or acoustic neuroma resection resulting in a deafferentation of the vestibular nerve. All enrolled patients were tested pre- and postoperatively on six consecutive days and 1 month after surgery. All patients had normal vision based on static visual acuity testing, and no patient had evidence of a vertical skew deviation or spontaneous nystagmus preoperatively. All patients provided written informed consent as approved by the Johns Hopkins University School of Medicine institutional review board.

Table 1 Enrolled patients and characteristics

Age	Gender	Diagnosis	Intervention	Preop mean ipsilesional VOR gain	Preop mean contralesional VOR gain	Preop imbalance?	Comments
27	F	Right vestibular schwannoma	Resection via retrosigmoid approach	0.85	0.90	Yes	Right facial numbness
34	F	Left vestibular schwannoma	Resection via retrosigmoid approach	0.87	0.88	No	NA
61	M	Left vestibular schwannoma	Resection via retrosigmoid approach	1.01	0.98	No	Radio-surgery 4 years ago
42	M	Right vestibular schwannoma	Resection via retrosigmoid approach	0.73	0.83	No	NA
62	F	Left vestibular schwannoma	Resection via retrosigmoid approach	0.85	0.91	Yes	Borderline unilateral caloric weakness (21 %), tinnitus
69	M	Left Meniere's disease	Labyrinthectomy	0.6	0.83	Yes	Prior intratympanic gentamicin, tinnitus
53	F	Right Meniere's disease	Labyrinthectomy	N/A	N/A	Yes	Prior intratympanic gentamicin, migraines, tinnitus

VOG device

Patients consented to receive repeated VOR tests (head impulse tests). We quantitatively assessed the horizontal HIT (h-HIT) by using a portable video-oculography device (EyeSeeCam^{TM}) [9] which consists of lightweight infrared goggles with built-in rate (velocity) and acceleration sensors. This video-oculography device has been previously validated in laboratory settings for recording head impulse data against gold standard scleral search coils [10, 11]. Two different start positions of ipsilesional h-HIT techniques were applied to compare compensatory saccade latencies.

Two h-HIT techniques

We captured horizontal HIT measurements while patients were either seated in bed or in a chair. One single examiner (GM) collected all data while sitting in front of the patient. Test subjects were asked to fix their gaze on a distant target (>1.5 m) on the opposite wall, looking around or over the shoulder of the examiner. Eye position was calibrated using laser targets projected forward from the goggles. After calibration, examiners performed at least ten HITs using both inward (i.e., lateral-to-center head rotations) technique and outward (i.e., center-to-lateral head rotations) technique at a pseudo-random sequence. The cadence and velocity of applied HITs was also random. The examiner placed his hand on the patient's jaw, slightly positioned on the right side of the patient taking care not to obscure the fixation target and not to touch the goggles (Fig. 1) [1]. Target peak head velocity was between 90 and 200°/s (monitored by the goggles) and head displacement ranged between 5° and 20°.

Outcome measures

All h-HITs collected were re-assessed by human raters 'off-line' by processing raw quantitative data exported from the EyeSeeCam^{TM} device using Matlab R2012b (Mathworks, Natick, MA). The start and the end of the saccade was identified by using an acceleration criterion (>2000°/s^2). All detected saccades were manually verified by visual inspection. The saccade latency was defined as the time from the onset of the head impulse to the onset of the saccade. The beginning of a head impulse was defined as the time when head velocity exceeded 20°/s [10]. We measured the latency of a compensatory saccade during or immediately after an abnormal slow phase VOR [12]. Compensatory saccades occurring during the head rotation were defined as covert saccades; those occurring shortly after the head stopped moving were labeled overt [3].

Statistics

Descriptive statistics and a mixed effects model with random intercepts were used to account for correlated (repeated measures) data. We adjusted for peak head velocity, since saccade latency was a function of HIT velocity [3]. We compared differences in saccade latency from POD 2 through 5, and POD 30 because we had complete data from all patients on those days. Results were considered statistically significant at alpha <0.05. All statistical analyses were performed in R (Version 2.15.2, Vienna, Austria).

Results

We performed 626 outward HITs and 452 inward HITs on six consecutive days and POD 30. The mean outward head velocity was 149 ± 43°/s SD versus mean inward head

velocity 157 ± 53°/s SD (Table 2). Figure 2a, b shows one example of HITs performed at POD 5 on the same individual using both techniques. Across all subjects, the average saccade latency (overt and covert) at the completion of surgery (POD 0) for outward HITs was 196.94 ± 8.58 ms SE. This latency declined on average about 13.46 ± 2.00 ms SE ($t = -6.71$, $df = 229$, $p < 0.0001$) per day after surgery for outward HITs. In contrast, the average saccade latency at POD 0 was 183.48 ± 4.47 ms SE for inward HITs and declined on average about 13.26 ± 4.47 ms SE per day.

Inward HITs and their related saccade latencies were on average 13.25 ms ± 4.46 SE lower than those recruited with an outward HIT during the first 6 days after surgery ($t = -2.968304$, $df = 229$, $p = 0.0033$, Fig. 3). The latency difference between the two HIT techniques remained significantly different over the time period of 6 days (Fig. 3), but by POD 30 there was no significant differences in latency (7.64 ms ± 4.31 SE; $t = -1.77289$, $df = 291$, $p = 0.0773$).

Outward HITs induced more overt saccades (Fig. 2a) compared to inward HITs (Fig. 2b). After vestibular nerve deafferentation, the average proportion of overt saccades during outward HITs was 85 %, which declined to 30 % after 6 days (Fig. 4). At POD 5, the proportion of overt saccades was <50 %. In contrast, for inward HITs the proportion of overt saccades was smaller (37 %) and declined to 12 % after 6 days (Fig. 4). At POD 30 days, the range of overt saccades was between 0 and 20 % for outward HITs and between 0 and 4 % for inward HITs.

Table 2 Mean peak head velocities from POD 2–5

POD	Mean peak head velocity (°/s)	
	Outward HIT	Inward HIT
2	133.2301	119.6411
3	141.2451	139.7471
4	129.6942	135.6806
5	148.7959	150.8073

Discussion

Our data show that in acute UVD, inward HITs result in shorter-latency compensatory saccades with a higher proportion of them being covert than those compensatory saccades occurring during outward HIT. This finding suggests that knowledge of the suspected head direction (i.e., prediction) provides an oculomotor advantage in the immediate postoperative days following UVD. As expected, saccade latency decreased over time during recovery, as patients centrally adapted to their new vestibular deficit. Interestingly, the inward vs. outward difference largely disappeared by POD 30, suggesting that central adaptation plays an important role in compensatory saccade latency with HIT, regardless of the starting neck position. These findings have important clinical implications, suggesting that the risk of erroneously identifying a 'normal' HIT is higher when the inward technique is used in a 'predictable' manner, within an acutely deafferented patient population.

Overall, saccade latencies decreased over time, which is believed to be part of a central vestibular compensation [13]; however, our data suggest that in the acute time frame following vestibular deafferentation (or perhaps any cause of acute vestibular asymmetry), the direction of head

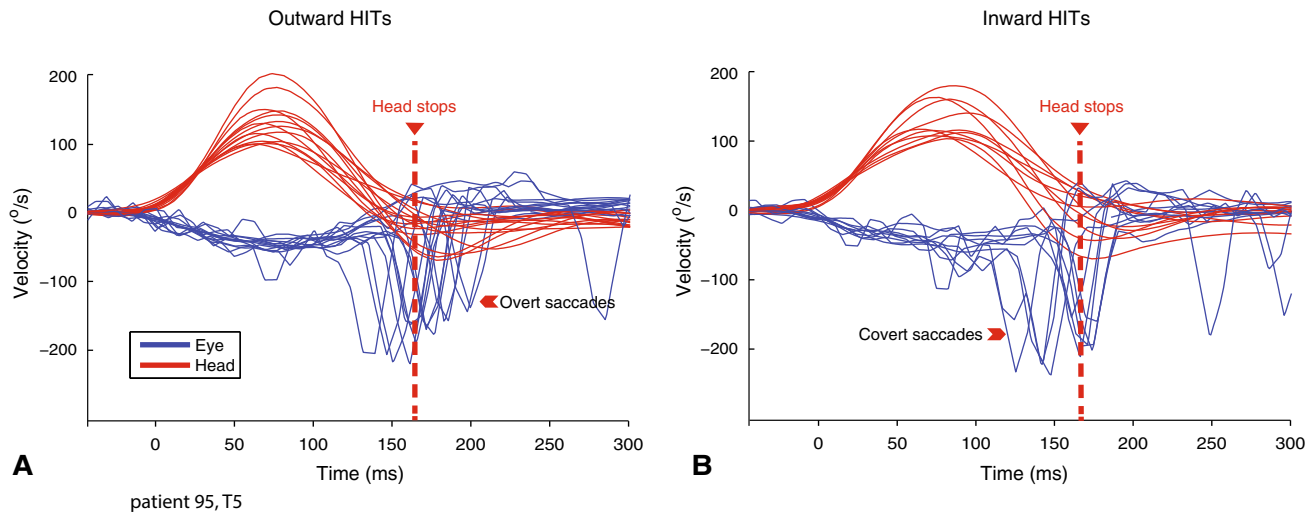

Fig. 2 One example of horizontal HITs is shown in one patient 5 days after vestibular deafferentation for outward and inward HITs. The saccade latency is shorter for inward HITs and the proportion of covert saccades is higher

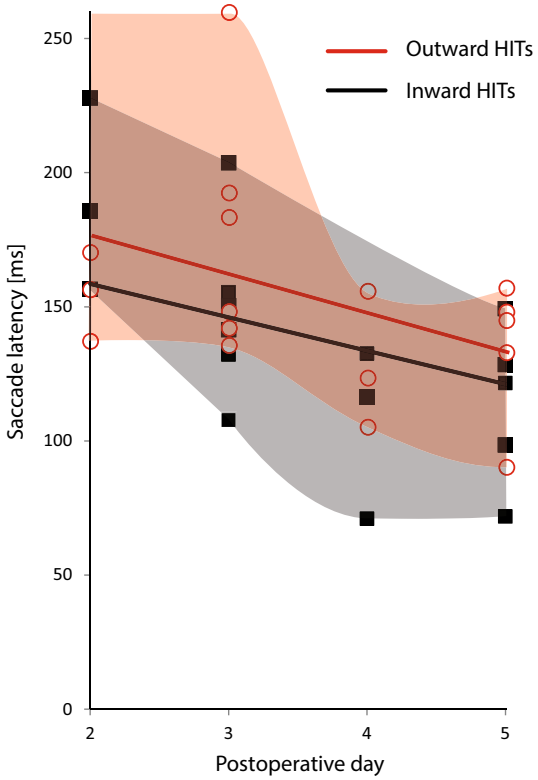

Fig. 3 Absolute saccade latencies are shown at each postoperative day (2–5). The *gray* and *red shaded areas* illustrate the maximal and minimal range of measured mean saccades latencies in seven patients. An average trend line for each HIT direction was calculated after plotting the best fit lines for each individual's data

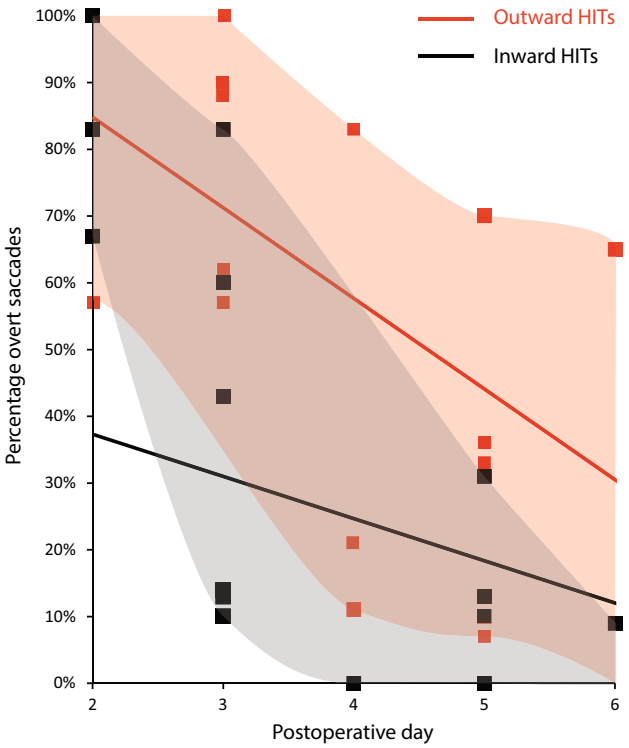

Fig. 4 The proportion of overt saccades is shown over time. An overt saccade was defined as a fast corrective eye movement after the head stopped. The *gray* and *red shaded areas* indicate the maximal and minimal range of measured percentages of overt saccades. An average trend line for each HIT direction was calculated after plotting best fit lines for each individual's data

impulse rotation also has an influence on the latency of the compensatory saccade. This may have important ramifications in the emergency department, where clinicians have the difficult responsibility of distinguishing vestibular tone asymmetries as generated from central versus peripheral causes [5, 14, 15]. The dependence of the compensatory saccade latency on the initial position of the head that we report ended by POD 30. Interestingly, a recently published paper found a similar result in chronic UVD: no difference in compensatory saccade latencies (overt vs. covert) for outward or inwardly applied HIT [6].

One explanation for why the direction of head rotation might have an impact on compensatory saccade latency is the predictability of the direction of head rotation. It is known that compared to passive head rotation, active head impulses (i.e., head rotation initiated by the patient rather than the examiner) result in higher VOR gains [16, 17]. Additionally, gaze stability during active head rotations is better than that during passive rotations [18] and latency of the VOR is shorter [19]. The mechanism presumed responsible for this is the principle of reafference—a motor efference copy, derived from planned and predictable actions, is compared to perceived sensory inputs [20]. It is therefore conceivable that head movements initiated from an eccentric (lateral) head position and moving toward the center might be predictable perhaps via cervical and oculomotor proprioceptive or visual cues, which may favor a saccade of reduced latency (covert) [21]. The origin of covert saccades, however, remains unknown. Covert saccades appeared immediately (at a smaller proportion) after vestibular deafferentation and might be triggered by multiple factors such as central compensation [13], anticipation of a planned head movement [22], neck proprioceptive cues and head velocity [3].

While our data suggest that outwardly applied HITs induce overt saccades more frequently, caution should be taken before deciding whether this approach should be applied in clinical practice. There are three disadvantages in performing this version of the HIT technique. First, outward HITs bear a higher risk of a 'head bounce', which is the most common artifact occurring with VOG-recorded HITs [23]. The head bounce effect is not an artifact, per se, but rather an error in technique. The examiner induces an overcorrection by pushing the head back to the start position following a head impulse during an outward head movement. Such additional and undesired head movements

toward the opposite direction at the end of the HIT might induce compensatory anti-saccades and, thus, influence HIT interpretation. Second, an asymmetric, gaze position-dependent nystagmus might bias HIT interpretation with outward impulses, but not with inward impulses. Third, outward impulses by an overzealous, inexperienced examiner risk neck over-rotation and, theoretically, injury to the vertebral artery.

Strengths and limitations of the study

Our study has limitations. Our sample size was small and may not generalize well to patients with subtotal vestibular loss (e.g., vestibular neuritis). Several patients had partial vestibular deficits before surgery, they received sedatives on POD 1 and the impact of prior vestibular adaptation (i.e., acute-on-chronic) is unknown. All patients developed spontaneous nystagmus after surgery, which might bias VOR gain measures; however, it is not known whether nystagmus fast phases interfere with corrective saccades and thus influence saccade latencies over time.

We did not record clinical HIT interpretations for each impulse or patient, and so cannot know whether the differences in compensatory saccade latency would have influenced a binary clinical judgment of 'normal' vs. 'abnormal' HIT VOR. We used a testing paradigm that likely maximized predictability of inward impulses, rather than deliberately trying to make them less predictable (e.g., by deliberately varying the cadence, velocity, sequence), so it is unknown whether we could have abolished this predictive effect of inward impulses. We have not addressed some technical issues related to the HIT technique. For example, VOR gain and saccade latency are functions of head velocity and acceleration [3], but we did not a priori standardize our applied impulses in this regard (e.g., 5 impulses per 50°/s head velocity increment with a symmetrical and identical velocity/acceleration profile). We did, however, apply a wide range of head impulse velocities and adjusted for head velocity in our statistical analysis. Mean head velocities were not statistically different between the two techniques across all PODs ($t = 0.8964$, $df = 623$, $p = 0.37$). We did not compare differences in oculomotor responses across different examiners (a single examiner gathered all data). Finally, we applied the vHIT from the front (being careful not to obscure the field of view), as would typically be done in clinical practice without recording equipment, but this has not systematically been compared with HIT applied from the back, which may have an effect.

Implications

In patients with acute loss of vestibular function, additional research on the impact of the head impulse technique on clinical interpretation is required (inward vs. outward, front vs. back, velocity/acceleration profile). Although our results suggest that inward impulses may risk erroneous interpretations through covert saccades, these findings are counterbalanced by other concerns as described above (e.g., asymmetric nystagmus with eye position-dependent intensity). For differentiating vestibular neuritis from stroke, a normal HIT is growing in acceptance as strong evidence for a cerebrovascular accident (CVA) [5]. While an error in this HIT interpretation (false-negative HIT) may result in unnecessary and costly stroke workups, such an error is not inherently dangerous. By contrast, outward impulses in this setting may risk a false-positive HIT interpretation (e.g., in a right vestibular neuritis, the ipsilesional outward impulse is interpreted with the eyes in a left orbital position, where the left-beating nystagmus is more intense) and could be dangerously misinterpreted as a peripheral vestibular cause instead of a central one (i.e., vestibular stroke).

Pending further research, we recommend that inward impulses be considered for use in an acute setting and the HIT technique be adjusted in an effort to minimize predictability (e.g., varying the cadence and velocity substantially) [22]. An alternative is to use portable VOG devices for patients where the clinical HIT is difficult to discern—these devices can measure vestibular hypofunction directly (reduced VOR gain) and help clinicians visualize covert saccades, in pursuit of a correct diagnosis [3, 4, 11, 24, 25].

In patients with chronic vestibular symptoms (where spontaneous nystagmus has disappeared and no longer a confound), clinicians might theoretically improve sensitivity of the HIT by performing less predictable, outwardly applied impulses, to reduce the chance of hidden (covert) saccades [22]. Our results, however, suggest that the discrepancy between inward and outwardly directed impulses vanishes 30 days after an acute deafferentation. So, it is unlikely that the outward technique will add much, though it still risks 'bounce' artifact as well as overzealous rotation of the neck.

Conclusions

Patients with acute unilateral vestibular loss likely use predictive cues of head direction to elicit early compensatory saccades to keep the image centered on the fovea. In patients with an acute loss of vestibular function, inwardly applied HITs risk a preponderance of covert saccades, although this effect largely disappears within 30 days. Potential advantages of inwardly applied HITs such as reducing bounce artifact, neutralizing the impact of nystagmus and decreasing the potential for cervical injury

must be balanced against the risk of a false-normal HIT interpretation. Further research on the clinical implications of HIT technique differences in acute vestibular patients is warranted.

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References

1. Halmagyi GM, Curthoys IS (1988) A clinical sign of canal paresis. ArchNeurol 45:737–739
2. Halmagyi GM, Aw ST, Cremer PD, Curthoys IS, Todd MJ (2001) Impulsive testing of individual semicircular canal function. AnnNYAcadSci 942:192–200
3. Weber KP, Aw ST, Todd MJ, McGarvie LA, Curthoys IS et al (2008) Head impulse test in unilateral vestibular loss: vestibulo-ocular reflex and catch-up saccades. Neurology 70:454–463
4. Newman-Toker DE, Saber Tehrani AS, Mantokoudis G, Pula JH, Guede CI et al (2013) Quantitative video-oculography to help diagnose stroke in acute vertigo and dizziness: toward an ECG for the eyes. Stroke 44:1158–1161
5. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE (2009) HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke 40:3504–3510
6. Lee SH, Newman-Toker D, Zee SD, Schubert MC (2014) Compensatory saccade differences between outward versus inward head impulses in chronic unilateral vestibular hypofunction. J Clin Neurosci 21(10):1744–1749. doi:10.1016/j.jocn.2014.01.024
7. Hirvonen TP, Aalto H (2009) Three-dimensional video-oculography in patients with vestibular neuritis. Acta Otolaryngol 129:1400–1403
8. Newman-Toker DE, Kattah JC, Alvernia JE, Wang DZ (2008) Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. Neurology. doi:10.1212/01.wnl.0000314685.01433.0d
9. EyeSeeCam (2013) VOG System. Munich, Germany
10. Agrawal Y, Schubert MC, Migliaccio AA, Zee DS, Schneider E et al (2014) Evaluation of quantitative head impulse testing using search coils versus video-oculography in older individuals. Otol Neurotol 35:283–288
11. Bartl K, Lehnen N, Kohlbecher S, Schneider E (2009) Head impulse testing using video-oculography. Ann N Y Acad Sci 1164:331–333
12. Mantokoudis G, Saber Tehrani AS, Kattah JC, Eibenberger K, Guede CI et al (2015) Quantifying the vestibulo-ocular reflex with video-oculography: nature and frequency of artifacts. Audiol Neurootol 20:39–50
13. Mantokoudis G, Schubert MC, Tehrani AS, Wong AL, Agrawal Y (2014) Early adaptation and compensation of clinical vestibular responses after unilateral vestibular deafferentation surgery. Otol Neurotol 35:148–154
14. Newman-Toker DE, Kerber KA, Hsieh YH, Pula JH, Omron R, et al (2013) HINTS outperforms ABCD2 to identify stroke in acute vestibular syndrome Atlanta, GA
15. Tarnutzer AA, Berkowitz AL, Robinson KA, Hsieh YH, Newman-Toker DE (2011) Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. CMAJ 183:E571–E592
16. Halmagyi GM, Black RA, Thurtell MJ, Curthoys IS (2003) The human horizontal vestibulo-ocular reflex in response to active and passive head impulses after unilateral vestibular deafferentation. Ann NYAcad Sci 1004:325–336
17. Della Santina CC, Cremer PD, Carey JP, Minor LB (2002) Comparison of head thrust test with head autorotation test reveals that the vestibulo-ocular reflex is enhanced during voluntary head movements. Arch Otolaryngol Head Neck Surg 128:1044–1054
18. Herdman SJ, Schubert MC, Tusa RJ (2001) Role of central preprogramming in dynamic visual acuity with vestibular loss. Arch Otolaryngol Head Neck Surg 127:1205–1210
19. Sprenger A, Zils E, Stritzke G, Kruger A, Rambold H et al (2006) Do predictive mechanisms improve the angular vestibulo-ocular reflex in vestibular neuritis? Audiol Neurootol 11:53–58
20. Goldberg JM, Minor LB (2012) Signal Processing in Vestibular Nuclei (VN) of Alert Animals during Natural Behaviors. The Vestibular System A Sixth Sense. Oxford University Press, Oxford
21. Schubert MC, Zee DS (2010) Saccade and vestibular ocular motor adaptation. Restor Neurol Neurosci 28:9–18
22. Tjernstrom F, Nystrom A, Magnusson M (2012) How to uncover the covert saccade during the head impulse test. Otol Neurotol 33:1583–1585
23. Mantokoudis G, Tehrani AS, Kattah JC, Eibenberger K, Guede CI, et al (2014) Quantifying the Vestibulo-ocular reflex with Video-Oculography: Nature and Frequency of Artifacts. Audiology & Neurolology: in press
24. MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS (2009) The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology 73:1134–1141
25. Weber KP, MacDougall HG, Halmagyi GM, Curthoys IS (2009) Impulsive testing of semicircular-canal function using video-oculography. Ann N YAcad Sci 1164:486–491