

Review Article

Measure of Central Vestibular Compensation: A Review

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The central vestibular compensation reduces vestibular symptoms and helps individuals improve balance affected by vestibular dysfunction. The video head impulse test provides an opportunity to study central vestibular compensation objectively. This study aims to methodically present existing information about the video head impulse test as a measure to evaluate central vestibular compensation in patients with unilateral vestibular dysfunction. Literature review comprised 12 research articles selected based on pre-set criteria and timeline (January 2010 to June 2020). The findings indicate that the appropriate video head impulse test measures to evaluate central vestibular compensation after the occurrence of temporary unilateral vestibular dysfunction are the improvement in vestibulo-ocular reflex gain. And, for permanent unilateral vestibular dysfunction are reduction in catch-up saccades percentage, velocity, amplitude, latency, and Perez and Rey score.

KEYWORDS: Head impulse test, saccades, vestibulo-ocular reflex

INTRODUCTION

The vestibular end organs such as otoliths and semicircular canals (SCs) of both ears lie in the same plane to facilitate working in tandem. They play a crucial role in body balance, postural control, visual stabilization, cognition, spatial orientation, and spatial memory. Disruption to these end organs causes vestibular dysfunction (VD) and affects the above-mentioned functions.¹ The resulting VD exhibits both static and dynamic symptoms. These symptoms are reduced by peripheral recovery and central vestibular compensation (CVC).² Unlike dynamic symptoms, it is easy to measure and monitor CVC of static symptoms (spontaneous nystagmus and head tilt), since the compensation of dynamic symptoms involves several neuronal networks.³

The CVC involves parallel and multiple processes such as (1) flexible changes in sensitivity and resting activity of vestibular nuclei and commissural network in response to signals from the vestibular end organs,⁴⁻⁷ (2) inhibitory response by the cerebellum,⁸ and (3) gliosis and neurogenesis in the lesioned vestibular nuclei.^{8,9} Central vestibular compensation processes include adaptation, restoration, and habituation, among which adaptation is prominent.¹⁰ It can be measured by a directional preponderance (DP) in the caloric test, sensory organization test scores of posturography, and vestibulo-ocular reflex (VOR) gain and re-fixation saccades during the VOR test.³

During head movements, the VOR pathway receives sensory input from otolith organs and SCs belonging to both sides; this aids in stabilizing the retinal image by moving the eye in the opposite direction at the same speed. The VOR gain is the ratio between the velocity of eye movement and head movement,¹¹ and its normative range lies between 0.8 and 1.2.¹² During VOR deficit (while head movements), catch-up saccades (CSs) are elicited to stabilize the gaze on the image.¹³ It appears that the measurement of the VOR and CS is subjective, thereby making their quantification difficult; however, with the video head impulse test (vHIT), objective measurement has become possible.¹⁴ In this review, we will attempt to review the role of vHIT as a measure of CVC in patients with UVD. The focus group for the review comprises patients with unilateral vestibular dysfunction (UVD).

METHODS

Literature Search

We will review articles published between 2010 and 2020 using the following keywords: “video head impulse test,” “vestibular compensation,” and “vestibular rehabilitation.” One of the criteria was that the keywords should be present in the title and/or abstract of the articles. Moreover, studies should be investigating the CVC in patients with UVD using the vHIT. Additionally, the articles should be in the English language. Brief comments, conference abstracts, animal studies, and non-English articles were excluded.

RESULTS

A PubMed search that focused on the vHIT as a measure of recovery after the occurrence of UVD yielded 294 articles. Subsequently, the application of the set criteria led to the selection of 12 articles. This review highlights each vHIT test parameter (e.g., VOR gain, CS parameters, and Perez and Rey [PR] score) separately for comprehensive understanding. Moreover, the PRISMA flow diagram and article details are mentioned in Figure 1 and Table 1, respectively.

VOR Gain and Compensation

The VOR gain correlates with the improvement or decline of the vestibular symptoms in various clinical conditions. Reversible or temporary vestibular disorders such as vestibular neuritis exhibit VOR gain improvement on the lesion side.¹⁵ In patients with unilateral vestibular neuritis a significant improvement on the lesion side was seen in VOR gain (horizontal canal) after 6 months of follow-up. The mean VOR gain in the acute stage was 0.47 ± 0.15 , and after a 6-month follow-up, it was 0.69 ± 0.23 . The authors attributed the improvement to peripheral (regeneration and sprouting) and central mechanisms (synaptic weighting, cellular recovery, and spontaneous recovery tonic firing rate).¹⁵

Unlike reversible vestibular conditions, the VOR gain measurement on the lesion side is unhelpful in irreversible conditions since the input is permanently shut on the lesion side.¹² The authors evaluated VOR gains in 17 patients who underwent vestibular rehabilitation training

(VRT) after unilateral vestibular deafferentation surgery. There was no significant improvement in VOR gain on the lesion side. Moreover, the behavioral scores on the risk of fall, confidence, and dynamic symptoms were significantly reduced in these patients.¹² Here, the absence of VOR gain improvement could be due to gaze stability exercises employed during VRT. Motor learning in VOR is specific to the training frequency¹⁶; hence, one should consider the non-lesion side's VOR gain as it also contributes to the central compensation.¹⁷ In patients with unilateral vestibular deafferentation, the VOR gain asymmetry, gait speed, and endurance-exhibited correlation (VOR gain asymmetry = contralesional gain – Ipsilesional gain/ contralesional gain).¹⁷ In case of a reduced VOR input, the cerebellum adjusts the gait through vestibular-mediated postural reflex, thereby reducing gaze-induced instability. An earlier study reported that VOR gains asymmetry reduction in the canal paresis recovery (CPR) group after 13 weeks.² Further, the findings agreed with sway during walking.² In contrast, ipsilesional VOR gain and gait did not. The authors postulated that the central reweighting of contralesional VOR input by the cerebellum preserved the ambulation after the occurrence of UVD.

The vestibulo-ocular reflex asymmetry index (AI%) is another outcome measure.¹⁸ This measure was studied in patients after the occurrence of acute unilateral vestibulopathy (AUV). The findings indicated that patients with adequate vestibular compensation (spontaneously recovered group) displayed higher VOR gain with low VOR AI% than patients who underwent VRT. This indicates that the lower AI% of VOR correlates with better compensation. However, the authors did not consider VOR gains of the vertical canals, citing the involvement of artifacts. The formula to calculate AI% is as follows: $1 - [(lower\ high-velocity\ VOR\ gain\ mean / higher\ high-velocity\ VOR\ gain\ mean) \times 100]$.

The vHIT provides an opportunity to assess each of the 6 SCs separately, thus making it a valuable tool to determine compensation in patients with a single SC damage. The VOR gain in patients with superior semicircular canal dehiscence (SSCD) was significantly less post plugging of SCC than pre-surgical status. Further, it remained low over time with improvement. The VOR gain of the other ipsilateral canals decreased immediately after plugging, but the VOR gain recovered in the horizontal canal and reached a normal baseline. However, the VOR gain of the posterior canal did not fully recover. Even the contralateral VOR gain of all SCs decreased slightly after plugging and recovered. This may be due to a lesser contribution from the operated side via commissural pathways, central adaptation by down-regulation, and suppressive effects of the anesthetic agents and medications postoperatively.¹⁹ This study shows that contralateral VOR gain modifications may be the correct measure for central compensation. An earlier study by the same authors reported similar findings.²⁰

Based on the above studies, the following could be elucidated. In temporary UVD, the lesion side VOR gains improvement reflecting central compensation. And, in the case of permanent UVD, it is the modification of VOR gain on the non-lesion side. Non-lesion side contribution could well be observed in the VOR gain asymmetry measure. Further, VOR gain is a good measure even to evaluate compensation in single canal dysfunction. It can help us understand the changes (compensation) occurring in other individual canals. Thus

MAIN POINTS

- Video head impulse test can be used as an objective measure to evaluate central vestibular compensation. Vestibulo-ocular reflex gain, catch-up saccades metrics, and PR scores are the video head impulse test measures.
- **VESTIBULO-OCULAR REFLEX GAIN:** Ipsilesional vestibulo-ocular reflex gain is a good measure to evaluate temporary unilateral vestibular dysfunction and for permanent unilateral vestibular dysfunction the contra-lesional vestibulo-ocular reflex gain information is useful.
- **CATCH-UP SACCADIC METRICS:** Lesion side reduction in catch-up saccade velocity, amplitude, latency, and a lesser prevalence of saccade indicates central vestibular compensation better.
- **PR SCORES:** Lesser the Perez and Rey score better the central vestibular compensation.
- **HEAD IMPULSE TESTING DEVICE-FUNCTIONAL TEST:** Reflects the real-life scenario and is a better measure to monitor vestibular rehabilitation training.

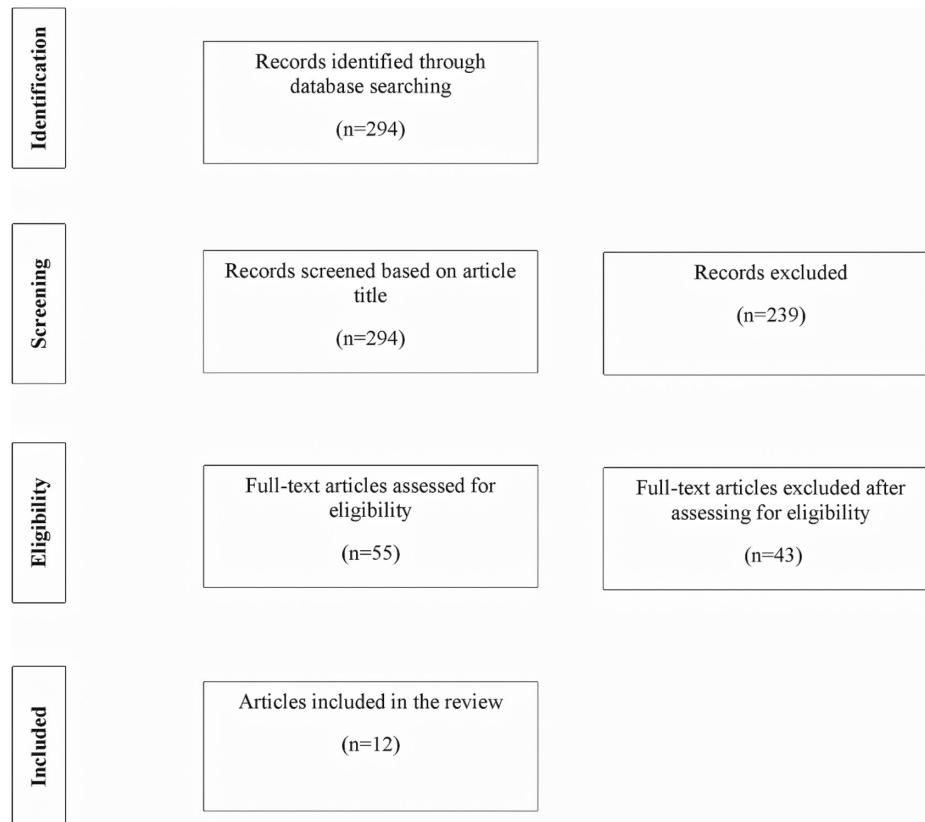


Figure 1. The number of studies identified by the search strategy, the number of studies excluded and included during primary screening and assessing for eligibility, and the final number of studies included.

VOR gain is a good measure to evaluate CVC and an appropriate VOR gain measure is to be employed.

Catch-up Saccades and Compensation

Catch-up saccades are high-velocity, corrective eye movements that compensate for the loss of VOR to fix the gaze on the target.²¹ Both covert and overt saccades play a crucial role in the process of compensation.²¹ The high-speed camera facility of the vHIT helps capture both overt and covert saccades.

The CS velocity, prevalence/percentage, amplitude, and latency of the saccade component are the various CS test measures. The CS velocity is the time taken by the eyes to fix the target when the image slips due to reduced VOR. Altered CS velocity even with no VOR gain improvement stabilizes the gaze.¹² The authors evaluated CS velocity after 5 weeks of VRT, in patients undergoing VS resection. The CS velocity was found to be significantly reduced for overt saccades during an ipsilesional passive head turn, which helped stabilize the gaze.¹² In a similar patient group, a gradual but significant reduction in covert and overt saccades velocities during the 6-month follow-up (covert saccade velocity during acute stage = 209.23 ± 48.17 , during follow-up = 186.14 ± 45.69 , P -value < .05; overt saccade velocity during acute stage = 203.00 ± 62.00 , during follow-up = 152.46 ± 29.70 , P -value < .01) was reported. Apart from the velocity, the authors also reported a gradual reduction in CS [covert saccades during acute stage (%) = 100, during follow-up = 87.23, P -value < .05; overt saccades during acute stage (%) = 100, during follow-up = 59.58, P -value < .01].¹⁵

The prevalence of CS and modification of CS amplitude (for both overt and covert saccades) compensate for the retinal slip and help stabilize the image faster. Higher prevalence and amplitude of overt saccades were observed in patients who required VRT (after acute vestibulopathy) than in the well-compensated patients' group.¹⁸ These were related to higher scores on DHI, which indicated a higher level of dysfunction. Furthermore, higher retinal slip in uncompensated patients is correlated with higher overt saccade amplitude. Similarly, the overt saccade's lower cumulative amplitude showed lower dynamic visual acuity (DVA) scores (better DVA performance) on the affected side.¹³ These correlations of CS characteristics with DVA performance reveal compensatory mechanisms in patients after the occurrence of UVD.¹³

The CS latency is the time taken for the eye to re-fixate on the target. The reduction in CS latency helps compensate for reduced VOR.¹⁹ Further, a decrease in CS latency can change the overt saccades to covert saccades.¹⁹ The saccade latency was significantly less in patients with a plugged SSC (from 186.2 ± 19 ms to 141.0 ± 17.5 ms; $P = .032$), which led to the conversion of the overt saccades into covert saccades.¹⁹ In another study, the visual acuity (VA) and analysis of saccade latency were used to measure the outcome after VRT. All patients showed a reduction in the difference between the head impulse halt and the last saccade to achieve a VA of 75%.²² This significant reduction in time to re-fixate on the target shows the compensation process.

Thus, measurement of the ipsilateral CS parameters such as velocity, prevalence/percentage, amplitude, and latency of the covert and

Table 1. Summary of Studies Included in the Review

Study	No. of Patients	Population		Tests	Parameters Assessed	Study Design
		Experimental Group	Control Group			
² 2017	n = 30	UVN	n = 30	vHIT, ROT	VOR gain	Retrospective
²⁷ 2017	n = 36	UVS	–	vHIT	VOR gain, PR score	Prospective
¹⁹ 2018	n = 60	AUV Group 1, n = 30 Group 2, n = 30	–	Caloric test, DHI	Canal Paresis, DHI score, VOR gain, AI, CS latency and amplitude, PR score	Retrospective
²⁸ 2018	n = 20	UVL (VS-8, VN-19, labyrinthitis-1, labyrinthectomy-2)	–	vHIT	VOR gain, CS latency, PR score	Prospective
¹⁵ 2016	n = 5	Patient with plugged SSC	–	vHIT	VOR gain, CS	Prospective longitudinal
¹² 2020	n = 43 (19 completed)	UVD due to VS surgery	n = 38 for DVA n = 28 vHIT	DVA, vHIT, VEMP, DHI, ABC, DGI, TUG, GS, and GE	VOR gain, CS-latency, frequency and velocity, PR score, VEMP Asymmetry ratio, DHI score, ABC score, Visual acuity, Fall risks, Gait speed	Prospective
¹⁸ 2020	n = 37	UVD from VS resection		vHIT	VOR gain, CS-lat, freq. and vel., GS & GE	Prospective
²⁹ 2012				vHIT, VEMP	VOR gain, CS	Review
²⁰ 2014	n = 5	UVD from VS resection		Video oculography, vHIT	SPV of SN, Vertical skewness, VOR gain, Saccade velocity	Prospective longitudinal
³⁰ 2018	n = 9	UVL (VS-8, Cog. UVL-1)		vHIT, HITD-FT	VOR gain, CS metrics, Visual Acuity	Prospective
¹⁶ 2019	n = 47	UVN		vHIT, DHI	VOR gain, CS occurrence (%), vel. & lat	Prospective
³¹ 2016	n = 24	Complete UVL	n = 113	DVA, vHIT	DVA, VOR gain, % of CS and amp	Prospective

vHIT, video head impulse test; ROT, rotating chair tests; UVN, unilateral vestibular neuritis; UVS, unilateral vestibular schwannoma; DHI, Dizziness Handicap Inventory; AUV, acute unilateral vestibulopathy; AI, asymmetry index; CS, catch-up saccade; PR, Perez and Rey; UVL, unilateral vestibular loss; VS, Vestibular Schwannoma; VN, Vestibular Neuritis; SSC, Superior semicircular canal; DVA, dynamic visual acuity; VEMP, vestibular-evoked myogenic potential test; ABC, Activities-Specific Balance Confidence Scale; DGI, Dynamic Gait Index; TUG, Timed Up and Go; SPV, slow phase velocity; SN, spontaneous nystagmus; HITD-FT, Head Impulse Testing Device-Functional Test; GS, Gait Speech; GE, endurance; Cog., congenital; Vel., velocity, lat., latency, Freq., Frequency, amp., amplitude; UVD, unilateral vestibular deafferentation.

overt saccades helps develop a quantitative measure for recovery after the occurrence of UVD. Further, ipsilateral–contralateral comparison of CS metrics and VOR gains facilitates the understanding of the involvement of the compensatory mechanism's multisensory internal model. Interestingly, the contralesional CS and VOR gains are unrelated, which indicates a specific stimulus for enrolling CS in the contralesional head rotation.¹⁷ The measured velocity and frequency of the CS on the ipsilesional side were more significant than on the contralesional side in patients with UVD after vestibular schwannoma resection. However, both ipsi- and contralesional CS latencies were the same. The authors reasoned that (1) both ipsi- and contralesional CS are initiated by the commonly shared central nervous system, (2) the connection of both the side vestibular nucleus (VN) through commissural fibers contributes to the spontaneous neural firing restoration in the ipsilesional VN, and (3) there is a change in contralesional VOR created by ipsilesional rotation stimulus. Based on these reasons, the authors speculated that for the CS initiation during UVD, the commissural connections on the brainstem and the intact side neurons might start the generation of the shared signal for both ipsi- and contralateral sides. This transmitted signal may contribute to multisensory recruitment

(internal model for contralesional CS). Further, the cervico-ocular reflex with proprioceptive information and visual system is relevant in generating CS.¹⁷

Various CS metrics like CS velocity, prevalence/percentage, amplitude, and saccade latency are useful in studying the CVC. A reduction in lesion-side CS velocity, amplitude, latency, and a lesser prevalence of saccade could indicate better CVC. Further, CS metrics can be used to derive some interesting predictions. As in case of, saccade amplitude, as the value gets lesser, the saccade may disappear (better CVC). In CS latency, as it reduces, a change of overt to covert saccade could be expected.

PR Score and Compensation

The CS can be analyzed for synchrony sequential re-fixation by measuring saccade gathering (low PR scores) or scattering (high PR scores). These gathered and scattered responses correlate with good (well-compensated) and poor (uncompensated) compensation, respectively.²³ Here, PR scores can be independent of VOR gain.²³ In patients with UVL who demonstrated improved VA, the PR scores were reduced after VRT; however, VOR gain did not show any

improvement.²² The patients with vestibular schwannoma were subgrouped based on canal paresis (CP), VOR gain, and the presence of saccades. In comparison between preoperative and postoperative PR scores within each subgroup, the preoperative CP group had higher PR scores than the no canal paresis (N-CP) group, whereas the condition was opposite postoperatively. Therefore, the authors implicated faster central compensation in the CP group. A similar phenomenon was observed in the other subgroups too. Preoperatively, the more severe groups (with low VOR gain and absent saccades) had higher PR scores than the less severe groups (with high VOR gain and present saccades), while the reverse held true postoperatively.²³ Hence, the lesser the PR score better the CVC. Interestingly, the PR score can be a standalone measure independent of VOR gain to reflect upon CVC.

Head Impulse Testing Device-functional Test (HITD-FT)

Real-life events involve both passive and active head movements, majorly while performing a visual task, for which cortical involvement is vital. Evaluation of vestibular function with regard to varying angular head acceleration (active and passive) while performing a visual task is challenging. However, it provides close-to-real-life information²⁴ and is a better way to assess vestibular compensation in patients with vestibular hypofunction.²⁵ While in the vHIT, only passive head movements are recorded, the head impulse testing device-functional test (HITD-FT) involves both the head movements and, therefore, is an ideal tool to understand the cortical involvement in CS. The HITD-FT showed better VA for active head impulses than for passive ones and assessed VOR function without directly measuring the eye movements.^{25,26} The HITD-FT results in patients with total vestibular loss revealed better VA on the ipsilesional side for active head impulse than on the contralesional side. Moreover, the latencies of generated saccades were shorter, and the HITD-FT scores for active head impulses on the ipsilesional side were higher than those for passive head impulses. Further, the dynamic VA was better for active head impulses than those for the passive head movements, as reflected by the shorter saccade latencies. These better scores could be attributed to cortical involvement (a feed-forward mechanism). The other reason may be the inhibition of the saccadic system's inhibitory neurons, which might have made the system "trigger-happy" for releasing any saccades.²⁵ Thus, the HITD-FT reflects the real-life scenario better (active and passive head movements) and is a better measure to monitor VRT. Unique to HITD-FT is that it evaluates VOR using VA and not the eye movements.

CONCLUSION

This study employs objective test measures to understand the underlying physiological mechanisms of CVC. The vHIT has considerable potential in measuring outcomes (compensation) after the occurrence of UVD. VOR gain, VOR asymmetry, AI, and CS are the various vHIT test measures. Whether the UVD is temporary or permanent is an important factor for test outcomes. Further, improvement in VOR gain is a good outcome measure in patients with temporary UVD; reduction in VOR asymmetry, lower AI, and reduced CS parameters like CS percentage, velocity, amplitude, latency, and lower PR scores are good outcome measures for permanent UVD. Additionally, the HITD-FT, a modified vHIT procedure, gives information on cortical inputs for CVC. The current review may motivate clinicians and

researchers to use vHIT in studying CVC. It may even encourage researchers to take up a longitudinal study involving vHIT on different vestibular conditions.

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