



Brazilian Journal of OTORHINOLARYNGOLOGY

www.bjorl.org



ORIGINAL ARTICLE

What does the video head impulse test tell us about post-caloric vestibular recruitment?



R. Mezzalira ^{a,*}, R.S.M. Bittar ^b

^a Universidade Estadual de Campinas (UNICAMP), Departamento de Otorrinolaringologia, Campinas, SP, Brazil

^b Universidade de São Paulo, Faculdade de Medicina, Departamento de Otorrinolaringologia, São Paulo, SP, Brazil

Received 21 June 2022; accepted 22 October 2022

Available online 11 November 2022

HIGHLIGHTS

- Post caloric recruitment index is the ratio between cold and warm stimulation.
- Post caloric recruitment index is useful to identify the affected ear separately.
- Video head impulse test did not prove to be a good predictor of post caloric recruitment.

KEYWORDS

Vestibulo-ocular reflex;
Vestibular recruitment;
Video head impulse test;
Caloric test;
Vestibular compensation

Abstract

Objective: The vestibular recruitment observed in caloric testing is a new tool in the study of the vestibulo-ocular reflex. This study aimed to determine the sensitivity and specificity of the video head impulse test to detect post-caloric vestibular recruitment.

Method: In this cross-sectional study, all participants underwent the standard otoneurological assessment of the service, caloric test, and video head impulse test. A non-linear mixed model was used to test for associations.

Results: The study group consisted of 250 (89 male and 161 female) patients, with a mean age of 54.84 years. The control group comprised 35 participants, 18 men and 17 women, with a mean age of 40.42 years. Sex and age had no effect on group responses. There was no difference between the study and control groups regarding the interaction between recruitment and gain ($p = 0.7487$); recruitment and overt ($p = 0.7002$) and covert saccades ($p = 1.0000$); and recruitment and anti-compensatory saccades in the contralateral ear ($p = 0.3050$). The video head impulse test had a sensitivity of 51% and a specificity of 50% as a predictor of post-caloric recruitment.

Conclusion: The video head impulse test results showed no relevance in predicting post-caloric vestibular recruitment.

© 2022 Associação Brasileira de Otorrinolaringologia e Cirurgia Cérvico-Facial. Published by Elsevier Editora Ltda. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

* Corresponding author.

E-mail: raquelmezzalira@uol.com.br (R. Mezzalira).

Introduction

The study of the Vestibulo-Ocular Reflex (VOR) is considered a fundamental tool in the identification of diseases of the vestibular system. The Caloric Test (CT) is the most used method for assessing VOR. In case of partial impairment of afferent impulses from the VIII nerve, warm (excitatory) stimuli cannot excite the motoneurons.¹ The deafferented nucleus has a reduced response due to reduced peripheral stimuli; furthermore, it is inhibited by the opposite (healthy) nucleus through the inter-commissural pathway. However, cold (inhibitory) stimuli inhibit the already compromised peripheral–input and the basal activity of the nucleus on the injured side. Based on this physiological knowledge, an increased cold/warm ratio for the same ear is suggestive of tone asymmetry between the vestibular nuclei. This asymmetry is called Post-Caloric Recruitment (PCR).²⁻⁵ PCR is an objective phenomenon that reflects peripheral input impairment and shows a state of equilibrium between the vestibular nuclei and the central compensation phase.^{6,7}

The video Head Impulse Test (vHIT) is useful in differentiating central and peripheral disease in acute vertigo and as a complement to the CT in the assessment of chronic dizziness. vHIT analysis considers two parameters: VOR gain and the presence of saccades. There are two types of saccades in uncompensated acute peripheral lesions. Overt saccades occur after the end of head movement and show VOR incompetence in maintaining the target on the retina. Anti-compensatory Quick Eye Movements (AQEM) or anti-compensatory saccades occur toward head movement, in the contralateral ear, and indicate vestibular tone asymmetry of peripheral origin. Its function is to reposition the eye to maintain ocular fixation.⁸

As vestibular recruitment indicates a phase of vestibular system compensation after a peripheral lesion, it would be possible to find overt and AQEM in patients who present with PCR. The vHIT can provide information regarding the current state of the disease and of the compensation, and provides effective therapeutic strategies; furthermore, its advantages include being quick and more comfortable for patients.

The objectives of this study are to evaluate vHIT results in patients with vestibular recruitment in CT, to compare these patients with a control group, and to calculate the sensitivity and specificity of vHIT in the diagnosis of PCR.

Methods

This cross-sectional study was approved by the Ethics Committee for the Analysis of Research Projects, and in accordance with the 1975 Declaration of Helsinki.

Sample

The Study Group (SG) comprised 250 participants with vestibular dizziness of variable duration and progression. The Control Group (CG) comprised 35 healthy volunteers without vestibular complaints who underwent the same assessment. The inclusion criteria for both groups were age over 18 years, cognitive ability to understand and perform the tests, and signing the informed consent form. The

exclusion criteria were chronic otitis media; ear surgery; somatoform or psychiatric diseases; and eye, cervical, rheumatic, or orthopaedic diseases not allowing the performance of the tests and neurologic diseases. The duration of the study was 3 years from November 2018 to December 2021.

Intervention

All participants underwent otoneurological assessment, caloric testing, and vHIT in the same week. Otoneurological assessment includes general anamnesis, otorhinolaryngological and cranial nerve examination, static and dynamic balance assessment, and coordination tests.

CT was performed using video-oculography (ICS Chartr 200, Otometrics®) and stimulation with water at 44°C and 30°C for 40s. Prior to each stimulation, the water temperature was checked with three thermometers and the first jet of water was discarded before starting the irrigation of the external auditory canal. Patients who presented no or doubtful responses were excluded from the study. The Post-Caloric Recruitment Index (PCRI) was calculated for each ear separately using the formula:⁵

$$\text{PCRI} = (\text{AVSP C} - \text{AVSP W}) / (\text{AVSP C} + \text{AVSP W}) \times 100;$$

PCRI, Post-Caloric Recruitment Index; AVSP C, Angular Velocity of the Slow Phase after Cold irrigation; AVSP W, Angular Velocity of the Slow Phase after Warm irrigation.

Ears that had PCRI > 17% were considered for recruitment, a value considered normal for our population.⁵

vHIT was performed using the Otometrics ICS impulse system®. Manual and unpredictable head impulses were administered at an angle of 15°–20° with a mean velocity of 200°/second and a mean acceleration of 3000°/s² in horizontal planes to assess the lateral semi-circular canals. The mean VOR gain calculated using the software was considered for quantitative analysis. Gain values between 0.8 and 1.2 were considered normal.⁹ The presence or absence of saccades and their location were considered for qualitative analysis. Saccades that occurred after the end of the head turn were called overt saccades, and those occurring before the end of head movement were called covert saccades. Eye movements toward the head movement that occurred after the head movement were called Anti-compensatory Quick Eye Movements (AQEM).

Based on the PCRI calculated in the CT, the subjects' ears were separated into two groups: Recruiting Ears (RE) and Non-Recruiting Ears (NRE). Gain changes and presence of saccades were studied in both groups and compared with the CG. The exams were analysed by the main investigator.

Statistical analysis

Recruitment associations with each of the vHIT responses were tested using logistic regression model adjustments. The factors considered for the model were population, recruitment, saccade, and gain. The effect of age and sex in the comparisons studied was tested by including age and sex in the models as covariates. A significance level of 5% was considered for all hypothesis tests ($p < 0.05$). The JMP 16 software was used in this study.

Table 1 Analysis of covariance to analyse similarity between groups.

Variable	Age (<i>p</i>)	Sex (<i>p</i>)
Gain	0.3239	0.9593
Overt saccade	0.4729	0.7817
Covert saccade	0.3166	0.9592
AQEM	0.2796	0.9785

AQEM, Anti-compensatory Quick Eye Movements.

Table 2 Gain and saccade frequency distribution in recruiting and non-recruiting ears in the study group and in the control group.

Variables	RE		NRE		Total	
	SG	CG	SG	CG	SG	CG
High gain	26	3	65	13	91	16
Low gain	7	0	29	0	36	0
Normal gain	94	11	279	43	373	54
Total	127	14	373	56	500	70
Overt +	21	0	77	2	98	2
Overt -	106	14	296	54	402	68
Total	127	14	373	56	500	70
Covert +	11	3	42	5	53	8
Covert -	116	11	331	51	447	62
Total	127	14	373	56	500	70
AQEM +	26	0	68	1	94	1
AQEM -	101	14	305	55	406	69
Total	127	14	373	56	500	70

RE, Recruiting Ears; NRE, Non-Recruiting Ears; SG, Study Group; CG, Control Group; AQEM, Anti-compensatory Quick Eye Movements.

Results

The SG consisted of 250 patients (89 men and 161 women; mean age, 54.84 years) who met all the inclusion criteria. The CG consisted of 35 participants, 18 men and 17 women, with a mean age of 40.42 years. An analysis of covariance verified sex and age similarities and showed no relationship with the vestibular tests of any of the groups (**Table 1**).

The **Table 2** show the gain and saccade frequency distribution in recruiting and non-recruiting ears in the study group and in the control group.

Relationship between PCR and the presence of gain or saccades in the vHIT in the SG

A logistic regression model was used to test the association between the results obtained in CT and vHIT in the SG. There was no interaction between recruitment and gain in subjects with vestibular symptoms (*p* = 0.3092). There was also no difference between the recruiting and non-recruiting groups regarding the presence of overt saccades (*p* = 0.7002), covert saccades (*p* = 1.000), and AQEM (*p* = 0.8600). **Table 3** shows the p-values for the analysis of the interaction between recruitment in the CT and gain and saccades in the vHIT in the SG.

Table 3 Relationship between PCR and the presence of gain or saccades in the vHIT in the SG.

Association of variables	<i>p</i>
Recruitment and gain	0.3092
Recruitment and overt saccades	0.7002
Recruitment and covert saccades	1.0000
Recruitment and AQEM	0.8600

AQEM, Anti-compensatory Quick Eye Movements.

Table 4 P-values from the comparison between the SG and CG regarding the variables of interest.

Association of variables	<i>p</i>
Recruitment and gain	0.7487
Recruitment and overt saccades	0.7002
Recruitment and covert saccades	1.0000
Recruitment and AQEM	0.3050

AQEM, Anti-compensatory Quick Eye Movements.

Comparison between the SG and the CG

Adjusted logistic regression models were used to compare the SG and CG regarding CT and vHIT results.

The analysis of gain in RE and NRE of both groups showed no low gain in the CG. The SG presented low gain in 7 RE (1%) and in 29 NRE (6%). High gain was present in 26 RE (5%) in the SG and in 3 RE (4%) in the CG, and in 65 NRE (13%) in the SG and in 13 NRE (1%) in the CG. No association was observed between PCR and vHIT gain (*p* = 0.7487).

The presence of overt saccades in RE and NRE was analysed in both groups. There were no overt saccades in RE in the CG, but they were present in 21 RE (4%) in the SG. As for NRE, overt saccades were present in 2 (3%) in the CG and in 77 (15%) in the SG. No association was observed between PCR and vHIT overt saccades (*p* = 0.7002).

The SG presented covert saccades in 11 RE (2%) and the CG presented covert saccades in 3 RE (4%). The CG presented covert saccades in 5 NRE (7%) and SG in 42 NRE (8%). No association was observed between PCR and vHIT covert saccades (*p* = 1.0000).

The analysis of the presence of AQEM in the contralateral RE and NRE in both groups showed no anti-compensatory saccades in RE in the CG, while in the SG it occurred in 26 (5%) contralateral RE. As for NRE, they were 68 (14%) in the SG and in one (1%) in the CG. No association was observed between PCR and vHIT AQEM (*p* = 0.3050).

The comparison between groups showed no differences regarding the interaction between recruitment and gain and between recruitment and overt, covert, and AQEM. **Table 4** shows the p-values from the comparison between the SG and CG regarding the variables of interest in CT and vHIT results.

The assessment of vHIT as a predictor of PCR showed a sensitivity of 51% and a specificity of 50% (**Table 5**). There was no relationship between vHIT and PCR.

Table 5 Calculation of sensitivity and specificity of vHIT in predicting post-caloric recruitment.

	Non-Recruiting CT	Recruiting CT	Total
Normal vHIT	69 ($Sp = 69/137 = 50\%$)	55	124
Altered vHIT	68	58 ($Se = 58/113 = 51\%$)	126
Total	137	113	250

Sp, Specificity; Se, Sensitivity.

Discussion

The term vestibular recruitment was used to characterise a lesion that originates in the peripheral vestibular organ.⁷ CT shows the involvement of the peripheral organ by verifying the hyperexcitability of the vestibular nucleus contralateral to the lesion. Increased vestibular tone can be identified by a more intense caloric response to inhibitory (cold) stimulation compared to direct (warm) stimulation of the lateral canal. Thus, CT can identify the compromised ear responsible for recruiting the vestibular pathway. In our clinical practice, we observed that "recruiting" patients usually present good progression after adequate treatment of the etiological factors causing the symptoms. Thus, in addition to aiding in making the right diagnosis, the PCRI improves the clinical management and prognosis of the lesion without additional costs to routine tests. Therefore, we decided to evaluate the usefulness of vHIT as a possible predictor of PCR due to its easy and increasingly frequent use.

Our sample presented low gain in 36 ears (7%) in the SG, which was not observed in the CG. This result was already predicted since the eye movement generated by the impulse does not reach the range of head movement in vestibular hypofunction, reducing gain. High gain was present in 91 (18%) ears in the SG and in 16 (23%) ears in the CG, with no statistical difference between the groups. Several factors can affect neural impulse transmission, such as metabolic and hormonal factors and anxiety. These situations affect VOR suppression by the cerebellum, especially by the flocculus.^{10,11} We believe that these situations may justify the presence of hyperactive VOR even in individuals without dizziness. In our clinical practice, we frequently observe high gains in patients with migraine, especially when anxiety is present. Other authors have described increased gain in endolymphatic hydrops,¹² although we have not encountered such. As already mentioned, recruitment shows a phase of asymmetry between vestibular nucleus discharges resulting from the lack of afferents from the peripheral vestibular organ. In this phase, the role of the cerebellum in vestibular nucleus hyperactivity has not been completely established. In this case, we could find some changes in the gain in the recruiting subjects, a situation that has been attributed to the lack of cerebellar action. However, we found no gain and PCR differences between the SG and the CG.

Available literature provides controversial details regarding the presence of saccades as a sign of VOR hypofunction, whether overt or covert. Our CG had overt saccades in 3% of ears and covert saccades in 11% of ears. These results corroborate the study by Salmito et al.,¹³ who also reported corrective saccades in asymptomatic individuals. Patients

who successfully compensate for a vestibular insult may experience saccadic correction before the end of head movement. Therefore, covert saccades may be present in normal subjects who previously had a vestibular dysfunction, even if asymptomatic.¹⁴ Other authors suggest that the VOR is a hypometric system and saccades are part of a normal correction of the reflex; thus, the presence of corrective saccades shows no VOR dysfunction.^{15,16} Considering these factors, the saccades observed in the CG may be due to normal VOR physiology.

Since vestibular recruitment depicts a phase of decompensation, our first hypothesis predicted the presence of overt saccades in vHIT in the presence of PCR. This happens because overt saccades appear in the acute phase of peripheral dysfunction; furthermore, as vestibular compensation occurs, they approach head movement and end up becoming covert. However, overt saccades were found in 98 (20%) ears in the SG, of which only 21 (4%) were RE. These observations invalidate the initial hypothesis and rule out overt saccades as evidence of PCR.

An interesting indicator in this study would be the AQEM, which indicate vestibular tone asymmetry of peripheral origin as a result of a lesion in the contralateral ear.⁸ Our previous hypothesis was that these saccades could function as a predictor of recruitment in the contralateral ear. They were present in 94 (19%) ears in the SG, but only 26 (5%) of them had a contralateral RE. On the other hand, they were present in 68 (14%) contralateral NRE. We found no interaction between recruitment and anti-compensatory saccades in none of the groups.

Disagreements between CT and vHIT have been discussed by several authors.^{7,26} The explanation may be based on the VOR anatomy itself. The angular VOR receptor is the crista ampullaris, composed of type I and type II hair cells. Type I hair cells populate the central part of the crista ampullaris and encode head movements at high frequency and high acceleration. Irregular afferent fibres carry the output from these cells toward second-order excitatory type I neurons located in the vestibular nucleus. Type II hair cells populate the periphery of the crista ampullaris and encode low frequency and low acceleration movements. They originate regular afferent fibres that synapse in the vestibular nucleus with second-order inhibitory type II neurons.^{18,19,21} Regarding vestibular recruitment, it is important to highlight the difference between vHIT and PC afferent pathways. In the first, ampullaris signals are transmitted directly to effector eye muscles, while the second is responsible for commissural inhibition, which inhibits the contralateral nucleus. This pathway depends on GABAergic type II neurons, the only inhibitory cells in the VOR circuit. PCR depends on these GABAergic neurons, which are responsible for com-

missural inhibition.⁶ Therefore, recruitment is established in CT because it assesses the commissural pathway, while vHIT responses depend on the direct VOR activation pathway. Thus, further explains the absence of relevant vHIT results in predicting PCR.

In this study, PCR-related vHIT sensitivity and specificity remained around 50%. Clinically, sensitivity 51% means that 51% of recruiting subjects may have changed vHIT. On the other hand, 49% of them may have had normal vHIT. A specificity of 50% indicates that only half of the vHIT may be changed in cases without PCR. Therefore, the test has negligible sensitivity or specificity in the diagnosis of PCR.

Conclusion

In conclusion, vHIT did not prove to be a good predictor of PCR. Our results are in agreement with those of previous studies that concluded that CT and vHIT assess semi-circular canals in different segments and different VOR pathways.¹⁷⁻²⁶

Acknowledgements

We thank José Ferreira de Carvalho, PhD, for the careful statistical analysis and Suelen Cesaroni, MS, for support in carrying out the exams.

Funding

This study did not receive external funding. The authors have no financial relationship to disclose.

Conflicts of interest

The authors declare no conflicts of interest.

Informed consent was obtained from each participant included in the study.

References

1. Azzi A, Giordano R, Spelta O. Does a vestibular recruitment exist? *Acta Otolaryngol*. 1953;43:352-68.
2. Parker W, Hamid M. Vestibular responses to different caloric stimulus intensities. *Am J Otol*. 1985;6:378-86.
3. Matsuhira T, Yamashita K, Yasuda M, Ohkubo J. Detection of the unilateral vestibular recruitment phenomenon using the rotation test. *Acta Otolaryngol Suppl*. 1991;481:486-9.
4. Brookler Kh. ENG in a patient with Ménière's syndrome and evidence of vestibular recruitment. *Ear Nose Throat J*. 2003;82:846-8.
5. Bittar RSM, Mezzalira R, Ramos ACM, Risso GH, Real DM, Grasel SS. Vestibular recruitment: New application for an old concept. *Braz J Otorhinolaryngol*. 2021, <http://dx.doi.org/10.1016/j.bjorl.2021.04.006>. Online ahead of print.
6. Tsemakhov SG. Vestibular recruitment. *Vestn Otorinolaringol*. 1979;4:75-83.
7. Van Egmond AA, Groen JJ, Hulk J, Jongkees LBW. The turning test with small regulable stimuli. Deviations in the cupulogram. Preliminary note on the pathology of cupulometry. *J Laryngol Otol*. 1949;63:306-10.
8. Luis L, Lehnend N, Muñoz E, Carvalho M, Schneider E, Valls-Solé J, et al. Anticompenstatory quick eye movements after head impulses: A peripheral vestibular sign in spontaneous nystagmus. *J Vestib Res*. 2015;25:267-71.
9. Hougaard DD, Abrahamsen ER. Functional testing of all six semicircular canals with video head impulse test systems. *J Vis Exp*. 2019;18(146):1-14, <http://dx.doi.org/10.3791/59012>.
10. Choi JY, Kim HJ, Kim JS. Recent advances in head impulse test findings in central vestibular disorders. *Neurology*. 2018;90:602-12.
11. Chen L, Halmagyi GM. Video head impulse testing: from bench to bedside. *Semin Neurol*. 2020;40:5-17.
12. Rey-Martinez J, Altuna X, Cheng K, Burgess AN, Curthoys IS. Computing endolymph hydrodynamics during head impulse test on normal and hydropic vestibular labyrinth models. *Front Neurol*. 2020;11:289.
13. Salmito MC, Ganança FF. Video head impulse test in vestibular migraine. *Braz J Otorhinolaryngol*. 2021;87:671-7.
14. MacDougall HG, Curthoys IS. Plasticity during vestibular compensation: The role of saccades. *Front Neurol*. 2012;3:21.
15. Anson ER, Bigelow RT, Carey JP, Xue QL, Studenski S, Schubert MC, et al. Aging increases compensatory saccade amplitude in the video head impulse test. *Front Neurol*. 2016;7:113.
16. Korsager LE, Faber CE, Schmidt JH, Wanscher JH. Refixation saccades with normal gain values: A diagnostic problem in the video head impulse test: A case report. *Front Neurol*. 2017;8:81.
17. Bell SL, Barker F, Heselton H, Mackenzie E, Dewhurst D, Sanderson A. A study of the relationship between the video head impulse test and air calorics. *Eur Arch Otorhinolaryngol*. 2015;272:1287-94.
18. McCaslin DL, Rivas A, Jacobson GP, Bennett ML. The dissociation of Video Head Impulse Test (vHIT) and bithermal caloric test results provide topological localization of vestibular system impairment in patients with "Definite" Ménière's Disease. *Am J Audiol*. 2015;24:1-10.
19. Alhabid SF, Saliba I. Video head impulse test: A review of the literature. *Eur Arch Otorhinolaryngol*. 2017;274:1215-22.
20. Vallim MGB, Gabriel GP, Mezzalira R, Stoler G, Chone CT. Does the video head impulse test replace caloric testing in the assessment of patients with chronic dizziness? A systematic review and meta-analysis. *Braz J Otorhinolaryngol*. 2021;87:733-41.
21. Limviriyakul S, Luangsawang C, Suwanit K, Prakairungthong S, Thongyai K, Atipas S. Video head impulse test and caloric test in definite Ménière's disease. *Eur Arch Otorhinolaryngol*. 2020;277:679-86.
22. Mahringer A, Rambold HA. Caloric test and video-head-impulse: a study of vertigo/dizziness patients in a community hospital. *Eur Arch Otorhinolaryngol*. 2014;271:463-72.
23. Zellhuber S, Mahringer A, Rambold HA. Relation of video-head-impulse test and caloric irrigation: A study on the recovery in unilateral vestibular neuritis. *Eur Arch Otorhinolaryngol*. 2014;271:2375-83.
24. Halmagyi GM, Aw ST, Cremer PD, Curthoys IS, Todd MJ. Impulsive testing of individual semicircular canal function. *Ann N Y Acad Sci*. 2001;942:192-200.
25. McCaslin DL, Jacobson GP, Bennett ML, Gruenwald JM, Green AP. Predictive properties of the video head impulse test: measures of caloric symmetry and self-report dizziness handicap. *Ear Hear*. 2014;35:e185-91.
26. Rambold HA. Economic management of vertigo/dizziness disease in a county hospital: video-head-impulse test vs. caloric irrigation. *Eur Arch Otorhinolaryngol*. 2015;272:2621-8.