Original article

A comparison of video head impulse test (vHIT) between patients with peripheral vestibular loss and healthy groups

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Objectives To compare the results of video head impulse testing (vHIT) of patients with peripheral vestibular loss (PVL) and healthy groups and to determine the sensitivity and specificity of vHIT for PVL patients.

Methods A total of 76 subjects participated in the study, 38 in the PVL group and 38 in the healthy group. VHIT was performed on all subjects using ICS impulses in the horizontal, left-anterior, right-posterior, and the right-anterior left-posterior planes, respectively between November 2018 and March 2019.

Results The mean gain in vertical vestibule-ocular reflex (VOR) was significant in both groups with the exception of the right-anterior plane. There were only nonsignificant differences in the mean gain for horizontal VOR and all the means for VOR gain asymmetry (GA). Using abnormal VOR gain and/or abnormal VOR GA, the sensitivity and specificity of vHIT for PVL patients were 55.3%, 84.2%, respectively.

Conclusions VHIT is able to identify significant dysfunction of the vertical semicircular canals (SCCs) in PVL patients. VOR gain and GA can be used to interpret of PVL patients with a high degree of specificity, especially within cases where SCCs dysfunction has caused a reduction in VOR gain. Although vHIT showed relatively low sensitivity, it is adequate for evaluation of PVL patients and should be considered complementary to other vestibular tests. **Chiang Mai Medical Journal 2021;60(4):427-35. doi: 10.12982/CMUMEDJ.2021.38**

Keywords: healthy group, peripheral vestibular loss group, video head impulse test

Introduction

Dizziness and vertigo are symptoms that patients frequently present with in clinics (1). For vertigo, the estimated 1-year prevalence is 4.9% with a female to male ratio of 2.7:1 and an incidence of 1.4% (2,3). Patients may experience symptoms such as imbalance, unsteadiness, nausea, vomiting, feeling faint, hearing loss and tinnitus. Dealing with treatment of patients with dizziness and vertigo should start with taking a complete history and conducting a physical examination. Vestibular function tests can have an important role in the evaluation of vestibular disorders. The caloric test is one of the vestibular function tests used to assess the vestibule-ocular reflex (VOR) function of the horizontal semicircular canals (SCCs) at a very low frequency of about 0.003 Hertz (Hz), but that test limits assessment of high frequency VOR (4). The head impulse test is used as a bedside test of the horizontal vestibule-ocular reflex (hVOR) function of peripheral vestibular loss (PVL) patients and uses a stimulus for head movement at a relatively higher frequency, about 2-5 Hz (4) which is similar to the natural physiological head movement frequency range of about 0.1-3 Hz (5).

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However, there are major drawbacks to that test. First, the examiner's report is based on subjective visual observation of the presence of an overt saccade, so can potentially result in a misdiagnosis. Second, each examiner uses different impulse trajectories during each trial, so velocity and acceleration can vary from one trial to the next. Third, some patients hide partial PVL with a covert saccade during head movements. This can lead to misdiagnosis and false negative results caused by the unobservable covert saccades.

The video head impulse test (vHIT) is used to measure eye and head velocity with high-speed video goggles and evaluates the VOR function of all six SCCs individually at physiological frequencies. VHIT can be complementary to other vestibular tests such as the caloric test and the vestibular evoked myogenic potential tests, improving the accuracy of identifying deficits of the individual vestibular apparatus. VHIT provides information on VOR gain and can detect corrective saccades (CS), both overt and covert, in PVL patients. VHIT is noninvasive, take a short time, is easily repeatable, and uses portable equipment (6,7).

Objective

The objective of this study was to compare the results of vHIT between the patients with PVL and healthy groups and to determine the sensitivity and specificity of vHIT with PVL patients.

Methods

Subjects

From November 2018 to March 2019, a total of 76 subjects were divided into two groups at the Dizziness and Balance Disorders Clinic, Ramathibodi Hospital, Mahidol University. The PVL group consisted of 38 subjects who had been diagnosed with PVL by an otolaryngologist or an otoneurologist using the criteria defined by the International Classification of Vestibular Disorders (8). The healthy group consisted of 38 volunteers with no history of vestibular disorders or symptoms. Subjects who had a history and/or symptoms of a neurological disorder, restricted head and neck movement, an eye abnormality and/or abnormal eye movements, active nystagmus caused by vestibular disorders due to having been examined during a non-active phase of their disease because they had vertigo and had already entered to the emergency department, and those who were not able to wear a head strap because of a skin lesion or were unable to understand the test method were excluded.

All subjects were performed by using the ICS vHIT system (GN Otometrics, Denmark) which consisted of portable goggles, three orthogonal gyroscopes, and a laser projector with a high-speed digital video camera (250 Hz) to record eye velocity. The subjects were instructed to maintain fixation on a target located on the wall one meter directly in front of them. They were instructed to relax their neck muscles, not to blink, and to keep their eyes wide open throughout the test. Calibration was achieved using laser dots on which the subject had to fixate for each direction. The subjects' heads were passively moved with brief, abrupt, high acceleration through a small angle in three planes which consisted of the horizontal SCC, left-anterior right-posterior (LARP), and right-anterior left-posterior (RALP), respectively. The horizontal plane was performed by applying a horizontal impulse to the head at a 10-20 degree angle, at a peak velocity of about 120-250 degrees/second and a peak acceleration of about 1,200-2,500 degrees/second^2. The vertical plane was performed while the subject's head was rotated to the right for LARP stimulation, to the left for RALP stimulation, and at 35-45 degrees from the fixation target. That was followed by backward (chin up) and forward (chin down) movements of the head through a 10 to 20 degree angle, a peak head velocity of 100-250 degrees/sec, and a peak acceleration of about 1,000-2,500 degrees/second^2. During a complete test, 20 acceptable impulses were delivered with unpredictable timing in each direction. VHIT analysis depends mainly on VOR gain and VOR gain asymmetry (GA). The normal VOR gain cut-off values were specified by the manufacturer (ICS otometric vHIT, as being more than 0.8 for horizontal SCCs and 0.7 for vertical SCCs. The normal VOR GA

cut-off value is less than 13.3% (7,9,10).

This study was approved by the Ethical Review Committee for Human Research, Faculty of Medicine, Ramathibodi Hospital, Mahidol University. All subjects signed consent forms before participating in the study.

Statistical analysis

Statistical analyses were performed using SPSS version 20. The data for all subjects were numerically captured. Continuous variables were expressed as mean ± standard deviation (S.D.) and categorical variables were expressed as frequencies and percentages. Two independent-sample t-tests were used to compare the mean VOR gain and the mean VOR GA between the two groups. $p < 0.05$ were considered statistically significant.

Results

The PVL group included patients with a diagnosis of vestibular migraine (n=12). It is hypothesized that the pathophysiology of vestibular migraine is due to activation of the trigeminovascular system contributes to a sterile inflammatory response of the intracranial vessels (11,12). The trigeminovascular system affects ischemia caused by vasospasms of the internal auditory artery in the inner ear which can lead to peripheral vestibular and cochlear dysfunction in migraine with and without vertigo (11,12). Benign paroxysmal positional vertigo (BPPV) (n=11), Meniere's disease (n=6), vestibulopathy (n=6) as well as other disorders, i.e., autoimmune inner ear disease (AIED) (n=1), otolith syndrome (n=1) and labyrinthine concussion (n=1). The demographic characteristics of the PVL and healthy groups are shown in Table 1. There were nonsignificant differences in gender and age group between the two study groups with the exception of age between 18-40. Table 2 shows a comparison of the mean VOR gain and the mean VOR GA (%) of all SCCs between the PVL and healthy groups. The mean VOR gain and VOR GA (%) in all planes of the healthy group were greater than those of the PVL group, except the left horizontal VOR gain. There were only nonsignificant differences between all the means of hVOR gain in the two groups. There were significant differences in the means of all vertical VOR gain except the right anterior (RA) VOR gain. Comparison of the means of all VOR GA tests in the two groups found only nonsignificant differences. Table 3 shows the mean VOR gain and VOR GA (%) for the PVL group. All means of the VOR gain were considered to be within normal limits, except RALP of the BPPV group and the RA of the vestibulopathy group. All means of VOR GA were considered to be within normal limits except LARP of the vestibular migraine group, RALP of the BPPV group, vertical planes of the Meniere's disease group, horizontal and RALP of the vestibulopathy group, and the horizontal plane of the other group.

Table 1. Comparison of demographic characteristics of the PVL and healthy groups

Characteristic	PVL group (n=38)	Healthy group (n=38)	*p*-value
Gender			
Male	10 (26.32)	10 (26.32)	1.000
Female	28 (73.68)	28 (73.68)	
Age (years)			
18-40	5 (13.16)	9 (23.68)	*0.000
41-60	21 (55.26)	14 (36.84)	0.690
61-70	12 (31.58)	15 (39.47)	0.270
Range min/max (years)	28/70	20/70	
Mean ± SD (years)	54.18±11.24	50.50±16.42	0.470

PVL, peripheral vestibular loss; SD, standard deviation; *$p < 0.050$

Table 2. Comparison of the means ± S.D. of VOR gain and the means ± S.D. of VOR GA (%) of all SCCs between the PVL and healthy groups

SCCs	vHIT	PVL group	Healthy group	*p*-value
H	Rt. HVOR gain	1.01±0.22	1.03±0.11	0.570
	Lt. HVOR gain	1.01±0.18	1.01±0.10	0.930
	Horizontal VOR GA (%)	11.68±11.27	8.05±5.95	0.080
LARP	LA VOR gain	0.75±0.16	0.87±0.12	0.000*
	RP VOR gain	0.81±0.18	0.91±0.10	0.010*
	Total VOR gain	0.78±0.17	0.89±0.12	0.00*
	LARP VOR GA (%)	12.84±10.05	9.71±5.64	0.100
RALP	RA VOR gain	0.71±0.19	0.78±0.10	0.060
	LP VOR gain	0.74±0.21	0.87±0.13	0.000*
	Total VOR gain	0.73±0.20	0.83±0.12	0.000*
	RALP VOR GA (%)	15.95±16.06	12.03±7.24	0.180

SCCs, semicircular canals; vHIT, video head impulse test; PVL, peripheral vestibular loss; H, horizontal; Rt., right; Lt., left; HVOR, horizontal vestibule-ocular reflex; VOR GA, vestibule-ocular reflex gain asymmetry; LARP, left-anterior right- posterior; LA, left-anterior; RP, right-posterior; RALP, right-anterior left-posterior; RA, right-anterior; LP, left-posterior; *$p < 0.050$

Discussion

This study found significant dysfunction of the vertical SCCs in the PVL group with the exception of the right-anterior plane between both groups, which indicated that some form of pathology might be involved. Almost all previous studies have reported the range of cut-off values of hVOR GA to be between 8.0-13.3% (7,13,14). One study reported the means of LARP VOR GA and RALP VOR GA as 5.1±4.2% and 7.4±5.2% in healthy groups, but the cut-off value of vertical VOR GA was not found (15). We think that the used cut-off value of vertical VOR GA as 13.3% according to default normative data by the manufacture in present study might be higher to determine abnormal criterion. If we had used a lower cut-off value or if we had established normative data from our clinic, significant differences between the two groups might have been found. Conversely, the horizontal SCCs showed significant non-dysfunction in the PVL group, which indicates that pathology might not be involved. The vast majority of subjects in the present study had posterior SCCs BPPV, results which are in concordance with Ismail, et al. (16) which reported that there were only nonsignificant differences in the mean hVOR gains between the BPPV and the healthy groups. The present study results for vestibular migraine are consistent with those of ElSherif, et al. (17) who reported that the means of hVOR gain were considered to be within normal limits. Most vestibular migraine patients (80.0-89.0%) had normal hVOR gains (12,18). However, the pathophysiology of vestibular migraine is not completely understood, making it a controversial issue. Vestibular migraine affects the vestibular nuclei at the brainstem or at the cortical spreading depression in the multisensory vestibular cortex,

Table 3. Means ± SD of VOR gain and the means ± S.D. of VOR GA (%) for the PVL group

SCCs	vHIT	VM	BPPV	MD	VP	Others
n		12	11	6	6	3
H	RHVOR gain	1.09±0.15	0.96±0.28	0.93±0.19	0.99±0.23	1.04±0.22
	LHVOR gain	1.01±0.10	1.00±0.21	0.85±0.18	1.06±0.21	1.11±0.14
	Total HVOR gain	1.06±0.13	0.98±0.24	0.89±0.18	1.03±0.21	1.08±0.17
	HVOR GA	7.08±2.91	12.36±12.91	12.50±7.66	**15.50±17.96	**18.33±16.62
LARP	LA	0.78±0.17	0.72±0.12	0.71±0.24	0.80±0.15	0.78±0.09
	VOR gain					
	RP	0.88±0.10	0.73±0.15	0.79±0.32	0.86±0.17	0.84±0.15
	VOR gain					
	Total VOR gain	0.83±0.15	0.72±0.13	0.75±0.27	0.83±0.16	0.81±0.11
	LARP VOR GA	**14.33±11.90	11.45±11.54	**16.50±6.66	11.33±8.41	7.67±5.03
RALP	RA	0.74±0.20	**0.69±0.16	0.740.24	**0.67±0.17	0.74±0.25
	VOR gain					
	LP	0.82±0.14	**0.66±0.25	0.70±0.25	0.77±0.18	0.75±0.25
	VOR gain					
	Total VOR gain	0.78±0.17	**0.67±0.20	0.72±0.23	0.72±0.18	0.75±0.22
	RALP VOR GA	13.08±11.69	**19.18±22.90	**21.17±17.70	**15.83±10.03	5.33±2.31

SCCs, semicircular canals; vHIT, video head impulse test; VM, vestibular migraine; BPPV, benign paroxysmal positional vertigo; MD, Meniere's disease; VP, vestibulopathy; others, autoimmune inner ear disease; otolith syndrome and labyrinthine concussion; H, horizontal; VOR GA, vestibule-ocular reflex gain asymmetry; RHVOR, right horizontal vestibule-ocular reflex; LHVOR, left horizontal vestibule-ocular reflex; HVOR, horizontal vestibule-ocular reflex; LARP, left-anterior right- posterior; LA, left-anterior; RP, right-posterior; RALP, right-anterior left-posterior; RA, right-anterior; LP, left-posterior; **out of normal limits

leading to normal VOR gain results (12,17,19). Similarly, Fallahnezhad, et al. (20) reported that there were significant differences in the means of posterior VOR gains between the affected and unaffected sides in posterior SCC BPPV. The free-floating otoconia in the posterior SCCs can disturb the endolymphatic flow, displacing the cupula and leading to abnormal VOR gain results (20). Conversely, the mean hVOR gains were considered to be within normal limits and most patients (92.0-100.0%) showed normal hVOR gain results (20,21). The pathophysiology of BPPV, which is a mechanical disease, should not affect VOR gain (16,18,21). For Meniere's disease, the means of hVOR gain were considered to be within normal limits (16,22) and two studies reported a normal hVOR gain in 100.0% of their patients (23,24). Supporting the hypothesis of crista ampullaris stimulation, these studies demonstrate that vHIT response is related to the neurophysiology of the crista (23,24). VHIT responds to Type I hair cells which likely encode high-frequency, high-acceleration head movement and high velocity conduction of the firing rate (23,24). Meniere's disease predominantly destroys Type II hair cells but leaves Type I hair cells intact, resulting in normal hVOR gain (24,25). A study of the mean hVOR gain did not find evidence of vestibulopathy, but the percentage of patients who had an abnormal hVOR gain has been variously reported to be 12.0% (22) and 12.5% (26). This small percentages of patients indicates that an abnormal hVOR gain is related to vestibular hypofunction but might not affect the VOR pathway (22,26). The pathways of these lesions were not clear and could not be definitively associated with specific vestibular end organs which could involve either SCCs or vestibulo-cochlear nerve dysfunctions being compensated for by a central mechanism. Results might be dependent on whether the test was administered during the active or inactive phase of the peripheral vestibular disorder.

Unfortunately, studies of vHIT of AIED, otolith syndrome, and labyrinthine concussion patients could not be found because of the small number of patients and fewer incidents compared with other PVL, although there have been a few studies on the vertical vHIT of PVL. It is probable that the vertical plane of head movement is more difficult to deliver than the horizontal plane. The vertical SCCs are laid in the diagonal plane on the head, causing a limitation in mobility. Head movement in the vertical direction causes the eyelid to obscure part of the pupil, leading to changes in the eye velocity response. The results of the vertical vHIT were less consistent, making it difficult to ensure the reliability of the test (20).

In the present study, we used only VOR gain to determine sensitivity and specificity levels because VOR gain is the primary parameter used for interpreting vHIT. In the data collected there was no normative data on the characteristics of corrective saccades (CS), which consisted of frequency (%), velocity (degrees/sec) and latency (ms). This was due to limitations of the software version of the instrument which showed only the presence or absence of CS. Furthermore, CS may usually be present in healthy subjects (7). Other relevant factors include, first, the VOR system might be hypometric, requiring small CS to maintain gaze in some healthy subjects (14). Second, CS can be present with normal VOR gain as it is part of the process of aging of the vestibular system (10, 27).

The range of cut-off values of vHIT reported in previous studies was 0.75-0.86 and 0.58-0.75 for horizontal (7,14,16,17,21,27) and vertical VOR gain (7,15-17), respectively. We determined cut-off values defining abnormal VOR gain of 0.8 and 0.7 for horizontal and vertical VOR gain, respectively, which are within range of the default normative data provided by the manufacturer. These cut-off values were reported from McGavie et al. (10) who measured normative values of the VOR gain for all SCCs in healthy subjects.

The sensitivity of vHIT in the current study was low (55.3%) compared with the results of Janky, et al. (78.8%) (27) which examined vestibular loss patients. We think that the reason of using Janky et al. (27) to compare this study would cover all PVL patients which were included our subjects. However, Janky used both VOR gain and the CS to identify vestibular loss patients. The inclusion of both of those parameters would have resulted in sensitivity and specificity levels different from those reported in the present study which used only VOR gain.

Most of the VOR gain means were abnormal and the presence of CS in the vestibular loss patients resulted in a relatively high sensitivity (78.8%). The low sensitivity of vHIT might be explained as follows. First, the PVL group was tested by vHIT during the non-active phase of their diseases. Most of the means of VOR gain of the PVL group were normal and might not have involved the VOR of SCCs or otolith organs. Some diseases, e.g., vestibular migraine, BPPV, and Meniere's disease, present fluctuating symptoms. When a patient is tested during the non-active disease phase, the VOR gain results are normal, which is different from the active phase. Variation in the duration of diseases might affect the VOR gain results as well. Moreover, some diseases could involve all SCCs or the disease could determine which SCCs are stimulated rather than being destroyed. Second, the healthy group might have had subclinical or mild vestibular dysfunction because the inclusion criteria did not use the Dizziness Handicap Inventory (DHI) questionnaire to assess the healthy subjects. Third, using different cut-off values to define abnormal VOR gain results meant there were different levels of sensitivity (22). The high specificity (84.2%) was relatively close to the percentages reported by Janky, et al. (90%) (27). Thus, the level of specificity could identify subjects who had pathologies in their peripheral vestibular system, i.e., abnormal results of vHIT could be an indication of pathology in the SCCs.

When we considered in specific disorder, study of using the only VOR gain to determine sensitivity and specificity levels was not found. Most previous studies used both VOR gain and CS as parameters (11,16). Blowdow et al. (11) re-

ported the sensitivity of vHIT as 40% and mean canal paresis (CP) to be 16±13% in vestibular migraine patients and 55% (mean CP=38±26%) in Meniere's disease patients compared to the caloric test. Ismail et al. (16) found the sensitivity and specificity of vHIT to be 55% and 53.33%, respectively, compared to the caloric test mean of 7.11±1.52% in BPPV. They also found the sensitivity and specificity of vHIT to be 63.21% and 86.67% compared to the caloric test mean of 32.4±4.25% in Meniere's disease patients (16). Park et al. (28) reported the sensitivity and specificity of vHIT using both VOR gain and GA to be 78.8% and 85.7% compared to the caloric test mean of 65.5% in cases of vestibulopathy.

This study did not use a combination of vHIT and caloric test results to determine PVL, but found sensitivity and specificity levels to be within the range of prior studies (11,16,22,28,29). VHIT is a clinical diagnostic test which adequately evaluates the VOR function of all six SCCs individually, which is different from the caloric test. Both vHIT and caloric test respond to different crista ampullaris stimulation which is related to the neurophysiology of the crista (16,23,24). VHIT stimulates at a physiological high frequency, about 5 Hz, which may destroy Type I hair cells, while the caloric test stimulates at a low frequency, about 0.003 Hz, which may preferentially destroy Type II hair cells. Some studies reported that vHIT is not capable of replacing the caloric test (22,28,29).

Limitations and recommendations

There were some limitations in this study. First, the study did not consider the duration of the symptoms and the stage of the disease in the PVL group. Also, the inclusion criteria for the PVL group did not classify patients according to whether they were in the active or non-active phase of their disease. The duration of the various diseases might also have affected the VOR gain results. Second, the inclusion criteria for healthy subjects did not include the DHI-T questionnaire results to evaluate self-perceived handicaps (30). Future studies should compile normative data on all planes for the general Thai population. VHIT should be used in combination with other diagnostic tests to increase the overall sensitivity and specificity and should be viewed as complementary, as one test in a test battery, to evaluate the audio-vestibular function in PVL. Using a combination of vHIT and caloric tests could increase the overall sensitivity and specificity of diagnoses of PVL. The two tests should be used in combination to test a broader spectrum of hVOR response frequencies.

Conclusions

The study found significant dysfunction of the vertical SCCs in the PVL group. The results suggest that VOR gain and GA could be used to interpret PVL in patients with high specificity, especially where SCCs dysfunction has caused a reduction in VOR gain, although vHIT showed a relatively low sensitivity for the evaluation of PVL patients.

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Conflict of interest

All authors declare there are no conflicts of interest.

References

1. Iwasaki S, Yamasoba T. Dizziness and Imbalance in the Elderly: Age-related decline in the vestibular system. Aging Dis. 2015;6:38-47.
2. Neuhauser HK. Epidemiology of vertigo. Curr Opin Neurol. 2007;20:40-6.
3. Agrawal Y, Ward BK, Minor LB. Vestibular dysfunction: prevalence, impact and need for targeted treatment. J Vestib Res. 2013;23:113-7.
4. Redondo-Martinez J, Becares-Martinez C, Orts-Alborch M, Garcia-Callejo FJ, Perez-Carbonell T, Marco-Algarra J. Relationship between video head impulse test (vHIT) and caloric test in patients with vestibular neuritis. Acta Otorrinolaringol. Esp. 2016;67:156-61.

5. Barin K. Clinical neurophysiology of the vestibular system. In: Katz J, Medwetsky L, Burkard R, Hood L, editors. Handbook of clinical audiology. 6th ed. Philadelphia, PA: Wolters Kluwer Health; 2009. p. 433.
6. Macdougall HG, McGarvie LA, Halmagyi GM, Curthoys IS, Weber KP. The video Head Impulse Test (vHIT) detects vertical semicircular canal dysfunction. PLoS One. 2013;8:e61488.
7. Curthoys IS, McGarvie LA, Weber KP, et al. The video head impulse test (vHIT). In: Jacobson GP, Shepard NT, editors. Balance function assessment and management. 2nd ed. San Diego, CA: Plural publishing; 2016. p. 391-430.
8. jvr-web.org [internet]. The Official Journal of the Barany Society. [cited 2020 May 15]. Available from: http://www.jvr-web.org/ICVD.html.
9. Otometrics. ICS Impulse USB Reference Manual. Taastrup: GN otometrics A/S; 2013.
10. McGarvie LA, MacDougall HG, Halmagyi GM, Burgess AM, Weber KP, Curthoys IS. The video head impulse test (vHIT) of semicircular canal function - Age-dependent normative values of VOR gain in healthy subjects. Front Neurol. 2015;6:154.
11. Blodow A, Heinze M, Bloching MB, von Brevern M, Radtke A, Lempert T. Caloric stimulation and video-head impulse testing in Meniere's disease and Vestibular migraine. Acta Otolaryngol. 2014;134:1239-44.
12. Yollu U, Uluduz DU, Yilmaz M, Yener HM, Akil F, Kuzu B, et al. Vestibular migraine screening in a migraine-diagnosed patient population, and assessment of vestibulocochlear function. Clin Otolaryngol. 2017;42:225-33.
13. Eza-Nunez P, Farinas-Alvarez C, Fernandez NP. Comparison of three diagnostic tests in detecting vestibular deficit in patients with peripheral vestibulopathy. J Laryngol Otol. 2016;130:145-50.
14. Yang CJ, Lee JY, Kang BC, Lee HS, Yoo MH, Park HJ. Quantitative analysis of gains and catch-up saccades of video-head-impulse testing by age in normal subjects. Clin Otolaryngol. 2016;41:532-8.
15. Guan Q, Zhang L, Hong W, Yang Y, Chen Z, Lu P, et al. Video Head Impulse Test for Early Diagnosis of Vestibular Neuritis Among Acute Vertigo. Can J Neurol Sci. 2017;44:556-61.
16. Ismail N, Behairy R , Brakat D. Video head impulse test finding in vestibular lesions. J Otolaryngol-ENT Res. 2019;11:29-36.
17. ElSherif M, Reda MI, Saadallah H, Mourad M. Video head impulse test (vHIT) in migraine dizziness. J Otol. 2018;13:65-7.
18. Mangabeira Albernaz PL, Zuma EMFC. The video head impulse test. Acta Otolaryngol. 2014;134:1245-50.
19. Lempert T, Brevern M. Vestibular migrain. In: Aminoff M, Boller F, Swaab D, editors. Handbook of clinical neurology. Amsterdam: Elsevier; 2016. p. 301-16.
20. Fallahnezhad T, Adel Ghahraman M, Farahani S, Hoseinabadi R, Jalaie S. Vestibulo-ocular reflex abnormalities in posterior semicircular canal Benign Paroxysmal Positional Vertigo: A pilot study. Iranian J Otorhinolaryngol. 2017;29:269-74.
21. Cinar Y, Bayram A, Culfa R, Mutlu C. Analyses with the video head impulse test during the canalith repositioning maneuver in patients with isolated posterior semicircular canal benign paroxysmal positional vertigo. Turk Arch Otorhinolaryngol. 2018;56:81-4.
22. van Esch BF, Nobel-Hoff GE, van Benthem PP, van der Zaag-Loonen HJ, Bruintjes TD. Determining vestibular hypofunction: start with the video-head impulse test. Eur Arch Otorhinolaryngol. 2016;273:3733-9.
23. Rubin F, Simon F, Verillaud B, Herman P, Kania R, Hautefort C. Comparison of video Head Impulse Test and caloric reflex test in advanced unilateral definite Meniere's disease. Europ Ann Otorhinolaryngol. 2018; 135:167-9.
24. 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" Meniere's disease. Am J Audiol. 2015;24:1-10.
25. Fukushima M, Oya R, Nozaki K, Eguchi H, Akahani S, Inohara H, et al. Vertical head impulse and caloric are complementary but react opposite to Meniere's disease hydrops. Laryngoscope. 2018:1-7.
26. Jafarzadeh S, Golrokhian Sani MR, Mokhtari Amirmajdi N, Firouzi M. Evaluation of bilateral vestibular dysfunction in Iranian adults and elderlies by electronystagmography and video Head Impulse Test. Iranian J Otorhinolaryngol. 2019;31:103-7.
27. Janky KL, Patterson J, Shepard N, Thomas M, Barin K, Creutz T, et al. video Head Impulse Test (vHIT): The role of corrective saccades in identifying patients with vestibular loss. Otol Neurotol. 2018;39:467-73.
28. Park P, Park JH, Kim JS, Koo JW. Role of video-head impulse test in lateralization of vestibulopathy: Comparative study with caloric test. Auris Nasus Larynx. 2017;44:648-54.
29. Bartolomeo M, Biboulet R, Pierre G, Mondain M, Uziel A, Venail F. Value of the video head impulse test in assessing vestibular deficits following vestibular neuritis. Eur Arch Otorhinolaryngol. 2014;271:681-8.
30. Emasithi A, Kosiyaporn N, Prapawanang Z. Translation and validity of the Thai version of the Dizziness Handicap Inventory. J Vestib Res. 2016;26:202.

การศึกษาเปรียบเทียบผลการตรวจ video head impulse test (vHIT) ระหว่างกลุ่มผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลายและกลุ่มคนปกติ

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วัตถุประสงค์ เพื่อเปรียบเทียบผลการตรวจ video head impulse test (vHIT) ในกลุ่มผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลายและกลุ่มคนปกติ และศึกษาค่าความไวและความจำเพาะของ vHIT ในกลุ่มผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลาย

วิธีการ ผู้เข้าร่วมการศึกษาทั้งหมดจำนวน 76 คน ประกอบด้วยกลุ่มผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลายจำนวน 38 คน และกลุ่มปกติจำนวน 38 คน ทั้งสองกลุ่มได้รับการตรวจ vHIT ด้วยเครื่อง ICS impulse ในระนาบ horizontal, left–anterior right–posterior (LARP) และ right–anterior left–posterior (RALP) ตามลำดับ ระหว่างเดือนพฤศจิกายน พ.ศ. 2561 ถึงเดือนมีนาคม พ.ศ. 2562

ผลการศึกษา ค่าเฉลี่ยของ vertical vestibule–ocular reflex gain (VOR) ทุกระนาบแตกต่างอย่างมีนัยสำคัญทางสถิติระหว่างสองกลุ่มยกเว้น anterior VOR gain ข้างขวา แม้ว่าค่าเฉลี่ยของ horizontal VOR gain และ VOR gain asymmetry (GA) ทุกระนาบไม่แตกต่างอย่างมีนัยสำคัญทางสถิติระหว่างสองกลุ่ม ค่าความไวและความจำเพาะของ vHIT คือ ร้อยละ 55.26 และร้อยละ 84.21 ตามลำดับเมื่อได้ผลตรวจ VOR gain ผิดปกติ และ/หรือผลตรวจ VOR GA ผิดปกติ

สรุป VHIT สามารถบ่งชี้ความผิดปกติอย่างมีนัยสำคัญทางสถิติของ vertical semicircular canals (SCCs) ในกลุ่มผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลาย ค่า VOR gain และ GA สามารถนำมาใช้ในการแปลผลในผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลายซึ่งได้ค่าความจำเพาะสูงโดยเฉพาะความผิดปกติของ SCCs ซึ่งเป็นสาเหตุทำให้ค่า VOR gain ลดลง ถึงแม้ว่าค่าความไวของ vHIT ต่ำ แต่ก็เพียงพอต่อการประเมินผู้ป่วยที่มีปัญหาระบบประสาทการทรงตัวส่วนปลาย และควรนำมาใช้ตรวจร่วมกับการทดสอบระบบการทรงตัวอื่น **เชียงใหม่เวชสาร 2564;60(4):427-35. doi: 10.12982/CMUMEDJ.2021.38**

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