

Analyses with the Video Head Impulse Test During the Canalith Repositioning Maneuver in Patients with Isolated Posterior Semicircular Canal Benign Paroxysmal Positional Vertigo

Yusuf Çınar¹ , Ali Bayram¹ , Ramazan Culfa² , Cemil Mutlu¹ 

¹Department of Otorhinolaryngology, Kayseri Training and Research Hospital, Kayseri, Turkey

²Department of Otorhinolaryngology, Sorgun State Hospital, Yozgat, Turkey

Original Investigation 

Abstract

Objective: To evaluate the posterior semicircular canal (PSCC) functions using video head impulse test (vHIT) during canalith repositioning maneuver (CRM) treatment in patients with isolated, posterior semicircular canal benign paroxysmal positional vertigo (PSCC-BPPV).

Methods: A total of 44 subjects comprising of 24 subjects with isolated PSCC-BPPV and 20 age- and sex-matched healthy control subjects were enrolled in the present study. vHIT was performed for the affected PSCC before and just after CRM and at the third and seventh day and first month to evaluate vestibulo-ocular reflex (VOR) gain, gain asymmetry (GA), and corrective saccades. Repeated determinations of VOR gain and GA were compared to evaluate the time course of vHIT measurements during CRM treatment in isolated PSCC-BPPV patients,

and the values were also compared with the control group.

Results: VOR gains and GA values were not statistically different before and after CRM and at the third-day, seventh-day and first-month visits for the affected PSCC. Moreover, values did not differ between the BPPV and control groups, and none of the subjects demonstrated corrective saccades.

Conclusion: To our knowledge, this study is the first report to investigate vHIT measurements with a time course of alterations during CRM treatment in PSCC-BPPV patients. vHIT may not provide an additional contribution for evaluating vestibular dysfunction during the diagnosis and treatment of isolated PSCC-BPPV.

Keywords: Benign paroxysmal positional vertigo, head impulse test, semicircular canals, vestibular function tests



ORCID IDs of the authors:

Y.Ç. 0000-0002-8617-0267;
 A.B. 0000-0002-0061-1755;
 R.C. 0000-0002-8994-271X;
 C.M. 0000-0003-3585-6549.

Cite this article as: Çınar 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(2): 81-4.

Introduction

Benign paroxysmal positional vertigo (BPPV) is a common vestibular disease with an estimated lifetime prevalence of 2.4% (1). BPPV is characterized by a sense of vertigo that arises in certain head positions (2), and the pathophysiology of the disease is based on two major theories: cupulolithiasis (3) and canalithiasis (4). According to the widely accepted theory (canalithiasis), otoconia dislodged from the utricular or saccular macula subsequently move into the semicircular canal (SCC) and cause an endolymph displacement that stimulates the cupula of the SCC (4). The posterior semicircular canal (PSCC) is the most frequently affected canal due to its anatomical alignment (5). Posterior semicircular canal benign paroxysmal positional vertigo (PSCC-BPPV) can be effectively treated with the canalith repositioning maneuver (CRM) described

by Epley with the aim of moving back the dislodged otoconia to the vestibule and thus resolving the symptoms (6). The available data in the literature demonstrate that CRM is a safe and effective short-term treatment for PSCC-BPPV (7). The short-term success for the complete resolution of symptoms and the conversion of the Dix-Hallpike test from a positive to a negative after one CRM treatment session ranges from 34% to 89% (8-11).

Video head impulse test (vHIT) is a video-camera system that enables the individual measurement of the vestibulo-ocular reflex (VOR) of each SCC. It records eye and head velocity response to brief, unpredictable, and passive head rotations, which is called a head impulse (12). The vHIT system enables calculation of VOR gains and gain asymmetry (GA) and also detects corrective saccades including overt or covert saccades (13).

This study was presented at the 39th Turkish National Congress of Otolaryngology Head and Neck Surgery, November 8-12, 2017, Antalya, Turkey.

Corresponding Author:
 Ali Bayram; dralibayram@gmail.com

Received Date: 03.01.2018

Accepted Date: 20.02.2018

© Copyright 2018 by Official Journal of the Turkish Society of Otorhinolaryngology and Head and Neck Surgery Available online at www.turkarchotorinolaryngol.net

DOI: 10.5152/tao.2018.3166

In the English literature, there is only one relevant study that investigated vHIT parameters in PSCC-BPPV (14). In the present study, we aimed to evaluate PSCC functions through vHIT during CRM of Epley in patients with isolated PSCC-BPPV, and to our knowledge, the present study is the first report investigating vHIT measurements with a time course of alterations during CRM treatment in isolated PSCC-BPPV patients.

Methods

Twenty-four patients with isolated PSCC-BPPV (BPPV group) and 20 age and sex-matched healthy subjects (control group) were recruited in the present study between February 2016 and August 2016. Approval for the study protocol was obtained from the Ethics Committee of Erciyes University with reference number 2016/377, and written informed consent was obtained from the participants.

After obtaining a detailed medical history and a meticulous evaluation of hospital records, all participants underwent physical and otolaryngological examinations including Dix-Hallpike and Roll tests. The diagnosis of isolated PSCC-BPPV was made according to the Dix-Hallpike test, and subjects with the presence of spontaneous nystagmus and positive signs or symptoms regarding lateral or anterior canal BPPV, ototoxic drug use, blindness, neurological disorders, or poor neck range of motion were excluded from the study.

In the BPPV group, vHIT was performed before and just after the CRM of Epley for the affected PSC in the first visit. Subsequent vHIT procedures were performed on the third and seventh day and one month after the first visit, and CRM was repeated if the presence of symptoms or positive signs was determined in the Dix-Hallpike test. Successful treatment was defined as the absence of positioning vertigo and positioning nystagmus. Non-responder patients were instructed to continue self-treatment until no vertigo could be induced. The values of VOR gains and GA measured before and after CRM, and at the third and seventh day and first month for the affected PSCC were compared among each other, and the values were also compared with the control group. In the first visit, we also measured lateral and anterior SCC functions before CRM for detecting any SCC dysfunction accompanying PSC-BPPV. Subjects in the control group underwent vHIT measurements for each SCC, and vHIT parameters were calculated for 40 ears (i.e., 20 patients).

The vHIT measurements were performed using a portable video-oculography system device (EyeSeeCam; Interacoustics, Eden Prairie, USA) in a seated position under room light. A pair of lightweight goggles integrated with a gaze-driven, high-speed digital camera system (sampling rate of 220 Hz) that recorded real-time eye movement, a motion sensor that measured head movement, and a laser light for calibration were tightly fitted onto the subject's head. The subjects underwent at least 20 unpredictable head impulses (amplitude 15–20 degrees, duration 150–200 ms, target head velocity 100–200 degree/s) along the planes of the lateral semicircular canal (LSCC) and in left anterior right posterior and right anterior left posterior planes for

testing the vertical SCCs. The subjects were fixated on a target located approximately 1 m straight ahead during the procedure. A pair of horizontal (left lateral/right lateral) VOR gains and two pairs of vertical (left anterior/right posterior and left posterior/right anterior) VOR gains were measured, and their bilateral asymmetry was automatically calculated separately. Corrective saccades were classified as a covert saccade when they occurred during the head movement and as an overt saccade when they occurred after the head movement.

Statistical evaluations were performed with the Statistical Package for the Social Sciences (v. 21; SPSS Inc., Chicago, USA). Data were tested for normal distribution using the Kolmogorov-Smirnov test and expressed as mean±SD. A repeated measures ANOVA was used for the comparison of repeated measures, and chi-square was used for the comparison of categorical variables. Independent samples t-test was performed for the comparison of VOR gain and GA values between the BPPV and control groups. A p value less than 0.05 was considered significant for all comparisons.

Results

The demographic features of the BPPV and control groups are shown in Table 1. In the BPPV group, 14 patients had right ear involvement (58.3%), whereas 10 patients (41.7%) had left ear involvement. The mean duration of vertigo was 10.47±9.5 days. On the third- and seventh-day visits, five patients (20.8%) had positive findings in the Dix-Hallpike test or the presence of symptoms, whereas there were three patients (12.5%) with positive findings on the 1st-month visit; however, these patients did not demonstrate any abnormality in vHIT measurements.

In the control group, there were no significant differences between left and right ears in terms of VOR gains, and the average VOR gains for PSCC, anterior semicircular canal (ASCC), and LSCC were 0.96±0.14, 0.96±0.13, and 0.99±0.09, respectively, for a total of 40 ears. VOR gains were classified as abnormal if gain values were below mean_{normal}-2SD (13). Hence, the gain thresholds were 0.68, 0.70, and 0.81 for PSCC, ASCC, and LSCC, respectively.

In the BPPV group, repeated measures of VOR gain and GA values of the affected PSCC were not statistically different before and after the CRM and for the following visits (Table 2). Also, there were no significant differences in terms of VOR gain (Table 3) and GA (Table 4) values between the BPPV and control groups before and after maneuver and for the third- and seventh-day and first- month visits for the affected PSCC. An abnormal VOR gain for PSCC was detected in two patients before maneuver in the BPPV group, whereas none of the sub-

Table 1. Demographic features of the groups

	BPPV (n=24)	Control (n=20)	p
Age (years)	42.8±11.7	37.4±13.3	>0.05
Gender (male/female)	10/14	10/10	>0.05

BPPV: benign paroxysmal positional vertigo

Table 2. Repeated measures of VOR gain and GA values for the affected PSSC in the BPPV group (n=24)

	Before maneuver	After maneuver	3 rd day	7 th day	1 th month	p
VOR gain	0.92±0.18	1±0.13	0.98±0.14	1.01±0.17	0.92±0.21	0.101
GA	3±1.79	2.77±1.86	2.81±1.9	3±2.01	2.81±1.95	0.498

GA: gain asymmetry; VOR: vestibulo-ocular reflex; PSSC: posterior semicircular canal; BPPV: benign paroxysmal positional vertigo

Table 3. Comparison of VOR gains for PSSC between the BPPV and control groups

	Before maneuver	After maneuver	3 rd day	7 th day	1 th month
BPPV (n=24)	0.92±0.18	1±0.13	0.98±0.14	1.01±0.17	0.92±0.21
Control (n=40)	0.96±0.14	0.96±0.14	0.96±0.14	0.96±0.14	0.96±0.14
p	0.299	0.262	0.638	0.239	0.462

BPPV: benign paroxysmal positional vertigo; PSSC: posterior semicircular canal; VOR: vestibulo-öküler refleks

Table 4. Comparison of GA for PSSC between the BPPV and control groups

	Before maneuver	After maneuver	3 rd day	7 th day	1 th month
BPPV (n=24)	3.38±1.46	2.75±1.77	2.88±1.87	3.38±2.12	2.88±2
Control (n=40)	2.78±1.94	2.78±1.94	2.78±1.94	2.78±1.94	2.78±1.94
p	0.197	0.959	0.84	0.252	0.844

BPPV: benign paroxysmal positional vertigo; PSSC: posterior semicircular canal; GA: gain asymmetry

jects had an abnormality in the control group. There were no significant differences between the BPPV and control groups in terms of the number of patients with VOR gain abnormality ($p>0.05$). For LSCC and ASCC, none of the subjects had a VOR gain abnormality in the BPPV and control groups in the first visit. None of the subjects in the BPPV and control groups demonstrated overt or covert saccades.

Discussion

Video head impulse test is an objective and rapid diagnostic tool that enables VOR measurements of each SCC individually at high frequency. Given the fact that it is well tolerated, as it does not induce nausea and vomiting, vHIT is recommended in the first line to test each SCC function (12, 13).

In the present study, we aimed to describe the time course of vHIT parameter alterations in isolated PSSC-BPPV patients during CRM treatment with repeated measures. A similar study was conducted by Bremova et al. (15), investigating vestibular-evoked myogenic potentials (VEMP) before and after liberatory maneuvers in patients with PSSC-BPPV. In their study, one week after liberatory maneuvers, 66.6% of the subjects were free of symptoms, whereas the therapy response rate rose to 73.9% after one month. They demonstrated that ocular VEMP amplitudes increased at one week after a successful maneuver, whereas cVEMP did not differ significantly. In the present study, the short-term success rate was 79.2% at the third- and seventh-day visits, which was consistent with literature (8-11). In addition, the repeated measurements of VOR gain and GA values were not significantly different for the affected PSC.

The number of studies focusing on vHIT findings in patients with BPPV is very small in the literature. In the single relevant study written in English, Fallahnezhad et al. (14) studied vHIT in 29 patients with unilateral PSSC-BPPV and demonstrated abnormal PSSC-VOR gain in 55.17% of patients and concluded that PSSC gains may be reduced in patients with PSSC-BPPV. In the present study, the majority of patients in the BPPV group had right ear involvement (58.3%), with female predominance (58.3%), which is consistent with previous reports (14-17). However, although high short-term resolution rates were achieved in BPPV patients, no significant differences were observed regarding VOR gain and GA between the repeated measurements before and after CRM. In addition, the values of vHIT parameters and the number of patients with VOR gain abnormality were not statistically different between the BPPV and control groups. These findings may be attributed to various factors related to the pathophysiology of BPPV disease as well as the limitations of the vHIT procedure. BPPV is a mechanical disease; hence, this pathological condition may not affect the VOR gains. In addition, the measurement of vHIT necessitates an experienced examiner who can perform a reliable rapid head movement. A peak head velocity, as large as lateral canal testing, may not be obtained during the vertical canal testing in the vHIT procedure. In order to overcome examiner dependent limitations, all vHIT procedures were performed by a single experienced examiner in the present study, and also a control group consisting of 20 healthy subjects was constituted for establishing normative data of VOR gain thresholds instead of using default values. However, a significant difference was not observed in terms of VOR gains in isolated PSSC-BPPV patients. In a literature review of vHIT, Alhabib and Saliba (12)

stated that vHIT will be abnormal only if there is more than 40% reduction in the vestibular function. After obtaining a detailed medical history and meticulous evaluation of hospital records, patients with any central or peripheral vestibular disorders other than BPPV were not recruited for this study. Thus, as BPPV is a vestibular dysfunction due to an overstimulation of SCCs rather than a vestibular reduction, vHIT parameters might not be influenced in patients with BPPV in the present study. Likewise, corrective saccades were not detected in the BPPV group, which is also consistent with literature (14). The presence of corrective saccades may be related to a severe amount of vestibular dysfunction, and saccades that were detected in BPPV could be referred to other comorbid vestibular pathologies.

Conclusion

To our knowledge, the present study is the first report investigating vHIT measurements with a time course of alterations during CRP treatment in PSCC-BPPV patients. The vHIT may not provide an additional contribution in evaluating vestibular dysfunction during the diagnosis and treatment of isolated PSCC-BPPV.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Erciyes University (2016/377; Date: 06/24/2016).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - Y.Ç., C.M.; Design - Y.Ç., C.M.; Supervision - A.B., C.M.; Resource - Y.Ç., C.M., A.B., R.C.; Materials - Y.Ç., C.M., A.B., R.C.; Data Collection and/or Processing - Y.Ç., C.M., A.B., R.C.; Analysis and/or Interpretation - Y.Ç., C.M., A.B., R.C.; Literature Search - Y.Ç.; Writing - Y.Ç., A.B.; Critical Reviews - A.B., C.M.

Acknowledgements: The authors thank Audiologist Ahmet Kaleli from the Department of Audiology, Kayseri Training and Research Hospital.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: This study was supported by Kayseri Training and Research Hospital (Grant number: 2016/50).

References

1. von Brevern M, Radtke A, Lezius F, Feldmann M, Ziese T, Lempert T, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry* 2007; 78: 710-5. [CrossRef]

2. Brandt T. Positional and positioning vertigo and nystagmus. *J Neurol Sci* 1990; 95: 3-28. [CrossRef]
3. Schuknecht HF. Cupulolithiasis. *Arch Otolaryngol* 1969; 90: 765-78.
4. Hall SF, Ruby RF, McClure JA. The mechanics of benign paroxysmal positional vertigo. *J Otolaryngol* 1979; 8: 151-8. [CrossRef]
5. Korres SG, Balatsouras DG. Diagnostic, pathophysiologic and therapeutic aspects of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 2004; 131: 438-44. [CrossRef]
6. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1992; 107: 399-404. [CrossRef]
7. Helminski JO. Effectiveness of the canalith repositioning procedure in the treatment of benign paroxysmal positional vertigo. *Phys Ther* 2014; 94: 1373-82. [CrossRef]
8. Munoz JE, Miklea JT, Howard M, Springate R, Kaczorowski J. Canalith repositioning maneuver for benign paroxysmal positional vertigo: randomized controlled trial in family practice. *Can Fam Physician* 2007; 53: 1048-53.
9. Froehling DA, Bowen JM, Mohr DN, Brey RH, Beatty CW, Wollan PC, et al. The canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo: a randomized controlled trial. *Mayo Clin Proc* 2000; 75: 695-700. [CrossRef]
10. Lynn S, Pool A, Rose D, Brey R, Suman V. Randomized trial of the canalith repositioning procedure. *Otolaryngol Head Neck Surg* 1995; 113: 712-20. [CrossRef]
11. Yimtae K, Srirompotong S, Srirompotong S, Sae-Seaw P. A randomized trial of the canalith repositioning procedure. *Laryngoscope* 2003; 113: 828-32. [CrossRef]
12. Alhabib SF, Saliba I. Video head impulse test: a review of the literature. *Eur Arch Otorhinolaryngol* 2017; 274: 1215-22. [CrossRef]
13. Bayram A, Kaya A, Mutlu M, Hira İ, Tofar M, Özcan İ. Clinical practice of horizontal video head impulse test in healthy children. *Kulak Burun Bogaz Ihtis Derg* 2017; 27: 79-83. [CrossRef]
14. 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. *Iran J Otorhinolaryngol* 2017; 29: 269-74.
15. Bremova T, Bayer O, Agrawal Y, Kremmyda O, Brandt T, Teufel J, et al. Ocular VEMPs indicate repositioning of otoconia to the utricle after successful liberatory maneuvers in benign paroxysmal positioning vertigo. *Acta Otolaryngol* 2013; 133: 1297-303. [CrossRef]
16. von Brevern M, Seelig T, Neuhauser H, Lempert T. Benign paroxysmal positional vertigo predominantly affects the right labyrinth. *J Neurol Neurosurg Psychiatry* 2004; 75: 1487-8. [CrossRef]
17. De Stefano A, Kulamarva G, Citraro L, Neri G, Croce A. Spontaneous nystagmus in benign paroxysmal positional vertigo. *Am J Otolaryngol* 2011; 32: 185-9. [CrossRef]