Vestibular Evoked Myogenic Potentials After Epleys Manoeuvre Among Individuals with Benign Paroxysmal Positional Vertigo

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Abstract VEMP abnormalities in individuals with BPPV are often reported to be associated with utricle and saccule degeneration. The aim of the present study is to assess the frequency of VEMP abnormalities using vestibular evoked myogenic potentials in individuals with Posterior canal Benign Paroxysmal Positional Vertigo (BPPV) after Epley's manoeuvre. 36 individuals (36 ears) with definite posterior canal BPPV and 36 healthy controls were considered for the present study. All the them underwent otoscopic examination, Dix–Hallpike manoeuvre to diagnose posterior canal BPPV. Further Audiological Evaluation including pure tone audiometry was carried out to rule out vestibular disorders associated with hearing loss. Epley's manoeuvre was performed on all individuals with BPPV by an experienced otorhinolaryngologist. Cervical and Ocular Vestibular Evoked Myogenic Potentials (VEMP) were used to investigate the saccule and utricle functions following Epley's manoeuvre. Cervical VEMP and ocular VEMP abnormalities were observed in 8/36 (22.22%) and 18/36 (50%) affected ears with BPPV respectively. Cervical VEMP responses were reduced in amplitude among 1/36 (2.77%) and absent in 7/36 (19.44%) of affected ears with BPPV. Ocular VEMP responses were reduced in amplitude on 11/36 (30.55%),

followed by absent responses in 5/36 (13.88%) ears with BPPV. Two patients with posterior canal BPPV i.e., 4/64 (5.55%) ears had bilateral absence of ocular VEMP responses. Two ears with BPPV 2/36 (5.55%) had absence of both cervical and ocular VEMP responses in BPPV affected ear. T test showed significant difference ($p < 0.01$) in the amplitude of ocular VEMP among posterior canal BPPV individuals when compared to cervical VEMP. The present study highlights individuals with Posterior canal BPPV may have otoconia dislodgement or macular degeneration of utricle, saccule, both utricle and saccule unilaterally, or bilaterally. VEMP may be useful in evaluating degeneration of both otolith organs associated with BPPV.

Keywords Vestibular evoked myogenic potentials · Benign paroxysmal positional vertigo · Epley's manoeuvre

Introduction

Benign paroxysmal positional vertigo (BPPV) is one of the most common causes of vertigo with a sensation of spinning due to sudden change in head position. For diagnosing posterior canal BPPV, Dix and Hallpike [1] test has been considered as gold standard test over the years. Posterior semi-circular canal is the most common site in more than 85% of individuals with BPPV [2]. Posterior canal BPPV is reported to occur due to canalithiasis i.e., dislodgement of otoconia from utricle and moving into the posterior semi-circular canal due to anatomical reasons [3]. Epley's [4] manoeuvre and other canalith repositioning manoeuvres (CRMs) [5–7] are reported to be very effective for treating BPPV.

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Vestibular Evoked Myogenic Potential (VEMP) are the short latency Myogenic potentials that are commonly used for the evaluation of saccular and utricular functions correspondingly [8]. Air conduction cervical and ocular VEMPs have been studied by several authors in individuals with BPPV and found to be useful in identifying otoconia dislodgement or macular degeneration from utricle [9–11] or saccular regions [12–14]. Review of articles on VEMP with BPPV have showed increased prevalence of ocular VEMP abnormalities when compared to cervical VEMP abnormalities due to the anatomical relation to semi-circular canals. Three types of findings have been reported regarding cervical and ocular VEMP abnormalities in individuals with BPPV namely, (1) VEMP abnormalities in affected ear—increased latencies [14], reduced VEMP amplitude and absent responses [9, 10, 12, 13, 15], (2) augmented responses in unaffected ear [9, 10] and (3) bilateral VEMP abnormalities in chronic patients [11, 12].

There are two research Studies [16, 17] that have assessed VEMP abnormalities after successful CRM. Bre-mova et al. [16] have reported that significantly increased ocular VEMP amplitude in CRM respondents when compared to non CRM respondents, but not in cervical VEMP. Kim et al. [17] did not find any significant effect of CRM over VEMP on 112 individuals with BPPV. Their findings suggest that unilateral BPPV can cause reduced amplitude, absence of response in affected and non affected ears and even bilateral absence of responses. Since there are sparse VEMP studies after canal repositioning in Indian population, the present study was carried out to assess the frequency of VEMP abnormalities in posterior canal BPPV after successful CRM using cervical and ocular VEMP.

Method

Participants

Thirty six acute BPPV individuals with posterior canal BPPV after Epley's manoeuvre (24 males and 12 females) and 36 age and gender matched controls were considered for the present study. Both the group were selected using convenience sampling method. The mean age for clinical group and controls were 38.9 years and 38.5 years respectively in the age range of 21–50 years. Participants in both the group were selected from the out-patient clinics in the upgraded Institute of otorhinolaryngology—Institute of Speech and Hearing, Rajiv Gandhi Government General Hospital Chennai. All of them in the clinical group reported positional vertigo and demonstrated positive result on Dix–Hallpike manoeuvre. Dix–Hallpike manoeuvre was carried out using the guidelines recommended by

American Academy of Otolaryngology Head and Neck Surgery [18]. The diagnosis of BPPV was made if the patient had following inclusion criteria's: (1) a history of positional vertigo, (2) torsional nystagmus/up beating nystagmus (< 60 s) on this Dix–Hallpike manoeuvre and (3) absence of central nervous system disorders associated with positional vertigo. After the diagnosis of BPPV Epley's manoeuvre [4] was carried out by an experienced otologist on the affected side. Participants with posterior canal BPPV were considered for further evaluation (1–3 days) after Epley's manoeuvre. All the participants in the BPPV group have not undergone any other type of treatment for BPPV. Control participants were volunteers who did not have any history of audiological and vestibular pathologies. Before conducting the study, ethical committee clearance was obtained from the local ethical body. Informed consent was obtained from all the participants in both the groups.

Procedure

Detailed case history, Routine Neurotological examination, Puretone audiometry (Threshold and Uncomfortable level testing) and Immittance audiometric evaluations were carried out before VEMP testing to rule out hearing loss and vestibular pathologies related to other causes. All the participants in both the groups had hearing sensitivity within 25 dB HL in the frequency range of 250–8000 Hz in air conduction testing and 250–4000 Hz for bone conduction testing. All of them had uncomfortable levels of 105 dB HL or more.

Both cervical and ocular VEMP recordings were carried out using a dual channel auditory evoked potential system Neuroaudio Version 1.0.103 (Neurosoft, Russia). During the assessment participants were seated in a comfortable chair with an upright posture in a sound treated room. Both the VEMPs were recorded using 500 Hz alternating tone burst stimuli (1-ms rise/fall time and 2-ms plateau) with intensity of 123 dB SPL (100 dB nHL) at a stimulation rate of 5.1/s delivered through ER3A insert ear phones. Stimulus and recording parameters for recording air conduction cervical and ocular VEMP used in this study were as described by various authors [19–22].

Cervical VEMP responses were recorded from the ipsilateral sternocleidomastoid (SCM) muscle using right and left stimulation separately. Surface electrodes were placed on upper one-third of sternocleidomastoid muscle (noninverting electrode), Sternoclavicular junction (inverting electrode) and forehead (common electrode). Participants were asked to turn their head contralaterally (away from the stimulation side) to activate the ipsilateral SCM muscle and the muscle contraction were monitored and maintained between 30 and 70 μ V. Visual feedback

was provided on the computer screen to maintain muscle contraction. Adequate rest periods were also given to acquire fatigue free recording. Pre-stimulus rectification protocol was used to control the effects of muscle tension over the VEMP amplitude within and across the individuals. The inhibitory responses from the Sternocleidomastoid muscle were recorded with analysis window of 80 ms (20 ms pre-stimulus period) and averaged for 200 stimuli. The responses were band pass filtered between 10 and 2000 Hz and amplified with a factor of 5000.

Ocular VEMP responses were recorded from contralateral eye using right and left ear stimulation separately. For ocular VEMP recording, surface electrodes were placed 1 cm below the lower eye lid (non-inverting electrode), 2 cm below the noninverting electrode (inverting electrode) and at forehead (ground electrode). Participants were instructed to elevate their gaze by 30°–35° in the midline to elevate their inferior oblique muscle near to the surface electrode. The excitatory responses from inferior oblique muscle were recorded using an analysis window of 70 ms (10 ms pre-stimulus period) and averaged for 200 stimuli. The responses were band pass filtered between 1 and 1000 Hz and amplified with a factor of 30,000. Each response was recorded twice to check its reproducibility and validity.

Analysis

p1–n1 Responses (Cervical VEMP), n1–p1 responses (ocular VEMP) were identified by two independent

experienced audiologists with minimum of 5 years of experience with VEMP. Absolute latencies of positive and negative peaks, corrected amplitude (cervical VEMP), peak to peak amplitude (ocular VEMP), interaural amplitude ratio (asymmetry ratio) were calculated for further analysis as used in other studies. Amplitude asymmetry ratio of > 31.6% was used as cervical or ocular VEMP abnormality as described in previous studies [9, 11–14]. Complete unilateral or bilateral absence of cervical and ocular VEMP was considered as abnormal response pattern. Descriptive analysis and between ear and group comparisons were carried out using SPSS software version 20.

Results

Out of 36 participants in BPPV group, Cervical VEMP and ocular VEMP abnormalities were observed in 8/36 (22.22%) and 18/36 (50%) ears with Posterior canal BPPV respectively. Cervical VEMP and Ocular VEMP responses were considered normal if the latencies and amplitudes fall within 2 standard deviation of control group. All the patients in control group had cervical responses and ocular VEMP responses. 64/64 (100%) ears had normal VEMP responses. Out of 8 Cervical VEMP abnormal, responses were reduced in amplitude in 1/36 (2.77%) and absent in 7/36 (19.44%) affected ears with BPPV. Out of 18 patients with abnormal Ocular VEMP, responses were reduced in amplitude on 11/36 (30.55%), followed by absent

Fig. 1 40-year-old Participant with right posterior canal BPPV after Epley's manoeuvre showing absence of both cervical and ocular VEMP on right side stimulation

Table 1 Parameters of cervical VEMP measured in BPPV and controls group

Cervical VEMP parameters	Ears	BPPV		Ears	Controls	
		Mean	SD		Mean	SD
p1 latency (ms)	Affected ear	13.62	.96	Matched ears	13.96	1.04
n1 latency (ms)		22.09	1.07		22.64	.98
p1–n1 amplitude (μV)		1.44	.62		1.58	.47
p1 latency (ms)	Unaffected ear	13.58	1.05	Non-matched ears	13.96	1.02
n1 latency (ms)		22.34	.79		23.06	1.37
p1–n1 amplitude (μV)		1.60	.70		1.50	.55
Assymetry ratio (%)		13.56	11.93		12.19	8.26

Table 2 Parameters of ocular VEMP measured in BPPV and controls group

Ocular VEMP parameters	Ears	BPPV		Ears	Controls	
		Mean	SD		Mean	SD
n1 latency (ms)	Affected ear	10.23	.46	Matched ears	10.03	.41
p1 latency (ms)		15.26	.98		15.23	.97
n1–p1 amplitude (μV)		3.57	3.82		9.89	3.87
n1 latency (ms)	Unaffected ear	10.59	1.14	Non-matched ears	10.05	.50
p1 latency (ms)		15.12	1.30		15.02	.98
n1–p1 amplitude (μV)		4.32	4.15		10.81	4.03
Assymetry ratio (%)		7.09	28.52		13.52	8.22

responses in 7/36 (19.44%) ears with BPPV. Two patients with posterior canal BPPV i.e., 4/64 (5.55%) ears had bilateral absence of ocular VEMP responses. Two ears with BPPV 2/36 (5.55%) had absence of both cervical and ocular VEMP responses in BPPV affected ear. A 40-year-old participant with posterior canal BPPV showing absence of both cervical and ocular VEMP in Right ear is shown in Fig. 1. Ears which had absent responses were excluded from further analysis.

The mean latencies and amplitude of cervical VEMP (p1–n1) and ocular VEMP (n1–p1) of affected ears, unaffected ears with BPPV and matched, nonmatched ears of controls were presented in Tables 1 and 2 respectively.

Independent sample *t* test was used to compare means of latencies and amplitude between groups (BPPV and control group). There was no statistically significant difference between the p1–n1, p1–n1 amplitude and asymmetry ratio of Cervical VEMP, n1–p1 latencies, asymmetry ratio of Ocular VEMP between BPPV and control group. N1–p1 amplitude of ocular VEMP were significantly lower than the control groups as seen in Table 3 and Fig. 2. Paired *t* test was used to compare means of affected and unaffected ears (BPPV) and matched and unmatched ears in control group. There was no significant difference ($p > 0.01$)

Table 3 Independent sample *t* test results between BPPV and control group

	<i>t</i>	<i>df</i>	<i>P</i>
<i>Cervical VEMP</i>			
p1 latency	– 2.102	135.00	0.037
n1 latency	– 6.329	135.00	0.031
p1–n1 amplitude	– 0.131	135.00	0.896 ^a
Assymetry ratio	0.547	63.00	0.586
<i>Ocular VEMP</i>			
n1 latency	4.816	130.00	0.033 ^a
p1 latency	1.023	130.00	0.308
n1–p1 amplitude	– 9.156	130.00	< 0.001
Assymetry ratio	– 1.284	60.00	0.204 ^a

Student's *T* test

^aLevene's test is significant ($p < 0.05$), suggesting a violation of the equal variance assumption

between affected and non-affected ears of BPPV individuals in the latency and amplitude parameters of cervical and ocular VEMP responses. Control group did not show any significant difference between matched and unmatched ears in all the parameters measured.

Fig. 2 Cervical and ocular VEMP amplitudes between BPPV and controls

Discussion

The present study was aimed to study individuals with posterior canal BPPV after Epley's manoeuvre using Cervical and ocular VEMP. From this study we found Cervical and ocular VEMP abnormalities in 22.22% and 50% of patients with posterior canal BPPV respectively. Result from the present study is supported by several studies who report 10–50% air conduction cervical VEMP [12–14] and ocular VEMP abnormalities [9–11] in patients with BPPV.

In the present study we observed frequent ocular VEMP abnormalities compared to cervical VEMP abnormalities which is supported by several studies with similar findings i.e., showing utricle being the most common site for dislodgement in BPPV patients [11, 17, 23]. Also, we observed a high proportion of patients with cervical VEMP and ocular VEMP abnormalities in contrast to the study by Kim et al. [17] who have reported 17.6% abnormality in cervical VEMP and 21.6% abnormality in ocular VEMP among 112 individuals with BPPV (Posterior, horizontal and mixed). This could be due to the difference in type of participants selected for the present study. Since there are sparse studies post Epley's manoeuvre comparing studies was not possible.

Though we have selected unilateral posterior canal BPPV patients, we observed a significant proportion of patients with reduced amplitude and absent responses in both cervical and ocular VEMP unilaterally and bilaterally. In two individuals with unilateral BPPV post Epley's manoeuvre, we observed bilateral absence of responses in ocular VEMP testing as reported in literature by Nakahara et al. [11] and Kim et al. [17] viewing BPPV as an Bilateral otolith dysfunction. Interestingly we observed two patients (5.55%) with unilateral BPPV presenting with absence of cervical and ocular VEMP in the affected ear which has not been yet reported in literature. This could be due to dislodgement of otoconia or degeneration from utricle and

sacculle into the posterior canal. Also, we have ruled out other causes of vestibular disorders by evaluating these two patients further with Video head impulse test and Videonystagmography.

Conclusion

The present study was aimed to study the frequency of Cervical and ocular VEMP abnormalities in individuals with BPPV after Epley's manoeuvre. We found a significant amount of BPPV showing macular dysfunctions which could be due to unilateral or bilateral otoconia dislodgement from utricle, sacculle and both utricle and sacculle. Though these dysfunctions are attributed to macular degeneration by several studies, detailed investigations using vestibular test battery, supplemented with histopathological findings and follow up over large cohort of BPPV patients may provide insight into the disorder and degenerative process. Since VEMPs are used often clinically used to evaluate otolith organs now-a-days, otolith dysfunctions indicated by VEMPs after Epley's manoeuvre needs to be carefully considered for differential diagnosis and further rehabilitation.

References

1. Dix MR, Hallpike CS (1952) The pathology symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc R Soc Med* 45(6):341–354
2. Cakir BO, Ercan I, Cakir ZA, Civelek S, Sayin I, Turgut S (2006) What is the true incidence of horizontal semicircular canal benign paroxysmal positional vertigo? *Otolaryngol Head Neck Surg* 134(3):451–454. <https://doi.org/10.1016/j.otohns.2005.07.045>
3. Brandt T, Stedding S (1993) Current view of the mechanism of benign paroxysmal positioning vertigo: cupulolithiasis or canalolithiasis? *J Vestib Res* 3(4):373–382

4. Epley JM (1992) The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 107(3):399–404. <https://doi.org/10.1177/019459989210700310>
5. Semont A, Freyss G, Vitte E (1988) Curing the BPPV with a liberatory manoeuvre. *Adv Otorhinolaryngol* 42:290–293
6. López-Escámez J, González-Sánchez M, Salinero J (1999) Meta-analysis of the treatment of benign paroxysmal positional vertigo by Epley's and Semont manoeuvres. *Acta Otorrinolaringol Esp* 50(5):366–370
7. Helminski JO, Zee DS, Janssen I, Hain TC (2010) Effectiveness of particle repositioning manoeuvres in the treatment of benign paroxysmal positional vertigo: a systematic review. *Phys Ther* 90(5):663–678. <https://doi.org/10.2522/ptj.20090071>
8. Welgampola MS, Carey JP (2010) Waiting for the evidence: VEMP testing and the ability to differentiate utricular versus saccular function. *Otolaryngol Head Neck Surg* 143(2):281–283. <https://doi.org/10.1016/j.otohns.2010.05.024>
9. Seo T, Saka N, Ohta S, Sakagami M (2013) Detection of utricular dysfunction using ocular vestibular evoked myogenic potential in patients with benign paroxysmal positional vertigo. *Neurosci Lett* 550:12–16. <https://doi.org/10.1016/j.neulet.2013.06.041>
10. Singh NK, Barman A (2015) Efficacy of ocular vestibular-evoked myogenic potential in identifying posterior semicircular canal benign paroxysmal positional vertigo. *Ear Hear* 36(2):261–268. <https://doi.org/10.1097/AUD.0000000000000097>
11. Nakahara H, Yoshimura E, Tsuda Y, Murofushi T (2013) Damaged utricular function clarified by oVEMP in patients with benign paroxysmal positional vertigo. *Acta Otolaryngol* 133(2):144–149. <https://doi.org/10.3109/00016489.2012.720030>
12. Boleas-Aguirre M, Sánchez-Ferrándiz N, Artieda J, Pérez N (2007) Vestibular evoked myogenic potentials and benign paroxysmal positional vertigo. *Acta Otorrinolaringol Esp* 58(5):173–177
13. Hong SM, Park DC, Yeo SG, Cha CI (2008) Vestibular evoked myogenic potentials in patients with benign paroxysmal positional vertigo involving each semicircular canal. *Am J Otolaryngol* 29(3):184–187. <https://doi.org/10.1016/j.amjoto.2007.07.004>
14. Yang WS, Kim SH, Lee JD, Lee W-S (2008) Clinical significance of vestibular evoked myogenic potentials in benign paroxysmal positional vertigo. *Otol Neurotol* 29(8):1162–1166. <https://doi.org/10.1097/MAO.0b013e31818a0881>
15. Korres S, Gkoritsa E, Giannakakou-Razelou D, Yiotakis I, Riga M, Nikolopoulos TP (2011) Vestibular evoked myogenic potentials in patients with BPPV. *Med Sci Monit* 17(1):CR42–CR47
16. Bremova T, Bayer O, Agrawal Y, Kremmyda O, Brandt T, Teufel J, Strupp M (2013) Ocular VEMPs indicate repositioning of otoconia to the utricle after successful liberatory manoeuvres in benign paroxysmal positioning vertigo. *Acta Otolaryngol* 133(12):1297–1303. <https://doi.org/10.3109/00016489.2013.829922>
17. Kim E-J, Oh S-Y, Kim JS, Yang T-H, Yang S-Y (2015) Persistent otolith dysfunction even after successful repositioning in benign paroxysmal positional vertigo. *J Neurol Sci* 358(1–2):287–293. <https://doi.org/10.1016/j.jns.2015.09.012>
18. Bhattacharyya N, Gubbels SP, Schwartz SR, Edlow JA, El-Kashlan H, Fife T et al (2017) Clinical practice guideline: benign paroxysmal positional vertigo (update). *Otolaryngol Head Neck Surg* 156(3_suppl):S1–S47. <https://doi.org/10.1177/0194599816689667>
19. Colebatch JG, Rosengren SM, Welgampola MS (2016) Vestibular-evoked myogenic potentials. *Handb Clin Neurol* 137:133–155. <https://doi.org/10.1016/B978-0-444-63437-5.00010-8>
20. Rosengren SM, Welgampola MS, Colebatch JG (2010) Vestibular evoked myogenic potentials: past, present and future. *Clin Neurophysiol* 121(5):636–651. <https://doi.org/10.1016/j.clinph.2009.10.016>
21. Todd NPM, Rosengren SM, Aw ST, Colebatch JG (2007) Ocular vestibular evoked myogenic potentials (OVEMPs) produced by air- and bone-conducted sound. *Clin Neurophysiol* 118(2):381–390. <https://doi.org/10.1016/j.clinph.2006.09.025>
22. Colebatch JG, Halmagyi GM, Skuse NF (1994) Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry* 57(2):190–197
23. Xu H, Liang F, Chen L, Song X, Tong MCF, Thong JF et al (2016) Evaluation of the utricular and saccular function using oVEMPs and cVEMPs in BPPV patients. *J Otolaryngol Head Neck Surg* 45:12

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