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Original Article

Ear, Nose and Throat Section

The Clinical Utility of Vestibular Evoked Myogenic Potentials in Patients of Benign Paroxysmal Positional Vertigo

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ABSTRACT

Context: Vestibular Evoked Myogenic Potentials (VEMP) is an emerging tool to diagnose Benign Paroxysmal Positional Vertigo (BPPV). The clinical utility of VEMP has been reported only in small accord in Indian literature.

Aim: To study the latency and amplitude of VEMP in patients with BPPV and compare it with that of normal subjects.

Settings and Design: The study included two groups. Group one (control group) were 18 normal subjects. Group two (test group) were 15 subjects with unilateral BPPV.

Materials and Methods: Those subjects who fulfilled the selection criteria based on case history and audiological assessment were taken for the VEMP recording. The VEMP response consist of positive and negative successive waves (pI-nI), with latency values in adults about 13 and 23 milliseconds respectively.

Statistical Analysis: Data was analysed using Statistical Package for Social Sciences (SPSS) version 12 (Chicago, IL, USA). Unpaired t-test was employed to measure the statistical difference between control group and test group.

Results: The difference in n23 and the peak to peak amplitude between the ipsilateral and contralateral ears of the test group were statistically significant, whereas such a difference in the p13 latency turned out to be statistically insignificant. It should be noted that, out of 15 patients in the test group, five patients showed only artifact tracer recordings in both the ears which is considered as no response. The heterogeneity of the results extended from absence of VEMP to prolongation of both p13, n23; prolongation of p13 alone; and even side to side variations.

Conclusion: Absent response from the ipsilateral ear, prolonged latency of n23 and decreased peak to peak amplitude (p13, n23), indicates the disease pathology. However, large sample size is required to draw further conclusions and to consolidate the usage of VEMP in the diagnosis of BPPV.

Keywords: Amplitude, Latency, n23 p13

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) causes sudden vertigo due to abrupt positional changes of head. There is a wide spectrum of severity. Mild symptoms are inconsistent positional vertigo. Moderate symptoms are frequent positional attacks with disequilibrium between. When severe, vertigo is provoked by most head movements, giving an impression of continuous vertigo. The symptoms can last for days, weeks, months, or years, or be recurrent over many years [1]. Vestibular evoked myogenic potentials (VEMP) have surfaced a new interest in the diagnosis of BPPV [2,3]. It is an emerging tool for those patients who find it difficult to undergo the classical Dix Hall Pike maneuver on the grounds of spinal issues. The clinical utility of VEMP has been reported only in small accord in Indian literature. This study is an earnest attempt to tap the VEMP as a potential diagnostic tool in BPPV patients.

OBJECTIVES

The objectives were to study the latency and amplitude of vestibular evoked myogenic potentials in patients with Benign Paroxysmal Positional Vertigo and compare it with that of normal subjects.

MATERIALS AND METHODS

This descriptive study was conducted in the Department of Otorhinolaryngology, JIPMER, between January 2011 and February 2012. The study included two groups. Group one (control group) were eighteen normal subjects who had no conductive or sensorineural hearing loss and no known history of vestibular disorders. Group two (test group) were 15 subjects with unilateral benign paroxysmal positional vertigo. Test group were clinically diagnosed as having typical nystagmus for posterior canal benign paroxysmal positional vertigo in Dix Hallpike maneuver and ruled out any hearing loss based on audiometric and results. Participants having any symptoms of otological and neurological disorders and bilateral benign Paroxysmal Positional Vertigo were excluded from the test group. The retrocochlear pathology was ruled out by administering auditory brainstem response on all the participants. Patients and controls with a clinical examination suggesting severe systemic diseases or pathologic conditions of the central nervous system were excluded from the study.

VEMP was recorded in a sound treated room. The ambient noise levels were within permissible limits as per ANSI S3.1 (1991).A calibrated diagnostic audiometer GSI 61 were used to estimate the pure tone threshold, speech identification score and uncomfortable loudness level for speech for all the subjects. A calibrated immitence meter (GSI Tympstar) was used for tympanometry and reflexometry. GSI Audera was used for testing vestibular evoked myogenic potentials. ER3A insert earphone was used to deliver the stimulus.

Those subjects who fulfilled the selection criteria based on case history and audiological assessment were taken for the VEMP recording. Subjects were seated upright on reclining chair. Neck was turned opposite to the side of the test ear to activate the sternocleidomastoid muscle unilaterally. Participants were instructed not to move their head and neck while VEMP recording and also to fix their gaze in front to control eye movement. Neuprep gel was applied to achieve low impedance (2-5kohm). The surface electrodes were at the midpoint of the sternocleidomastoid muscle (noninverting), sternoclavicular junction (inverting electrode) and forehead (ground electrode). 500 Hz tone burst at 105 dB nHL was used as stimuli. The VEMP response included a positivity at 13ms (p13) and followed by a negativity at 23ms (n23). Consecutive runs

were performed to confirm reproducibility of peaks p13 and n23 and thus VEMP responses were considered to be present. When reproducibility of biphasic waveform was lacking, VEMP responses were considered absent. Unpaired t-test was employed to measure the statistical difference between control group and clinical group. The study was approved by the institutional ethical board.

VEMP settings used in our study

Stimulus- 500 Hz

Stimulus level - (105dBnHL)

Stimulus potency – (rarefaction)

Repetition rate – (5.09/s)

Sweeps –(150 or 200)

High filter – high/band pass filter 10Hz HP@ 6 -12dB/ oct band width)

Low filter – 750Hz LP > 40 dB/Octave

Sensitivity – 150 NV

RESULTS

The mean age of the test group was 41.3 years with 5 males and 10 females. The mean values of p13 and n23 latencies and peak to peak amplitudes are shown in [Table/Fig-1] for the test subjects, for both contralateral ears and test ears. We found that the difference in n23 and the peak to peak amplitude between the ipsilateral and contralateral ears of the test group were statistically significant, whereas such a difference in the p13 latency turned out to be statistically insignificant. It should be noted that, out of 15 patients in the test group, five patients showed only artifact tracer recordings in both the ears which is considered as no response. Also, one patient in the test group showed no response in ipsilateral ear but the response in the contralateral ear was normal. The mean values were calculated excluding these parameters.

The mean age of the control group was 42.7 years containing 9 males and 9 females (total of 18 subjects).The mean values of latency of p13, n23 in millisec and peak to peak amplitude of p13 to n23 in millivolts are shown in [Table/Fig-2]. We also analysed the contralateral ear of test group BPPV patients with the values of the control group. This did not reveal any statistical significance.

	Ipsilateral ear mean	95% CI Ipsilateral ear	Contralateral ear mean	95% CI Contralateral ear	p-value
p13millisec	18.3 (3.9)	15.3, 21.3	16.3(1.5)	15.2, 17.5	0.26
n23millisec	28.1(2.5)	26.2, 30.0	25.1 (1.7)	23.8, 26.5	0.004
Amplitude millivolt	26.1 (5.0)	22.2, 29.9	30.2 (4.8)	26.5, 33.8	0.07

[Table/Fig-1]: Mean p13 and n23 latencies and peak to peak amplitudes of test group (n=9/15)

	Right ear(18)	95% CI Right ear	Left ear(18)	95% CI Left ear	p-value
p13 (millisec)	15.9 (1.9)	15.0, 16.9	16.8 (1.2)	16.1, 17.5	0.16
n23 (millisec)	24.7 (2.9)	23.3, 26.2	25.0 (1.8)	24.1, 25.9	0.76
Amplitude (millivolt)	29.7 (4.9)	27.3, 32.1	30.2 (5.4)	27.5, 32.9	0.74

[Table/Fig-2]: Mean p13 and n23 latencies and peak to peak amplitudes of control group (n=18)

It is in particular to note that, when we looked into the individual patient data for a diagnosis with VEMP, the results were not consistent to reflect a clear cut diagnosis. The heterogeneity of the results extended from absence of VEMP to prolongation of both p13, n23; prolongation of p13 alone; and even side to side variations.

DISCUSSION

Vestibular-dependent myogenic responses to intense sound were first described by Bickford et al., [2]. "Vestibular-evoked myogenic potentials" (VEMP) was widely used since the work of Halmagyi and Colebatch [3]. The gold standard vestibular function test is Electronystagmography (ENG) [4]. The caloric test induces vertigo and assesses only the horizontal semicircular canal function [5]. Compared to the ENG, VEMP testing is easier to perform, less complicated for interpretation, induces less dizziness or nausea, and is more tolerable to patients [6].The pathway of VEMP circuit starts with the sound stimulating the saccule, which activates the inferior vestibular nerve, lateral vestibular nucleus, 11th nerve nucleus, and then ending with the sternocleidomastoid muscle (mostly ipsilaterally) through the vestibulospinal tract [7]. The sternocleidomastoid muscle has more homogenous responses than other muscles [8-10]. Gacek [11] reported loss of vestibular ganglion cells in the inferior vestibular nerve, ganglion degeneration in the saccular nerve in the temporal bones of the BPPV patients. Shucknedt [12] proposed the theory of "cupulolithiasis" and Hall [13] proposed the concept of "canalolithiasis", explain the pathogenesis of BPPV. Currently, Epley's canalith theory explains most of the features of BPPV [14,15]. The Dix-Hallpike positional test is the gold standard for diagnosing posterior canal BPPV. However, patients experiencing lower back, cervical spinal problems or for the elderly, rotation and extension of the neck during the positioning may require a caution [16].VEMP as a diagnostic tool has been proposed for BPPV in such patients. There has been a recent interest in tapping the potential of this investigation in various vestibular disorders.

p13 which is the most used parameter in the VEMP analysis [17,18] though showed a prolongation in our test group patients, failed to achieve statistical significance between the test ear and normal ear. The difference in n23 and the peak to peak amplitude between the ipsilateral and contralateral ears were statistically significant in BPPV patients between the test and contralateral ear. But a meaningful conclusion may not derive because of the inconsistent absent responses confounding in five out of fifteen patients. It should be noted that, out of 15 patients in the test group, five patients showed only artifact tracer recordings in both the ears which was considered as no response. Some studies have also reported that the absence of response is more common in the affected side (10/19) than in the healthy side (5/19) in their study population. They also reported no response was detected in either of the ears in 5 subjects [19].The possible explanation could be that our study used 105dB of tone burst stimuli and in an absent response situation we should have delivered a different stimulus type in a probability of eliciting the response from the participants. Mean amplitude of p13-n23 is lower in our study than those described by some authors [10,20]. Differences of amplitude values probably resulted from the other technique of examination, especially greater tonic activity of muscles. But there are only a few studies analysing the amplitude difference on BPPV due to variations attributed to individual subjects, laboratory and SCM contraction. So, it is difficult to directly apply those amplitude-related values on clinical decisions [21]. Hence, an abnormal VEMP obtained will nonspecifically suggest a vestibular pathology, even may not be able to locate the side of pathology, severity or regression [22].

CONCLUSION

It is inferred that either as absent response from the ipsilateral ear, prolonged latency of n23 and decreased peak to peak amplitude (p13, n23), indicates the disease pathology. Contra-lateral ear will show either an absent or normal response in patients with unilateral BPPV. However, large sample size with subset matched control analysis and post treatment long term follow up are required to draw further conclusions and to consolidate the usage of VEMP in the diagnosis of BPPV.

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