

ORIGINAL ARTICLE

Air Conduction Ocular Vestibular-Evoked Myogenic Potentials (AC oVEMPs): Diagnostic Correlates in Peripheral Vestibular Disorders

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Background: Ocular vestibular-evoked myogenic potentials (oVEMPs) are myogenic potentials that can provide another diagnostic tool for assessing otoliths function. While many studies concluded that (bone conduction) BC oVEMPs predominantly represent utricular function, no conclusions can be made yet about the (air conduction) AC oVEMPs Objectives: This study aimed to compare the findings of AC oVEMPs versus AC cVEMPs in different peripheral vestibular disorders.

Subjects and Methods: Twenty healthy subjects without previous ear disorders served as a control for AC oVEMPs testing. The study group consisted of (25) patients having unilateral peripheral vestibular deficit. All the study group underwent AC oVEMPs and AC cVEMPs testing

Results: AC oVEMPs were 100% identifiable in all normal subjects (40 ears). Among patients with vestibular neuritis (no.=11), abnormal oVEMPs were found in (6/7) '85%' of patients who had unilateral caloric weakness. Only two patients had additional cVEMPs abnormalities. All the patients with endolymphatic hydrops (no.=7) had both oVEMPs and cVEMPs abnormalities regardless presence or absence of caloric weakness. While only one patient with benign paroxysmal positional vertigo had abnormal oVEMPs and another one had abnormal cVEMPs. Abnormal oVEMPs responses were in the form of reduced amplitude and /or shift in the absolute latencies or absent response, recorded from the contralateral side of lesion .

Conclusions: These findings might suggest that AC oVEMPs were correlated with superior vestibular nerve or utricular lesion as an extensive peripheral end organ lesion. AC oVEMPs should be complementary to cVEMP in the clinical diagnosis of vestibular disorders

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Introduction

The peripheral vestibular system is composed of two types of sensory organs, the semicircular canals and the otoliths. The semicircular canals sense rotational head movement or angular acceleration while the otolith organs respond to linear acceleration, head tilt and gravity. Although lateral semicircular canal function can be tested with caloric irrigation or rotational stimuli, safe simple tests of otolith function are not common. Recently with the development of the VEMPs (vestibular evoked myogenic potentials) first described by Colebatch and Halmagyi ^[1] and Colebatch et al. ^[2], cervical VEMPs (cVEMPs) have become a standard clinical test of otolith (predominantly saccular) function, with documented diagnostic utility in various vestibular and central nervous system disorders ^[3].

In recent years, short latency myogenic potentials, oVEMPs (ocular VEMPs), produced by synchronous activity in the extraocular muscles in response to different stimuli including sound. It has been reported that oVEMPs can provide another diagnostic tool for assessing the vestibulo-ocular reflex. Analogous to cVEMPs, stimulation by loud sound can be via air or bone conduction. In human, studies demonstrated that bone conduction (BC) vibration stimulation efficiently evokes both oVEMPs ^[4] and cVEMPs ^[5]. The prevalence of oVEMPs responses to air conduction stimuli near to threshold is less than that of cVEMPs . However , oVEMPs are excitatory responses that may be elicited more easily than cVEMPs even in elderly or disabled subjects who fail sustained neck contraction ^[6].

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While many studies assumed that air conduction cVEMPs and oVEMPs predominantly represent saccular stimulation and bone vibration activates both saccular and utricular afferents, the populations of primary afferents contributing to the vestibulo-collic and vestibulo-ocular reflex pathways may not be identical. Therefore cVEMPs and oVEMPs in peripheral vestibular lesions are not necessarily expected to yield identical results [6,3]. Despite few animal studies concluded that BC oVEMPs predominantly represent utricular function, no conclusions yet can be made about the origin of AC oVEMPs [3].

Hence this research aimed to compare findings of AC oVEMPs versus AC cVEMPs in different peripheral vestibular disorders trying to identify neural origin of AC oVEMPs.

Materials and Methods

Subjects

I- Control group: Twenty healthy subjects (10 males and 10 females), aged from 22 to 55 years (mean 36.4 ± 11 years), with no history of ear disorders, dizziness or sense of imbalance.

They served as control for AC oVEMPs testing.

II-Study group : Twenty five patients (12 males and 13 females), aged from 20 to 68 years (mean 44.1 ± 14.8 years) were enrolled in this study. All patients included in this study had unilateral peripheral vestibular deficit. Their diagnosis was based on full history taking, otological examination and documented by Videonystagmography (VNG).

For all subjects normal middle ear function was mandatory inclusion criteria. Any case with conductive hearing loss was excluded.

All subjects of control group underwent AC oVEMPs, while the study group underwent both AC oVEMPs and AC cVEMPs using Evoked potential measuring system Neuropack, four mini (Nihon Koden) MEB-530 4K . All patients were examined one to two months after last attack of vertigo.

The procedures followed were ethically approved by Audiology and ENT Department Board. All subjects participated in this study were informed and they gave their consent prior to study.

AC oVEMPs test

The subject was in a sitting position. Surface potentials predominantly electromyograph (EMG) were recorded with three Ag/AgCl electrodes. The active electrode was placed on the face just inferior and at center of lower eye lid. The reference electrode was positioned at the chin and one ground electrode was on the forehead [7]. The electrode impedance was kept under 5 k Ω . During recording, the subject was instructed to look upward at a small fixed target >2 m from the eyes. The vertical eye position was at angle of approximately 30–35° above horizontal.

The EMG signals were amplified and band pass-filtered between 10 and 1000 Hz. Acoustic stimuli were delivered at 95dBnHL using head phone (Dynamic receiver, type DR-531, Elega. Acous, Japan) Short tone bursts (500 Hz, rise/fall time = 1 ms, plateau time = 2 ms) with rarefaction polarity were delivered through the headphone. Monaural stimulation with contralateral eye recording was employed for recording oVEMPs. The stimulation rate was 5 Hz, with the analysis time for each response of 50 ms, and 70-100 responses were averaged for each run. The initial negative-positive biphasic waveform comprised peaks nI and pI. Two runs were performed to confirm the reproducibility of peaks nI and pI. Conversely, oVEMPs were termed absent when the biphasic waveform was lacking. The latencies of peaks nI and pI, amplitude nI–pI, and interaural amplitude difference (IAD) ratio were measured. The latter was defined as the difference of the amplitude nI–pI on the right and left ears divided by the sum of amplitude nI–pI of both

AC cVEMPs test

The active electrode was placed on the on symmetrical sites at midpoints of each sternocleidomastoid (SCM) muscles, with a reference electrode on the suprasternal notch, and a ground electrode on the forehead. EMG signals were amplified, band pass-filtered between 10 and 1000 Hz and monitored to maintain background muscle activity. Acoustic stimuli were the same as oVEMPs test and 70-100 responses were averaged for each run, while the subject was sitting with the head rotating sideways toward one shoulder to activate the SCM muscle [10]. Monaural acoustic stimulation with ipsilateral recording was employed for recording cVEMPs. n13 –p13 latencies and IAD. They were

classified to normal, abnormal according to our lab normative data.

Statistical analysis: All the data were collected and analyzed using an IBM computer statistical package for social science (SPSS) program version ^[13] by expert statistician. Mean, Standard Deviation (SD) were calculated. Paired t test was used for two dependent means, Student "t" test was used for two independent means with normal distribution while comparison between two independent groups for non-parametric data was by using Wilcoxon. Rank Sum test. Ranked Sperman correlation test was used for non-parametric data .P< 0.05 was considered a significant.

Results

I. control group:

There was no statistical difference as regards age and gender distribution between control group and study group (P > 0.05) .

AC oVEMPs were 100% identifiable in all normal subjects (40 ears) (fig. 1). The first negative peak n1 latency mean was at 10.05(±0.86) with range (9- 13) ms. The second positive peak (p1) mean was at 14.43 (±1.01) with range (12.2-17.1) ms. n1-p1 amplitude mean was 6.53 (±1.86) with range (4.0-12.2) µv and. Interaural amplitude difference (IAD) ratio ranged from 0.9-24.7% with mean of 8.09 % (±8.01) .

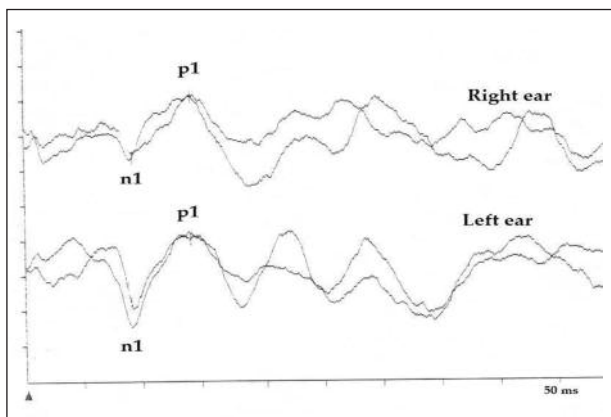


Figure 1. Normal AC oVEMPs response

There was no statistically difference between right and left ears in the control group (table 1).

Table 1. Comparison between right and left side in normal subjects(no.=20)

	Right Mean ±SD	Left Mean ±SD	P value
N1 latency (ms)	9.92(±0.75)	10.17±0.87	>0.05
P1 latency (ms)	14.39(±0.96)	13.97±1.0	>0.05
N1-p1 amp (µv)	6.41(±1.84)	6.65±1.92	>0.05

II. study group

Among 25 patients with unilateral peripheral vestibular disorders;

eleven patients had vestibular neuritis (VN) aged 20-64 years; mean age 42 ± 16.2. All VN patients reported primary attack of vertigo lasted for several hours, accompanied with vegetative symptoms (no hearing loss, or tinnitus) with gradual recovery within days to one week. They had recurrence of their vertiginous attacks (but less in severity),or brief sense of rotation and imbalance with rapid head movement .

Seven patients had endo-lymphatic hydrops (ELH) aged 32-68 years; mean age 46 ± 12.6. (three patients with Meniere's disease, stages 2&3 and four patients had unilateral profound sensorineural hearing loss with symptoms of delayed endolymphatic hydrops). Patients were considered as having definite Meniere's disease according to the AAO-HNS committee on hearing and equilibrium ^[8, 9]. The remaining seven patients had benign paroxysmal positional vertigo (BPPV) aged 27-68 years; mean 44.2 ± 16.2. They reported brief verigionous attacks (last for few minutes) accompanied with vegetative symptoms when truning to one side in bed, leaning downwards or looking upwards.

Unilateral weakness was encountered in ^[7,11] "63.6%" of patients with VN and the remaining ^[4,11] "36.3%" of patients had symmetrical caloric response with significant directional preponderance (≥ 25%). Unilateral weakness was also encountered in ^[4,7] "57.1%" of patients with ELH. All patients with BPPV had normal symmetrical caloric response with positive Dix-Hallpike maneuver on the affected side.

1-AC oVEMP test results:

Among patients with VN (no.=11) abnormal oVEMP responses were found in seven patients, the majority ^[6,7] "85%" of these patients had unilateral caloric weakness and in one patient who had symmetrical caloric response. All the seven patients with ELH had abnormal oVEMP responses regardless presence or absence of caloric weakness. While only one patient with BPPV had abnormal response.

Abnormal responses were in the form of absent response (fig.2), reduced amplitude and /or shift in the absolute latencies (fig. 3 & 4) recorded on the contralateral side of affected ear. However, bilateral affection was present in ^[2,7] of affected patients with VN and ^[3,7] of affected patients with ELH .

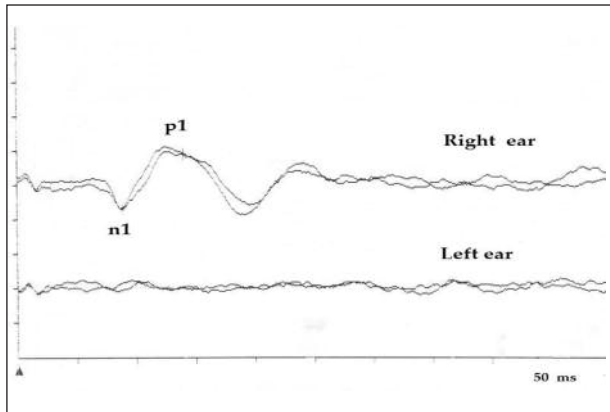


Figure 2. Absent AC oVEMPs in the left ear

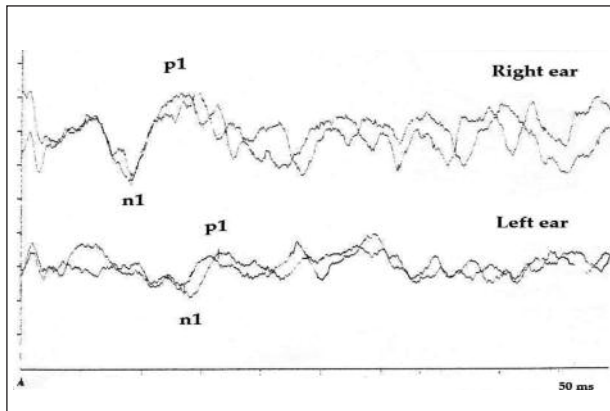


Figure 3. AC oVEMPs showing delayed latency and decreased amplitude in the left ear

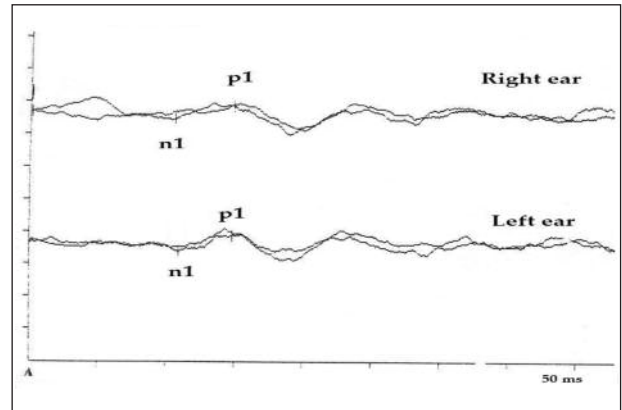


Figure 4. AC oVEMPs showing decreased amplitude bilaterally

According to side of lesion we divided ears of patients into affected ears (25 ears, 5 of them had absent response) and unaffected ears (25 ears). The mean and standard deviation of affected side and unaffected side are shown in table ^[2]. Comparison with normal ears (40 ears) revealed that there was a very high statistical significant difference between affected side of patients and normal ears regarding all oVEMPs parameters (n1 and p1 latencies and n1-p1 amplitude). Also there was a statistical significance difference between non affected side of patients and normal ears regarding n1-p1 amplitude. (Table 2) Comparison between affected ears and unaffected ears within all patients revealed significant difference between n1 latencies.

Mean and SD of the affected ears for each subgroup revealed that ELH subgroup had more delayed n1 and p1 latencies and the lowest n1-p1 amplitude (table 3).

Table 2. Comparison between oVEMP of the control group and the study group

	Control group (no.=40 ears)	Study group			
		Unaffected ears (no.=25)		Affected ears (no.=20)	
	Mean \pm SD	Mean \pm SD	P value		P value
n1 latency (ms)	10.05(\pm 0.86)	10.22(\pm 1.44)	.47	11.53(\pm 2.42)	0.01**
p1 latency (ms)	14.43 (\pm 1.01)	14.80(\pm 1.54)	.25	15.77(\pm 2.49)	0.01**
n1-p1 amp (μ v)	6.53(\pm 1.86)	5.23 \pm 2.74	.04*	4.85(\pm 2.31)**	0.00**

Table 3. Mean and SD of oVEMPs parameters of the three subgroups

	VN (no.=7)	ELH(no.=7)	BPPV(no.=11)
	Mean \pm SD	Mean \pm SD	Mean \pm SD
n1 latency(ms)	11.9 (\pm 2.92)	12.87(\pm 1.72)*	10.36(\pm 1.26)
p1 latency(ms)	14.14(\pm 3.24)	16.03(\pm 0.45)*	14.68(\pm 1.29)
n1-p1 amp(μ v)	5.05(\pm 3.04)	3.36(1.90)*	5.21(\pm 0.74)

2-AC cVEMPs test results:

In contrary to oVEMPs results, abnormal cVEMPs responses were found only in ^[2,7] patients having unilateral caloric weakness of VN patients. All patients with ELH had abnormal cVEMPs responses regardless the presence or absence of caloric weakness. While only one patient with BPPV had abnormal response

Abnormal responses were considered when absolute latencies lies outside two SD of our lab normal values (p13 latency mean 12.3 ± 1.2 , n23 latency 21 ± 1.7 ms). Abnormal responses were in the form of absent response, or shift in the absolute latencies on the

ipsilateral recording side of affected ear. Bilateral affection was present in ^[2,7] patients with VN and ^[3,7] patients with ELH.

The affected side p13 latency mean was $14.16(\pm 1.16)$ ms, n23 mean was at $22.72 (\pm 1.82)$ and p13-n23 amplitude mean was $44.24(\pm 19.17) \mu$ v. Comparison between affected ears and non-affected ears within all patients revealed no statistical significant difference regarding all the cVEMPs parameters (p13, n23 latencies and p13-n23 amplitude)

Abnormalities in oVEMPs and cVEMPs that were encountered in the three subgroups of patients are summarized in table (4). This table shows that in VN subgroups the majority (85%) of patients with unilateral weakness had oVEMPs abnormality, while only two patients had additional cVEMPs abnormality. In contrast to VN subgroup, all endolymphatic hydrops subgroup had both oVEMPs and cVEMPs abnormalities regardless the presence or absence of caloric weakness. BPPV subgroup showed only one patient with affected oVEMP and another patient with affected cVEMPs.

Table 4. Distribution of affected VEMPs according to caloric weakness in each subgroup

Subgroup	Vestibular neuritis (no.=11 patients)		Endolymphatic hydrops (no.=7 patients)		BPPV (no.=7 patients)	
	Normal	UW	Normal	UW	Normal	UW
	4(36.3%)	7(63.6%)	3(42.8%)	4(57.1%)	7(100%)	-
oVEMP	1(25%)	6(85%)	3(100%)	4(100%)	1(14.2%)	-
cVEMP	-	2(28%)	3(100%)	4(100%)	1(14.2%)	-

Discussion

I. Normal group

AC oVEMPs were successfully recorded in all healthy subjects (40 ears) in the control group when either the right or left ear was stimulated by air conduction mode. Two components were identified in oVEMP response: biphasic negative-positive wave n1-p1 (fig1). This agrees with recent reports that both air and bone conduction can activate otoliths using intense sound stimuli and could be recorded in subjects with intact vestibular function, but absent in patients with vestibulopathy ^[11].

The latencies of n1, p1 and n1-p1 amplitude were comparable to those reported by Welgampola et al. ^[12] & Wang et al. ^[6] when using monaural air conduction mode of stimulation. No statistically significant difference was detected between the right and left side recording of n1,p1 latencies and n1-p1 amplitude (Table 1).

oVEMPs were 100% identifiable in our control group this agrees also with Wang et al. ^[6] who found monaural –oVEMPs were elicited in 95% "19 of 20" healthy subjects examined. Also this coincides with the consistency of n1 response and its repeatability within the same subjects ^[11,4]. The mean of Inter-aural amplitude

difference (IAD) of n1-p1 was 8.09 % (± 8.01) that was comparable to values by Iwasaki et al. ^[11].

II. Study group

In an attempt to investigate the diagnostic value of AC oVEMPs, different peripheral vestibular disorders were included in this study (Table 4). Vestibular neuritis (VN), Endolymphatic hydrops (ELH) and benign paroxysmal vertigo (BPPV).

In the present study, the most frequent abnormal oVEMPs responses were in the form of reduced amplitude and /or shift in the absolute latencies followed by absent response, recorded on the contralateral side of affected ear. However, bilateral affection was present in 2/7 of affected patients with VN and 3/7 of affected patients with ELH.

These abnormalities were evident by the very high statistical significant difference ($P \leq 0.01$) between the normal ears and the affected ears of the study group at all oVEMPs parameters (n1 and p1 latencies and n1-p1 amplitude). Moreover, n1 latency was statically significant between the affected and the non affected ears. This might reflect that n1 latency is an important parameter to identify unilateral otolith dysfunction. This agrees with Iwasaki et al. ^[11] who stated that in patients with unilateral vestibular lesions n10 (n1) is mainly identified contralateral to the healthy side.

On the other hand, there were statistical significant difference between the normal ears and the non affected side of patients difference regarding n1-p1 amplitude. This might be explained by the presence of bilateral affection in some patients.

To identify the role of AC oVEMPs and AC cVEMPs in the diagnosis, distribution of vestibular findings in each subgroup of patients were analyzed

Findings in vestibular neuropathy subgroup:

Previous reports pointed to the superior vestibular nerve as the main site of lesion in vestibular neuritis disorder, less often both superior and inferior vestibular nerves and rarely only inferior vestibular nerve ^[13]. In the current study, unilateral caloric weakness was encountered in ^[7, 11] 63.3% of VN patients, reflecting superior vestibular nerve involvement. The majority ^[6, 7] "85%" of these patients had oVEMPs abnormality (contralateral to the affected side), among them two patients had additional cVEMPs abnormality (Table 4).

This agrees with the Iwasaki et al. ^[14] who has shown that patients with vestibular neuritis affecting superior vestibular nerve had mean n10 amplitude asymmetry ratio similar to that found in patients with total post surgical unilateral vestibular loss, suggesting that superior vestibular nerve fibers are particularly important in the genesis of the response.

While Iwasaki et al. ^[14] have proposed these findings to bone conduction stimuli, Similar findings to air conduction stimuli were suggested by Murofushi et al. ^[15]. They found absence of AC oVEMPs response in a case with vestibular schwannoma from the affected side, while the AC cVEMPs were normal. Thus they suggested that air conduction oVEMPs could also reflect functions of populations of the peripheral vestibular system other than those reflected by AC cVEMPs perhaps, predominantly utricular afferent. Reconsideration of the interpretation of n10 of the AC oVEMP as being much more likely that AC activates utricular neurons and they cause the n10 of the contralateral oVEMP was suggested by Curthoys et al. ^[16]

In the current study, findings in vestibular neuritis patients are in agreement with previous studies that suggest that neural pathway of oVEMPs is mainly transferred via superior vestibular nerve. Also, air conduction stimuli were successful to detect unilateral vestibular deficit comparable to the previous reports using bone vibration. The additional cVEMPs affection which was only in two patients could reflect extensive lesion involving the inferior division of vestibular nerve as well

Findings in endolymphatic subgroup:

There is no single test that makes the diagnosis of Meniere's disease. Rather, diagnosis is made most importantly by a complete history supported by other tests. According to Okuno and Sando ^[17] severe hydrops was observed in the saccule most frequently in their histopathological study of the temporal bone. Therefore, a high incidence of abnormal vestibular evoked myogenic potentials (VEMPs) in Meniere's disease is expected.

In the current study, in contrast to vestibular neuritis, all endolymphatic hydrops subgroup had abnormal cVEMPs response (mainly absent response) constituted the most robust indication of saccular and/or inferior vestibular nerve lesion. Their VNG reflected lateral semicircular canals affection in ^[4, 7]

"57%" of patients as detected by unilateral caloric weakness. These cVEMPs abnormalities were accompanied by oVEMPs affection regardless presence or absence of unilateral weakness.

This agrees with Taylor et al.^[18] who found reduced oVEMPs from diseased ear and their asymmetry were comparable to those obtained with cVEMPs. Also, they found predominance of abnormalities in oVEMPs and cVEMPs responses to AC sound stimuli compared to other stimuli (BC and tapping). They concluded that AC oVEMPs abnormalities were the characteristic for Meniere's disease

In the current study, bilateral affection was in 3/7 of affected patients with ELH. This agrees with previous reports showing that unaffected ears of unilateral Meniere's subjects might show abnormal changes (elevated VEMP thresholds), though of lesser degree^[19]. This finding could be attributed to occult saccular hydrops in the asymptomatic ear or binaural interactions in the vestibular evoked myogenic potential otolith-cervical reflex arc.

According to Young et al.^[20] affection of saccule is more related to late stages of Meniere's disease, so all patients of this subgroup had saccular hydrops. In this subgroup, despite of their few number, significant AC oVEMPs abnormalities were correlated with cVEMPs abnormalities in all patients. This might be interpreted as that AC oVEMPs might share same afferent origin of cVEMPs (saccule and or inferior vestibular nerve). However, this disagrees with the assumption made in the view of the abnormalities we found in VN patients. Similar contradictory results were encountered by Chou et al.^[21]. Moreover, Chairwoman et al.^[22] found that oVEMPs were frequently more absent than cVEMPs.

So there might be other explanation for this possible co-affection of both oVEMPs and cVEMPs in this subgroup of patients. It might be related to an extensive otolith end organs lesion, assuming that endolymphatic hydrops might affect both saccular and utricular receptors in late stages. The oVEMPs values of affected ears in ELH subgroup had the most delayed latencies and most reduced amplitude. This might further assume that severity of lesion was more when utricular end organ was affected than when the superior vestibular nerve was affected in VN subgroups, however further studies are needed to

assess oVEMPs in larger group of patients with different stages of Meniere's disease

Findings in Benign paroxysmal positional vertigo (BPPV) subgroup:

The BPPV subgroup showed only one patient with affected oVEMPs and another patient with affected cVEMPs (abnormality was in the form of delayed latencies). BPPV is known to be most often idiopathic in nature though it has been known to occur in patients following head injury, vascular injury, Meniere's disease, acoustic neuroma and viral neuronitis^[23,24].

In recent report, Yang et al.^[25] indicated that while most patients have normal AC cVEMP, some amplitude attenuation and/or latency prolongation. Particularly those require multiple canal repositioning. Degenerative changes have been suggested which may occur in utricle or saccule to precipitate to BPPV. This might explain findings in those two patients.

The use of VEMPs test in patients with BPPV is mainly to assess the extent of lesion rather to diagnose the condition itself. It may explain the limited improvement observed in some patients^[24]. So, it could have an important role in rehabilitation planning especially if patients did not improve after repositioning maneuvers.

To summarize oVEMP abnormalities were consistently found in subgroup of VN who had caloric weakness, this is strongly suggestive that superior vestibular nerve is the main origin of oVEMPs pathway. However, Different findings were encountered in ELH. For explanation of such findings, other factors should be taken in consideration as the anatomical proximity of saccule and utricle and the nature of endolymphatic hydrops pathology in the late stages. So extensive lesion that might involve both otoliths organs might be considered in this subgroup of patients.

Conclusion

Air conduction oVEMPs is an easy feasible test, that could identify unilateral peripheral vestibular lesions same as bone conduction oVEMPs. It should be complementary to cVEMPs in the diagnosis of peripheral vestibular disorders. Yet, further studies needed to evaluate diagnostic role of AC oVEMPs in different stages of Meniere's disease and central vestibular disorders.

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