Research Article

Toneburst Vestibular Evoked Myogenic Potential (VEMP) in Patients with Ménière's Disease

Mohamed M. Elbadry, **B H Aly, H S Mahmoud, Dalia F. Mohammed**
Department of Otolaryngology, Audiology Unit, Minia University Egypt

Abstract
Objective: In this study, we test the hypothesis that the cochlear hydrops of Meniere's syndrome leads to alteration in saccular motion that change the dynamics of the vestibular evoked myogenic potential. **Study Design:** Prospective cohort study. **Setting:** A university hospital. **Subjects:** Twenty normal adult volunteers and ٢٥ adult patients with unilateral Meniere's disease by American Academy of Otolaryngology-Head and Neck surgery diagnostic criteria. **Intervention:** All subjects underwent vestibular evoked myogenic potential testing using ipsilateral broadband click and short tone burst stimuli at ٥٠٠ and ١٠٠٠ Hz. **Main Outcome Measure:** P١-N١ corrected amplitude, P١ and N١ latency, ١٠٠٠/٥٠٠ FPA ratio, AR, IAL difference, **Results**: Normal subjects show a frequency dependent vestibular evoked myogenic potential with best response (frequency tuning) at ٥٠٠ Hz. Compared with normal subjects and unaffected ears of Meniere's subjects. Affected Meniere's ears showed frequency shift and there was less tuning apparent at ٥٠٠ Hz. Unaffected ears of Meniere's subjects also showed affected frequency tuning. Both Meniere's ear and other ear FPA ratio was larger compared to control ears while no statistically significant difference between Meniere's ear and other ears. **Conclusion:** Meniere's ears display alteration in vestibular evoked myogenic potential tuning, supporting our hypothesis of altered saccular motion mechanics arising from hydropic distention. Unaffected ears of unilateral Meniere's subjects show similar changes but to lesser degree. This finding may be because of occult saccular hydrops in the asymptomatic ear or binaural interaction in the vestibular evoked myogenic potential otolith cervical reflex arc.
Key Words: Toneburst, Vestibular, Evoked, Myogenic Potential

Introduction
The Vestibular evoked myogenic potential (VEMP) is a large, short latency, myogenic potential produced by contraction of the sternocleidomastoid (SCM) muscle in response to loud acoustic stimulus[١]. Studies have shown that ٩٠-١٠٠ dB nHL click or toneburst stimuli produce optimal VEMP recording. However, Low frequency tone burst stimuli give more pronounced results than click or other frequencies[٢]. The VEMP has been applied clinically to evaluate the integrity of the sacculocollic reflex and is widely clinically applicable in many disease as Ménière's disease (MD), superior semicircular canal dehiscence, large vestibular aqueduct syndrome, acoustic neuroma, multiple sclerosis and brainstem stroke[٣].

It has been reported that VEMP may be useful in assessing MD because the saccule, next to the cochlea is the second most frequent site of hydrops formation[٤]. According to stage of MD. VEMP may take different forms either enhanced, decreased amplitude or absent. Furthermore, Rauch et al.,[٥] reported the alteration of tuning characteristics of VEMP in MD patients. They reported that, in healthy subject, the largest amplitude and the lowest threshold were obtained to ٥٠٠ Hz toneburst, whereas patients with MD showed less tuning at ٥٠٠ Hz and shift of the best frequency to ١٠٠٠ Hz. This result was based on their findings that the ratio of the amplitude of P١-N١ of VEMP of ٥٠٠ Hz stimulus to that of ١٠٠٠ Hz was significantly less in Ménière's patients than

in normal subjects. Rauch et al attributed this shift to change of resonant frequency in the saccule and suggest that amplitude relation of VEMP at ٥٠٠ Hz to ١٠٠٠ Hz

may help in staging, diagnosis of early Ménière's, diagnosis in other ear and confirm diagnosis of probable Ménière's.

The current work was designed to study VEMP in patients with MD using click and tonebursts with ٥٠٠ and ١٠٠٠ Hz. The specific aims of the current work were:
١- To determine VEMP findings in MD patients and compare the findings with those of healthy control subjects. The specific parameter of interest is the frequency peak amplitude (FPA) ratio, which is ratio of P١-N١ VEMP amplitude at ١٠٠٠ Hz toneburst to P١-N١ VEMP amplitude at ٥٠٠ Hz toneburst.
٢- To determine if VEMP can diagnose the involvement of the other ear in MD patients.

Subjects

The current study included ٢ groups. Group I was the group of Meniere's patients, which included ٢٥ patients with definite MD. group II was the control group, which included ٢٠ healthy volunteers.

I- Patients with definite MD were selected according to the criteria of AAO/HNS [٢] which are

A- Two or more definitive spontaneous episodes of vertigo ٢٠ minutes or more.
B- Audiometrically documented hearing loss on at least one occasion.
C- Tinnitus or aural fullness in the treated ear.
D- Other causes excluded.

II- Subjects in the Control group were selected according to the following criteria

A- No history suggestive for vestibular disorder as vertigo, dizziness, or sense of imbalance.
B-No history suggestive for migraine as migrainous headache, phonophobia and photophobia.
C- Bilateral normal hearing sensitivity.
D- Bilateral normal tympanograms with intact acoustic reflex and no current or history of middle ear disease.
E- Normal cochlear function, as shown from normal DPOAE.
F- No history of medical disorders known to affect hearing as diabetes, hypertension.

Methods

All subjects were subjected to:

I- Full history taking:

History included description of the vertiginous attack, frequency of attack, duration of each attack, condition of the patients in between attacks, degree of disability during attack, associated hearing loss and tinnitus, sense of ear fullness, nausea and vomiting.

II- Otoscopic examination.

III- Audiological evaluation in the form of:

١- ***Immittancemetry*** using Zodiac ٤٠١ immittancemeter (GN Otometrics A/S, Taastrup, Denmark) to measure middle ear pressure and stapedial muscle reflex threshold at frequencies of ٥٠٠, ١٠٠٠, ٢٠٠٠ and ٤٠٠٠ Hz and to exclude middle ear pathologies.
٢- ***Pure tone audiometry*** using Amplaid ٣٠٩ audiometer (Amplus Corp. Boulder, CO, U.S.A.) and sound treated room (amplisilence) to assess hearing sensitivity. Air conduction threshold was obtained for the frequency range ٢٥٠–٨٠٠٠ Hz at single octave intervals using a TDH ٤٩ ear phone (Telephonics Corporation, Farm ingdale, NY, U.S.A.), while bone conduction threshold was obtained for the frequency range ٥٠٠–٤٠٠٠ Hz at single octave intervals using a B٧١ bone vibrator (Radioear, New Eagle, PA, U.S.A.). Speech reception threshold (SRT) and speech discrimination score using bisyllabic and monosyllabic phonetically balanced words respectively.
٣- ***DPOAE*** using the Intelligent Hearing system (IHS) two channel evoked potential recording apparatus with Smart OAE ٤.٥ software (Intelligent Hearing Systems, Miami, FL, U.S.A.). Two tones were used: L١=٦٥ dB SPL and L٢=٥٥ dB SPL, while f٢/f١ was ١.٢٢. Both the amplitude of response of the distortion product (DP) at ٢f١–f٢ and background noise (Ns) were obtained at nine points corresponding to f٢ frequencies of ٥٥٣, ٧٨٣, ١١٠٥, ١٥٦٠, ٢٢١١, ٣١٢٥, ٤٤١٦, ٦٢٥٠ and ٨٨٣٧ Hz. These measurements were used to build a DP-gram by displaying the DP against the f٢ frequency. The signal to noise ratio (SNR) was measured (SNR=DP–Ns) at each of these nine points. DPOAE was considered

normal (pass), thereby reflecting normal cochlear function, if the SNR was > ٣dB SPL on at least ٧٠٪ of the tested frequencies[٦].

٤- ***ABR*** using IHS with smart evoked potentials software version ٤.٥.The stimuli were ١٠٠ μs alternating click delivered through head phone at intensity level of ٨٠-٩٠ dB nHL with repetition rate of the stimuli was ٢١ p/s and ٧١ p/s. Electrode montage was high forehead to ipsilateral mastoid. The common electrode was placed on the contralateral mastoid. The response was filtered between ١٠٠ and ٣٠٠٠ Hz, amplified ١٠٠.٠٠٠ times, recorded over ١٠.٢٤ ms time window and ٢٠٠٠ sweeps were averaged for each run. ABR was done to exclude retrocochlear pathologies when hearing sensitivity permit ABR recording. MRI petrous bone with gadolinium was done in some patients instead of ABR when hearing loss is severe enough (> ٧٠ dB HL) to preclude ABR recording.

IV-<u>Vestibular evaluation in the form of</u>

١- ***A complete VNG test battery*** using the ICS Chartr binocular four channel system. VNG examination included the search for spontaneous, gaze-evoked and post-headshake nystagmus. It also included the recording of smooth pursuit, saccadic and optokinetic eye movements. The search for positioning and positional nystagmus was also performed during the right and left Dix–Hallpike tests, and when patients were in the supine position (with head centered, head to the right and head to the left), right decubitus and left decubitus positions. Finally, a monothermal caloric test was conducted using cool water irrigation at ٣٠°C [٧].

٢-***VEMP testing*** was performed using IHS with Smart EP software, version ٤.٥. The patients were tested either in the sitting position or in the supine position and head rotated away from the stimulated side during recoding. EMG activity was recorded ipsilaterally from the middle of the SCM using a surface (active) electrode, with a reference electrode on the ipsilateral sternoclavicular joint and a ground electrode on the forehead. Care was taken to place the bilateral electrodes symmetrically. During each recording session, patients were instructed to rotate their heads towards the contralateral side away from the tested ear to keep the SCM under tension. The patients were instructed to tense the SCM during acoustic stimulation and relax it between recording sessions. The stimulus will be click ١٠٠μs and STB at ٥٠٠ and ١٠٠٠ Hz (١ms rise and fall time and ٥ msec plateau time). The stimulus were presented ipsilaterally through Telephonics TDH-٤٩ headphone. The EMG signal was amplified (٥٠٠٠ times), bandpass filtered (١٠–١٥٠٠Hz). The repetition rate of the stimuli was ٥.١ stimulus/second and an average of at least ٢٠٠ sweep were taken. The intensity of each stimulus was ٩٥dBnHL and two waveforms were recorded for each stimulus type (click, ٥٠٠ Hz TB and ١٠٠٠ Hz TB) to test wave reproducibility. The analysis window started ٣٠ ms before stimulus onset and ended ٧٠ ms after stimulus onset (i.e.from–٣٠ msec to ٧٠ msec). Each ear was stimulated separately and the first ear to be tested was selected randomly.

To decrease the effect of tonic activity of the SCM on the recorded VEMP and ensure equal muscle contraction on both sides, the recording device and the Smart EP software only accepted data acquisition when the root mean square EMG activity was between ٥٠ and ١٠٠μV. Data acquisition was rejected when root mean square EMG activity was below ٥٠μV or above ١٠٠μV. The level of root mean square EMG activity was monitored and appeared on the computer screen, allowing the examiner to give feedback responses to the patient to increase or decrease muscle contraction and maintain constant muscle tension.

Upon recording the waveforms, the first positive deflection was marked as P١ and the first negative deflection will be marked as N١. Such wave (P١-N١) was wave to be analyzed. The analyzed VEMP parameters for these stimuli (click, ٥٠٠ Hz TB, ١٠٠٠ Hz TB) were:

i- P١-N١ peak to peak amplitude.

ii- P١-N١ corrected amplitude.

Corrected amplitude of P١-N١ was used to control the differences in muscle activation. The CA was computed with the software by dividing the P١–N١ amplitude

by the root mean square of EMG for the first ٣٠ ms before stimulus onset according to this formula[٨].

$$CA = \frac{\text{Raw amplitude of P1} - \text{N1} \quad \text{(peak to peak amplitude)}}{\text{Mean background amplitude } (-30 \text{ prestimulus rms EMG activity})}$$

iii-P١and N١ latency

iv-١٠٠٠/٥٠٠ Hz frequency peak amplitude (FPA) ratio:

which is defined as P١-N١ peak to peak corrected amplitude at ١٠٠٠ Hz TB divided by P١-N١ peak to peak corrected amplitude at ٥٠٠ Hz.

$$FPA = \frac{\text{P1} - \text{N1 Corrected amplitude at 1000 Hz}}{\text{P1} - \text{N1 Corrected amplitude at 500 Hz}}$$

v- Asymmetric ratio (AR):

For the control subjects, AR was calculated according to such formula

$$AR = \frac{\text{Larger corrected P1} - \text{N1 amplitude} \; - \; \text{Smaller corrected P1} - \text{N1amplitude}}{\text{Sum of corrected amplitude in both ears}}$$

For the Ménière's patients, AR was calculated according to such formula

$$AR = \frac{\text{corrected amplitude of other ear} - \text{corrected amplitude of Meniere's ear}}{\text{Sum of corrected amplitude in both ears}}$$

The VEMP results for the control group served as normative data. The normative limit for VEMP parameters were calculated as mean ± ٢.٥ SD of the results of control group.

Results

١- Intact vs Absent

Using ٥٠٠ Hz TB, VEMP results were compared between the Meniere's group and control group. While all the control ears had intact VEMP, only ١٥ (٦٠٪) Meniere's ears had intact VEMP and ١٠ ears (٤٠٪) had absent VEMP. Such results was statistically significant as shown from Binomial test (P=٠.٠٠١). On the other hand, ٢١ (٨٤٪) from the other ears had intact VEMP and only ٤ (١٦٪) had absent VEMP. This results was not statistically significant as shown from Binomial test (P=٠.١٥٤).

٢- P١-N١ Corrected amplitude (CA)

Results showed that the best stimulus that produced largest amplitude was ٥٠٠ Hz TB followed by ١٠٠٠ Hz TB and lastly click in control group. Clearly, the P١-N١ CA was largest for ١٠٠٠ Hz TB in Meniere's patients.

ANOVA test and Post hoc test were performed to compare between the Meniere's group and control group as regard P١-N١ CA. The P١-N١ CA was statistically smaller in both Meniere's ears and other ears as compared to the control ears (p value= ٠.٠٠١) while there was no statistical significant difference between Meniere's ear and other ears (p value = ٠.٠٩٧) .

٣- ١٠٠٠/٥٠٠ Hz frequency peak amplitude (FPA) ratio:

In the current study, FPR was considered abnormal if it was above the mean +٢.٥value of the control subjects (i.e; above ٠.٩٥). Similar to P١-N١ CA, the FPA ratio in both the Meniere's ear and other ear was larger compared to the control ears while there was no statistical significant difference between Meniere's ears and other ears. Table ١, ٢ show this results.

Table ١: ANOVA for comparison between Meniere's group and Control group.

	Control	Meniere's ear	Other ear	F value	P value
١٠٠٠/٥٠٠ Hz FPA ratio	٠.٦١	٠.٩٦	١.٢٧	١٣.٧٨٥	٠.٠٠٠

Table ٢: Post hoc test for comparison between Meniere's ear, other ear and control group as regard ١٠٠٠/٥٠٠ Hz FPA ratio.

	P value
Control vs Meniere's ear	٠.٠٤٧
Control vs Other ear	٠.٠٠٠
Meniere's ear vs Other ear	٠.١٦١

٤- P١ and N١ latency

Contrally to P١-N١ CA, there was no statistical significant difference between the Meniere's group (either the Meniere ear or the other ear) and control group as regard P١ latency and N١ latency (P value by ANOVA test=٠.٦٠٣ and ٠.٥٤٦ respectively)

٥- AR

Similar to P١ latency and N١ latency there was no significant difference between the Meniere's group and the control group as regard the AR (P value by independent sample T test = ٠.٠٦٠).

Discussion

TB stimulation at ٥٠٠ Hz tone was considered as an ideal stimulation[٩,١٠], with the stimulus intensity that ranged between ٩٥-١٠٥ dB nHL or ١١٥-١٣٠ dB SPL. Although the TB stimulation at ٩٥ dB nHL was the most commonly used. For clinical diagnosis using VEMP, we recommend STB stimuli because latencies and amplitude of click were significantly different among several lab[١١,١٢].

So we use both click, ٥٠٠ Hz and ١٠٠٠ Hz STB for comparison between MD group and control group, MAV group and control group in our study.

Our results show that in ٥٠٠ Hz TB, ١٥ (٦٠٪) Meniere's ear had intact VEMP and ١٠ (٤٠٪) absent which was statistically significant different. The other ear had ٢١ (٨٤٪) intact VEMP and ٤ (١٦٪) absent which is not statistically significant different.

In affected ear, VEMP was intact in ١٥ ears (٧٥٪) by Ribiero[١٣], ١٨ ears (٧٢٪) by Seo et al.,[١٤], ٢٣ ears (٥٤٪) by waele et al,[١٥], ٦٥٪ by Murofushi[١٦]. Young et al.,[١٧], de waele et al.,[١٥] Murofushi et al.,[١٦] reported that variation between in the incidence of intact VEMP can be explained by the different stages of MD.

In other ear VEMP was absent in ٢٠٪ by Ribiero[١٣], ٦ ears (٢٤٪) by Seo et al.,[١٤]. It was found that VEMP could identify occult endolymphatic hydrops on ears that were apparently a symptomatic in patients with MD[١٣].

Previous studies found reduced VEMP responses to clicks in Meniere subjects, with a substantial proportion of subjects showing no VEMP response[١٥,١٦] in comparison to normal subject there was reduced amplitude in both ears of unilateral Meniere's subjects with greater changes in the affected ears as the average VEMP amplitude at the affected side is significantly lower than that at the unaffected side. It was explained by reduced VEMP amplitude at the affected side points toward a permanently affected otolith system in unilateral Meniere's patients at the side of Meniere's ear. The affection of the cochlea and part of the vestibular system are related. Kingma[١٨], Rauch et al.,[١٠] which agree with our results.

Our result shows that there was no statistically significant difference between Meniere's group and control group in P١ and N١ latency.

There was no statistically significant difference between absolute latencies of wave P١ and N١ on affected and a symptomatic ears[١٣]. The same was found by young et al.,[١٧], Waele et al.,[١٥] observed latency of response in subjects MD similar to that of normal subjects detecting differences only in amplitude of response. These finding are similar to our results. This can be explained by that the latency depends predominantly on central signal transmission and muscle activation, is unaffected by saccular deformation (hydrops) in Meniere's disease[١٠].

Young et al.,[١٩] reporting ١٠٪ of cases of late endolymphatic hydrops with prolongation of absolute latency of P١, justifying these finding owing to high endolymphatic pressure that would affect the transmission of sound, provided that hearing was also affected to Murofushi et al.,[٢٦], prolongation of latency of P١ suggested retrolabyrinthine damage. These are against our results.

Because MD reduces VEMP responses, it might be thought that a symmetry of the VEMP response would provide a good clinical test of unilateral MD but unexpectly this difference was not statistically significant this is explained by Rauch et al.,[١٠] that there is occult disease in the unaffected ear [occult bilateral disease in Meniere's subjects] and this is similar to our results.

Our results show in both Meniere's ear and other ear FPA ratio was larger compared to control ears while no statistically significant difference between Meniere's ear and other ears. Kim-Lee[٢٠] show that ١٠٠٠/٥٠٠ Hz FA ratio is elevated in MD and this represents a useful diagnostic criteria in the diagnosis of MD. Kim-Lee[٢٠], Rauch et al.,[١٠] suggested that the elevated FPA ratio in their VEMP responses using TB stimuli of ٥٠٠ and ١٠٠٠ Hz. Likely due to a shift in the morphologic features of the saccule.

The change in VEMP frequency tuning produced by MD provides a clue to the origin of VEMP tuning. Welgampola and Colebatch[٢١] suggested that VEMP tuning originated in an electrical resonance of the hair cells, whereas Todd et al.,[٢٢] suggested that VEMP tuning was attributed to that VEMP tuning was attributed to the mass spring damping properties of the saccule.

We found that the FPA ratio was significantly different between patients with MD and normal subjects. So we suggest that the resultant FPA cutoff value would be clinically useful in diagnosis of MD which agree with Kim lee and Rauch et al.,

Conclusion

The altered VEMP dynamics seen in Meniere's disease offer the possibility that VEMP testing may be clinically useful in the assessment and early detector of involvement of sacculus in endolymphatic hydrops of MD, both on the affected and on the asymptomatic ear and/or monitor of evolving Meniere's disease or of the efficacy of potential Meniere's treatments.

Reference

١. Akkuzu G, Akkuzu B, Ozluoglu LN. Vestibular evoked Myogenic potentials in benign paroxysmal positional vertigo and Meniere's disease. Eur Arch Otorhinolaryngol ٢٠٠٦; ٢٦٣: ٥١٠-٥١٧.

٢. American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS). Committee on Hearing and Equilibrium. Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. Otolaryngol Head Neck Surg ١٩٩٥; ١١٣(٣): ١٨١–١٨٥.

٣. Brantberg K, Trees N, Baloh RW. Migraine associated vertigo. Acta Otolaryngol ٢٠٠٥; ١٢٥: ٢٧٦-٢٧٩.

٤. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by click evoked vestibulocolic reflex. Je Neurol Neurosurg psychiatr ١٩٩٤; ٥٧:١٩٠-١٩٧.

٥. Cutrer FM, Baloh RW. Migraine associated dizziness. Headache ١٩٩٢; ٣٢:٣٠٠-٣٠٤.

٦. Gorga MP, Neely ST, Ohlrich B et al., From laboratory to clinic: a large scale

study of distortion product otoacoustic emission in ears with normal hearing and ears with hearing loss. Ear Hear ١٩٩٧; ١٨:٤٤٠–٥٥.

٧. Becker GD. The screening value of monothermal caloric tests. Laryngoscope ١٩٧٩; ٨٩:٣١١–١٤.

٨. Murofushi T, Ozeki H, Inoue A N, Sakata A Does migraine associated vertigo share a common pathophysiology with Meniere's disease? Study with vestibular evoked myogenic potential. Cephalalgia ٢٠٠٩; ٢٩:١٢٥٩-١٢٦٦.

٩. Young YH. Vestibular evoked myogenic potentials: optimal stimulation and clinical application. Journal of Biomedical Science ٢٠٠٦; ١٣(٦):٧٤٥–٧٥١.

١٠. Rauch SD, Zhou G, Kujawa SG, Guinan JJ and Herrmann BS. Vestibular evoked myogenic potentials show altered tuning in patients with Meniere's disease. Otology and Neurotology ٢٠٠٤; ٢٥(٣): ٣٣٣–٣٣٨.

١١. Ozgur A, Erdivanli OC, Coskun ZO, Terzi S, Yigit E, Demirci M and Dursun E. Comparison of Tone Burst, Click and Chirp Stimulation in Vestibular Evoked Myogenic Potential Testing in Healthy People. Int Adv Otol ٢٠١٥; ١١(١):٣٣-٣٥.

١٢. Wu HJ, Shiao AS, Yang YL and Lee GS. Comparison of short tone burst-evoked and click-evoked vestibular myogenic potentials in healthy individuals. Journal of the Chinese Medical Association ٢٠٠٧; ٧٠(٤): ١٥٩–١٦٣.

١٣. Ribeiro S, de Almeida RR, Caovilla HH and Gananca MM. Vestibular evoked myogenic potentials in affected and asymptomatic ears in unilateral Meniere's Disease. Rev Bras Otorrinolaringol ٢٠٠٥; ٧١ (١):١-١٠.

١٤. Seo T, Node M, Yukimasa A and Sakagami M. Furosemide loading vestibular evoked myogenic potential for unilateral Meniere's disease. Otol Neurotol ٢٠٠٣; ٢٤:٢٨٣-٢٨٨.

١٥. de Waele C, Huy PTB, Diard JP, Fraysse G, Vidal PP. Saccular dysfunction in Meniere's disease. Am J Otol. ١٩٩٩; ٢٠: ٢٢٣-٢٣٢.

١٦. Murofushi T, Shimizu K, Takegoshi H, Cheng P. Diagnostic value of prolonged latencies in the vestibular evoked myogenic potential. Arch Otolaryngol Head Neck Surg ٢٠٠١; ١٢٧:١٠٦٩-١٠٧٢.

١٧. Young YH, Huang TW, Cheng PW. Assessing the stage of Meniere's disease using vestibular evoked myogenic potentials. Arch Otolaryngol Head Neck Surg ٢٠٠٣; ١٢٩:٨١٥-٨١٨.

١٨. Kingma C M and Wit HP. Asymmetric vestibular evoked myogenic potentials in unilateral Meniere patients. Eur Arch Otorhinolaryngol ٢٠١١;٢٦٨: ٥٧-٦١.

١٩. Young YH, Huang TW, Cheng PW. Vestibular evoked myogenic potentials in delayed endolymphatic hydrops. Laryngoscope ٢٠٠٢; ١١٢: ١٦٢٣-١٦٢٦.

٢٠. Kim-Lee Y, Ahn JH, Kim YK and Yoon TH. Tone burst vestibular evoked myogenic potentials: diagnostic criteria in patients with Meniere's disease. Acta Oto-Laryngologica ٢٠٠٩;١٢٩:٩٢٤-٩٢٨.

٢١. Welgampola Todd NP, Cody FW, Banks JR. A saccular origin of frequency tuning in myogenic vestibular evoked potentials? implications for human resp-onses to loud sounds. Hear Res ٢٠٠٠; ١٤١:١٨٠-١٨٨.

٢٢. MS, Colebatch JG. Characteristics and clinical applications of vestibular evoked myogenic potentials. Neurology ٢٠٠٥; ٦٤:١٦٨٢-١٦٨٨.