# Utility of Vestibular Evoked Myogenic Potentials in the Differential Diagnosis of Suspected Meniere's Disease and Benign Paroxysmal Positional Vertigo

## Vivekanandh M. & Animesh Barman\*

### Abstract

VEMP is a valuable clinical tool in the differential diagnosis of various conditions affecting the normal physiology of the vestibular system. It provides information about the functioning of the otolith organ and the functional integrity of the inferior vestibular nerve. Meniere's disease (MD) and benign paroxysmal positional vertigo (BPPV), which exhibit almost similar patterns of symptom, has to be differentiated from each other. Hence, the present study was aimed to identify the pattern of VEMP's results in individuals with MD and BPPV and also how these wave forms are different from waveform that are recorded from the individuals with any vestibular abnormalities. The results indicated a significant difference in the latency of the p13 and n23 and also the peak to peak amplitude across the groups. The VEMP's responses rates of the MD group were the least among the groups. The interaural amplitude difference ratio was significantly higher in the MD group. A difference in the VEMP responses between the unaffected and affected side in individuals with unilateral MD was also observed. Thus, the IADR value could be used to identify individuals with MD.

Abbreviations: VEMP: Vestibular Evoked Myogenic Potentials. MD: Meniere's Disease BPPV: Benign Paroxysmal Positional Vertigo IADR: Interaural Amplitude Difference Ratio

### Introduction

Vestibular evoked myogenic potential (VEMP) is an electromyographic response to loud auditory stimuli that is recorded from the sternocleidomastoid muscle during tonic contraction. It is used as a clinical test to assess vestibular system as it provides information about the functioning of the otolith organ and the functional integrity of the inferior vestibular nerve, (Zhou & Cox, 2004).

Vestibular neuritis, benign paroxysmal positional vertigo (BPPV), and Meniere's disease (MD) are the most common diseases that cause peripheral vertigo. The development of peripheral vertigo can be associated with the saccule or inferior vestibular nerve. Patients with vestibular neuritis also show unilateral peripheral vestibular dysfunction mainly in the superior vestibular nerve (Fetter & Dichgans, 1996). Recent studies have also demonstrated that some patients with having vestibular neuritis in the inferior vestibular nerve (Halmagyi, Aw, Karlberg, Curthoys, & Todd, 2001).

Heide, Freitag, Wollenberg, Schimrigk, and Dillmann (1999) reviewed VEMP response in three BPPV patients, in which all the patients had normal VEMP responses.

<sup>\*</sup> Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore, India email: nishiprerna@rediffmail.com

However, a more recent study on BPPV patients indicated that 30% of the patients had abnormal VEMP responses (Akkuzu, Akkuzu & Ozluoglu, 2006). Matsuzaki and Murofushi, (2001) reported bilateral absence of VEMPs in cases with bilateral vestibulopathy. Ochi, Ohashi, and Watanabe, (2003) reported abnormal VEMPs and its recovery in patients with ipsilateral vestibular neuritis.

Vestibular-dependent short-latency electromyographic (EMG) responses to intense sound were initially recorded from the posterior neck muscles inserting at the inion, (Bickford, Jacobson & Cody, 1964). VEMPs are now recorded using symmetric sites over the sternocleidomastoid muscles (SCMs), (Colebatch, Halmagyi, & Skuse, 1994). The response consists of an initial positivity or inhibition (p13) followed by a negativity or excitation (n23). Later components (n34, p44) have a lower stimulus threshold and are non-vestibular (probably cochlear) in origin.

The VEMP arises from modulation of background EMG activity and it requires tonic contraction of the muscle. It is best observed in averaged unrectified EMG (Colebatch & Rothwell, 2004).

A morphologic and physiologic study in experimental animals confirms that intense sound selectively activates otolith afferents, (Murofushi, Curthoys, & Gilchrist, 1996). Stimulation of the saccular nerve in cats results in inhibitory postsynaptic potentials in the ipsilateral SCM motor neurons, which travel in the medial vestibulospinal tract, (Uchino, Sato, & Sasaki, 1997; Kushiro, Zakir, Ogawa, Sato, & Uchino, 1999) with only weak effects on the contralateral neurons. Utricular nerve stimulation, in contrast, evokes excitatory postsynaptic potentials in about two-thirds of contralateral SCM neurons, (Uchino, Sato, & Sasaki, 1997). Thus, the predominantly ipsilateral, inhibitory SCM responses (e.g., click VEMPs) are likely to represent saccular activation, and prominent crossed responses (observed in direct current and tap-evoked VEMPs) may indicate utricular stimulation.

By using the vestibular apparatus, VEMP has been used to assess not only the inferior vestibular nerve, also the activity of extra ocular muscles using Ocular-VEMP (Iwasaki et al, 2007), the crossed and uncrossed pathways of spinal cord (Rudisill, & Hain, 2008), and vestibular evoked potentials recorded from human masseter muscles and from scalp electrodes are the new techniques whose characteristics are still being explored.

#### Need for the study

Vestibular-evoked myogenic potential testing may provide additional information about the vestibular system and allow site of lesion testing (e.g. saccule and inferior vestibular nerve) in patients of all ages. Its role has yet to be defined in the diagnosis and treatment of common vestibular disorders, including Meniere's disease, vestibular neuronitis, labyrinthitis, and other diseases. Further, research is needed to support its clinical usefulness in patients with balance disorders, to optimize patient selection, and to establish its cost effectiveness (Honaker, & Samy, 2007). New applications for vestibular evoked myogenic potential is needed in diagnosis and monitoring of neurotologic disease, and in shedding light on inner ear diseases by mapping anatomic sites of involvement. The most informative work is still in the areas of Benign paroxysmal positional vertigo and in Meniere's disease. Also, many aspects of vestibular evoked myogenic potential and its use have not yet been adequately studied or described. It holds great promise for diagnosing and monitoring Meniere's disease and Benign paroxysmal positional vertigo. The methods, equipment, and applications for vestibular evoked myogenic potential testing are not yet standardized (Rauch, 2006).

VEMP is a testing method that evaluates the saccule and the inferior vestibular nerve in the peripheral vestibular system. The test is easy, noninvasive and causes minimal patient discomfort. VEMP has been used as a complimentary test with the conventional vestibular function test in patients with peripheral vertigo. The main parameters of the VEMP responses used in clinical diagnosis are p13 and n23 latencies and the peak to peak amplitude. Recently, interaural amplitude difference ratio (IADR) has been recognized as one of the valuable clinical tools in the assessing individuals with vestibular dysfunction (Young, Huang & Cheng, 2003). Any conditions affecting the normal physiology of the vestibular system will have a significant effect on its evoked potentials. The most common conditions affecting the vestibular system are Meniere's disease and benign paroxysmal positional vertigo. IADR might throw some important information in identification of BPPV and MD. Thus, the current study has been taken up, with the following aim.

# Aim of the study

- To identify the pattern of VEMP responses in individuals with normal auditory and vestibular functioning, individuals with MD and in individuals with BPPV.
- To compare the parameters of VEMP responses between the groups.
- To compare the interaural amplitude difference ratio (IADR) across the groups.
- To check for ear effect on VEMP responses for individuals with unilateral MD.

## Method

The main aim of the study was to identify the pattern of VEMP's recorded from individuals with conditions indicating disturbances of vestibular system and to compare it with the VEMP's recorded from normal individuals. Three groups of subjects were taken to arrive at the objectives.

## **Subjects**

A total of 75 ears of 43 subjects were taken for the study. They were divided into three groups. Group I consisted of individuals with normal hearing sensitivity without vestibular symptoms served as the control; group II consisted of individuals who were diagnosed as having Meniere's Disease, and group III consisted of individuals who were diagnosed as having BPPV by an otologist.

# Group I

Consisted of 33 ears of 20 individuals with normal auditory and vestibular functioning and was ruled out by taking detailed case history. These individuals were between the age range of 18-24 years with a mean age of 20.45 years. The subjects were selected based on the following criteria:

# Selection Criteria

- Pure tone audiometric thresholds were within 15 dB HL in octave frequencies from 250 Hz to 8000 Hz for air conduction and between 250 Hz and 4000 Hz for bone conduction.
- Uncomfortable level was equal to or greater than 100 dB HL for Speech.
- All the subjects had 'A' type tympanogram with acoustic reflex threshold within normal limits, indicating a normal middle ear function.
- Auditory brainstem evoked response (ABR) results did not indicate of having space occupying lesions (retro cochlear pathology).
- No relevant otologic history was present in those subjects.
- No history of any observable medical or neurological signs.

# Group II

Consisted of 22 ears of 12 individuals with suspected Meniere's disease. Out of 12 individuals 8 individuals had bilateral and 4 individuals had unilateral indications of Meniere's disease. These individuals were between the age range of 20-60 years with a mean age of 41.3 years.

# Group III

This group had 21 ears from 11 individuals with suspected BPPV. The mean age of this group was 39.7 years with a range of 20 to 60 years.

# Selection Criteria for group II and III

- The hearing sensitivity varied from normal hearing sensitivity to severe sensorineural hearing loss for meniere's group whereas for BPPV group the thresholds varied from normal hearing to mild sensorineural hearing loss.
- All the subjects had uncomfortable level greater than 100 dB HL for Speech.
- All of them had 'A' type tympanogram with normal, elevated or absent acoustic reflexes.
- No relevant history of middle ear pathology was reported.
- All of them were devoid of having retro cochlear pathology (RCP), which was ruled out based on ABR results.
- The subjects diagnosed as having Meniere's disease or BPPV by an experienced otologist or a neurologist was taken for the study.

- All the subjects had the triad symptoms of Meniere's disease: fluctuating hearing loss, tinnitus and, giddiness.
- All the subjects with BPPV had symptoms of tinnitus, and giddiness induced by rapid head movement.

# Procedure

A detailed case history was taken from all the subjects. Later all of them underwent routine audiological assessment which consisted of pure tone audiometry, speech audiometry, immitance testing. Auditory brainstem response (ABR) was also administered using standard test protocol to rule the presence of any retro-cochlear pathology.

Inter wave latency was noted from the ABR waveform recorded at 11.1/ sec stimulus rate and wave morphology and presence or absence of ABR wave V was noted from the ABR wave recorded at 90.1/ sec stimulus rate to identify retro-cochlear pathology (RCP). Those who had normal inter wave latency and good morphology at 90.1/ sec was considered as not having RCP and was included for the study.

All the subjects selected for the study had undergone VEMP recording. Procedure cited below has been adopted to record VEMP. The subjects were placed in a comfortable environment, where the subjects were made to sit upright position on an arm chair. The subjects were asked to turn their head to one side (opposite to the ear being stimulated) to tense the Sternocleidomastoid (SCM) muscle. The SCM muscle tension were monitored to be within 30–100 micro volt Electromyographic (EMG) level for the reliable recording of VEMP responses. Protocol given by Damen, (2007) was used to record VEMP is shown in the Table 1.

Stimulus Parameters		Acquisition Parameters	
Stimulus	500 Hz Tone Burst	Mode	Ipsilateral
Duration	10 ms	Electrode type	Disc electrode
Stimulus rate	5.1 per sec	Electrode	Ground: Forehead
		montage	Non inverting : middle portion of
			Sternocleidomastoid (SCM)
			Inverting: : Sterno-clavicular junction
Polarity	Alternating	Analysis window	-30 to 70 ms
No. of Sweeps	200	Filter settings	10 to 1500 Hz
Intensity	95 dBnHL	Notch Filter	Off
Transducer	ER 3A Insert	Impedance	Intra electrode : $< 5$ k ohm
	receiver		Inter electrode: within 2 k ohm

Table 1: Protocol used to record VEMP

Acoustically evoked VEMPs were recorded twice to check for its reliability and stored in the computer. Later it was retrieved and shown to three audiologists independently to identify the VEMP waves. The p13 and n23 peak latency and also peak to peak amplitude was noted, in case there was an agreement in identifying peaks among the audiologists. The interaural amplitude difference ratio was calculated for all the three groups.

#### Results

The P13 and N23 latency and peak to peak amplitude was noted from all the subjects for all the three groups. The data were subjected to appropriate statistical analysis. The VEMP responses were present in 100% of individuals with normal auditory and vestibular functioning, 42% in Meniere's disease group, and 60% in individuals with BPPV.

A group comparison was made by comparing the responses recorded from the three groups by analyzing the latencies of p13 and n23 peaks and the peak to peak amplitude. Also the mean and standard deviation for the individual parameters were calculated using descriptive statistics. For the group comparison the VEMP responses of right and left ear were combined for all the groups as there was no significant difference in latency or amplitude values between the ears for all the groups.

Table 2: Mean, SD and Range of p13, n23 and peak to peak amplitude obtained in all the groups

Group		p13	n23	PPA
Normals	Mean	13.81	21.00	59.19
	SD	1.66	1.97	24.50
	Range	9.40 - 18.00	17.00 - 24.40	1.54 - 104.60
MD	Mean	16.53	22.43	25.44
	SD	2.54	3.64	15.11
	Range	11.40 - 19.60	16.20 - 29.40	6.29 - 47.00
BPPV	Mean	17.81	27.08	29.61
	SD	5.48	6.21	15.67
	Range	9.60 - 27.00	18.20 - 36.00	3.26 - 59.87

It is apparent from the table 2 that the latency values obtained from individuals with normal auditory and vestibular functioning were shorter when compared to the clinical group. Within the clinical group, MD group's latency was shorter than the BPPV group. Also, BPPV group had maximum variation in latency values than the MD group and individuals with normal auditory and vestibular functioning. Individuals with normal auditory and vestibular functioning had highest peak to peak amplitude followed by BPPV group and the MD group had the least peak to peak amplitude. Also, there was maximum variation in the peak to peak amplitude recorded from individuals with normal auditory and vestibular functioning, whereas the MD group had the least variation.

To see the significant difference among the latencies of p13 and n23 and peak to peak amplitude of the VEMP responses recorded from the three groups, MANOVA was done. The results of the MANOVA revealed that there was a significant difference in the latencies of p13 [F (2, 53) = 8.912, p<0.001], n23 [F (2, 53) = 12.335, p<0.001] and also for the peak to peak amplitude [F (2, 53) = 15.414, p<0.001] across the three groups.

Since, there was uneven sample size among the three groups taken for the study due to presence of no responses which cannot be taken for statistical analysis; Kruskal-wallis test was done to cross check the results of the MANOVA. The results of Kruskal-wallis also

revealed that there was a significant difference in the latency values of p13, n23 and peak to peak amplitude respectively which is in accordance with the results of MANOVA (Table 3).

Parameter	Chi Square Value	Degree of Freedom	Sig. Level
p13	12.996	2	.002*
n23	9.171	2	.010*
PPA	21.273	2	.000*

Table 3: Chi square values along with significant level across the groups

Duncan's Post hoc test was done to compare the latencies of p13 and n23 and peak to peak amplitude between any two groups since the MANOVA showed significant differences across the groups. For the positive peak p13, the individuals with normal auditory and vestibular functioning group had significantly shorter p13 latency than the individuals with MD and BPPV group. However, individuals with MD and BPPV group did not differ significantly in the p13 latency obtained.

There was no significant difference in n23 latency observed between normal group and MD group. However, BPPV group significantly differed from the other two groups. For the peak to peak amplitude, there was no significant difference in peak to peak amplitude observed between MD group and BPPV group. Whereas, there was a significant difference observed when compared with the individuals with normal auditory and vestibular functioning group.

# Inter-aural amplitude difference

The mean IADR was calculated for normal group and MD group and not for BPPV group since only two patients showed bilateral VEMP responses which cannot be considered for statistical analysis. The mean and SD of IADR value was calculated for the normal and MD group which is given in the figure I.



Figure I: Mean and SD values of IADR measured for normal group and MD group.

The mean IADR of MD group (0.3775) is greater than the IADR of normal group (0.1578). The Mann Whitney-U test was done to see the significant difference in IADR values between the groups. The results revealed a significant difference between the IADR values of normal group and MD group (Z = 2.551, p< 0.05).

## Ear effect in Meniere's disease group

Out of 12 individuals with MD, 4 of them had unilateral MD. The mean latencies of p13 and n23 from the unaffected side of the unilateral subjects were 16.95 ms and 24.05 ms respectively. And the mean peak to peak amplitude in these subjects was 25.48 micro volts. However, in the affected side 2 individuals showed absent VEMP responses and the others showed prolonged latencies and reduced amplitude values. The Wilcoxon's signed rank test was done to compare the latency and amplitude between the unaffected and affected ears of unilateral MD group.

Table VI: Z-values and significant levels of the VEMP parameters obtained between the ears in individuals with unilateral Meniere's disease.

Unaffected ear Vs	Z-value	Level of Significance
Affected ear		
P13	1.342	.180
N23	1.342	.180
PPA	0.447	.655

The result showed that there were no significant differences among latency and amplitude values between the ears. Whereas, descriptively the latency of p13 and n23 of the unaffected ears were shorter than the latency values of the affected ears. The amplitude of unaffected ears showed greater value than the affected ears. The VEMP responses were either absent or delayed in latency and reduced in amplitude in the affected ear when compared with the responses from the unaffected ear.

## Discussion

VEMP responses in individuals with normal auditory and vestibular functioning, Meniere's disease, and BPPV.

The present study revealed a 100% response rate in individuals with normal auditory and vestibular functioning. This is in accordance with the results obtained by Castelein, Deggouj, Wuyts and Gersdorff, (2008). The mean p13 and n23 latencies recorded in the present study were  $13.81\pm1.66$  ms and  $21\pm1.97$  ms respectively. Welgampola and Colebatch (2001) found that the average p13 and n23 latencies to a tone burst stimulus were 13.1 and 22.8 ms respectively.

The peak to peak amplitude obtained in the present study was  $59.19\pm24.50$  micro volts with a range from 1.54 to 104.60 micro volts. Castelein, et al. (2008) also cited that the amplitude of the p13 n23 varies widely among individuals making it difficult to use the amplitude parameter for clinical evaluation.

In the present study VEMP responses were recorded from 42% of individuals with MD with poor wave morphology. De Waele et al., (1999) reported a 46% response rate in individuals with MD. The mean p13 and n23 latency in the present study was  $16.53\pm2.54$  ms and  $22.43\pm3.64$  ms respectively. The mean peak to peak amplitude was about  $25.44\pm15.11$  micro volts. Hong et al (2008) obtained the mean p13 and n23 latency of about  $17.1\pm3.2$  ms

and 23.0 $\pm$ 3.2 ms respectively and also the peak to peak amplitude of about 20.8 $\pm$ 19.7 micro volts.

In the present study VEMP responses were recorded in individuals with BPPV with a response rate of 60% and the mean p13 and n23 latencies were  $17.81\pm5.48$  ms and  $27.08\pm6.21$  ms respectively whereas the mean peak to peak amplitude was  $29.61\pm15.67$  micro volts. Hong, Yeo, Kim and Cha, (2008) recorded VEMP responses in 75% of individuals with BPPV with mean p13 and n23 latency of about  $16.5\pm2.6$  ms and  $22.6\pm2.8$  ms respectively with mean peak to peak amplitude of about  $15.3\pm22.0$  micro volts.

## Comparison of VEMP responses across the groups

A significant difference in the p13 and n23 latency and also the peak to peak amplitude across the groups was observed. Akkuzu, Akkuzu, & Ozluoglu, (2006) also found similar results from their study by comparing VEMP responses from individuals with MD and BPPV and concluded that there was a significant difference in the VEMP responses recorded from these two clinical groups.

The latency of the first positive peak p13 obtained from the individuals with normal auditory and vestibular functioning group were significantly shorter than the individuals with MD and BPPV group. However, individuals with MD and BPPV group did not differ significantly in the p13 latency obtained. This is in contrary to Hong et al. (2008), according to them the prolongation of the p13 latency in BPPV group helped in differentiating from the individual with MD and vestibular neuritis.

The present study also showed n23 latency for BPPV group was significantly different when compared with either normal or MD group. There was no significant difference in peak to peak amplitude observed between MD group and BPPV group. The difference in the prolongation VEMP in individuals with BPPV can be attributed to the direct involvement of the saccular maculae, whereas in the MD group the hydrops could have been confined only to the cochlea thereby affecting the sound transmission to the saccule but not affecting the physiology of saccule directly (Welling et al, 1997 & Hong et al, 2008).

### Inter-aural amplitude difference ratio

The mean IADR of MD group  $(0.3775\pm0.17)$  was greater than the IADR of normal group  $(0.1578\pm0.22)$ . This result was in accordance with the study done by Young, Huang and Cheng (2003). They studied the IADR and grouped the MD individuals into different stages. They grouped individuals with MD with an IADR of  $0.30\pm0.30$  into Stage III, which is characterized by a depressed or absent VEMP responses and also flat audiometric configuration. A dilated saccule with an atrophied saccular macula, which was described in one histopathologic study of Meniere's disease (Schuknecht & Gulya, 1983), could be an explanation for depressed VEMPs which supports the results of the present study. So, the increased IADR in the MD group can be attributed to the presence of an atrophied macula.

## Ear effect in Meniere's disease group

In the present study, the VEMP responses recorded in individuals with unilateral MD showed either prolonged latencies with reduced amplitude or absent responses in the affected side. But the unaffected side showed VEMP responses in all the ears. This difference among the unaffected and the affected ears were not statistically significant but it was observed that the latency was relatively shorter in the unaffected side. Also, the peak to peak amplitude was relatively greater in the unaffected side. A recent study compared VEMP in patients with Vestibular Drop Attacks (VDA) and non-VDA secondary to MD and reported that the incidence of absent VEMP in the affected ear with VDA was significantly larger than that in the affected ear with non-VDA (Timmer et al., 2006). While their findings suggested that VDA could arise from damaged otolithic organs, their results did not reveal reversibility of damage or the possible existence of endolymphatic hydrops in the otolithic organ.

## Conclusions

The present study aimed at differentiating Meniere's disease and benign paroxysmal positional vertigo based on VEMP results. The VEMP response rates of the MD group were the least among the groups. There was a significant difference in the latency of p13 and n23 and also the peak to peak amplitude across the groups. The p13 latency of MD and the BPPV group were comparable whereas the n23 latency of the BPPV group was significantly prolonged than the MD group. There was difference in the VEMP responses of MD and BPPV group between the ears descriptively but statistically it was not significant. The Interaural amplitude difference ratio was significantly higher in MD group. Descriptively, there was a difference in the VEMP responses between the unaffected and affected side in individuals with unilateral MD. Thus, the IADR value could be used to identify individuals with MD.

## Implications of the study

- The peak latency and the amplitude data can be used as normative for future research and clinical evaluation.
- The VEMP response rate, peak latencies, IADR can be used as reliable tools to differentially diagnose between MD and BPPV.
- The results can be added to the current literature in the evaluation of vestibular disorders using VEMP.

#### References

- Akkuzu, G., Akkuzu, B., & Ozluoglu, L. N. (2006). Vestibular evoked myogenic potentials in benign paroxysmal positional vertigo and Meniere's disease. *Europian Archives* in Otorhinolaryngology, 263, 510-517.
- Bickford, R. G., Jacobson, J. L., & Cody, D. T. (1964). Nature of average evoked potentials to sound and other stimuli. *Annals of New York Academy of Sciences*, *112*, 204-223.
- Castelein, S., Deggouj, N., Wuyts, F., & Gersdorff, M. (2008). Vestibular evoked myogenic potentials *B-ENT*, *4*, *Supplement*. *8*, 39-43.
- Colebatch, J. G., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click-evoked vestibulocolic reflex. *Journal of Neurology, Neurosurgery, and Psychiatry*, 57, 190-197.
- Colebatch, J. G., Rothwell, J. C. (2004). Motor unit excitability changes mediating vestibulocollic reflexes in the sternocleidomastoid muscle. *Clin Neurophysiol*; 115: 2567-2573.
- Damen, (2007). Vestibular Evoked Myogenic Potential (VEMP), Clinical application of the threshold. *Master thesis Medical Engineering*.
- De Waele, C., Huy, P. T., Diard, J. P., Freyss, G., & Vidal, P. P. (1999). Saccular dysfunction in Meniere's disease. *The American Journal of Otology*, 20, 223–232.
- Fetter, M., & Dichigans, J. (1996). Vestibular neuritis spares the inferior division of the vestibular nerve. *Brain*, 119, 755-63.
- Halmagyi, G. M., Aw, S. T., Karlberg, M., Curthoys, I. S., & Todd, M. J. (2001). Inferior vestibular neuritis. *Annals of the New York Academy of Sciences*, *956*, 306-313.
- Heide, G., Freitag, S., Wollenberg, I., Iro, H., Schimrigk, K., & Dillmann, U. (1999). Click evoked myogenic potentials in the differential diagnosis of acute vertigo. *Journal of Neurology, Neurosurgery, and Psychiatry*, 66, 787-790.
- Honaker, J. A., & Samy, R. N. (2007). Vestibular-evoked myogenic potentials. *Current Opinions in Otolaryngology, Head and Neck Surgery*, 15(5), 330-4.
- Hong, S. M., Yeo, S. G., Kim, S. W., & Cha, C. I. (2008). The results of vestibular evoked myogenic potentials, with consideration of age-related changes, in vestibular neuritis, benign paroxysmal positional vertigo, and Meniere's disease. Acta Otolaryngologica, 128(8), 861-5.
- Iwasaki, S., McGarvie, L. A., Halmagyi, G. M., Burgess, A. M., Kim, J., Colebatch, J. G., & Curthoys, I. S. (2007). Head taps evoke a crossed vestibulo-ocular reflex. *Neurology*, 68(15), 1227-9.
- Kushiro, K., Zakir, M., Ogawa, Y., Sato, H., & Uchino Y. (1999). Saccular and utricular inputs to sternocleidomastoid motorneurons of decerebrate cats. *Experimental Brain Research*, 126, 410-416.

- Matsuzaki, M., & Murofushi, T. (2001). Vestibular evoked myogenic potentials in patients with idiopathic bilateral vestibulopathy. Report of three cases. *Journal of Otorhinolaryngology*, 63, 349–352.
- Murofushi, T., Curthoys, I. S., & Gilchrist, D. P. (1996). Response of guinea pig vestibular nucleus neurons to clicks. *Experimental Brain Research*, 111(1), 149-52.
- Rauch, S. D. (2006). Vestibular evoked myogenic potentials. *Current Opinons in Otolaryngology Head and Neck Surgery*, 14(5), 299-304.
- Rudisill, H. E., & Hain, T. C. (2008). Lower extremity myogenic potentials evoked by acoustic stimuli in healthy adults. *Otology & Neurotology, 29(5),* 688-92.
- Schuknecht, H. F., & Gulya, A. J. (1983). Endolymphatic hydrops: an overview and classification. *Annals of Otology Rhinology Laryngology Supplement*, 106, --20.
- Timmer, F. C. A., Zhou, G., Guinan, J. J., Kujawa, S. G., Herrmann, B. S., & Rauch, S. D. (2006). Vestibular evoked myogenic potential (VEMP) in patients with Meniere's disease with drop attacks. *Laryngoscope*, 116, 776-779.
- Uchino, Y., Sato, H., & Sasaki, M. (1997). Sacculocollic reflex arcs in cats. *Journal of Neurophysiology*. 77, 3003-3012.
- Welgampola, M. S., & Colebatch, J. G. (2001). Characteristics of tone burst-evoked myogenic potentials in the sternocleidomastoid muscles. *Otology & Neurotology*, 22, 796-802.
- Welgampola, M. S., & Colebatch, J. G. (2005). Characteristics and clinical applications of vestibular evoked myogenic potentials. *Neurology*. 64. 1682-1688.
- Welling, D. B., Parnes, L. S., O'Brien, B., Bakaletz, L. O., Brackmann, D. E., Hinojosa, R. (1997). Particulate matter in the posterior semicircular canal. *Laryngoscope*, 107, 90-94.
- Young, Y. H., Huang, T. W., & Cheng, P. W. (2003). Assessing the stage of Meniere's disease using vestibular evoked myogenic potentials. Archives in Otolaryngology and Head and Neck Surgery, 129, 815-818.
- Zhou, G., & Cox, C. L. (2004). Vestibular Evoked Myogenic Potentials: History and Overview. *American Journal of Audiology*, *13*, 135-143.