# VESTIBULAR EVOKED MYOGENIC POTENTIALS IN NORMALS AND IN INDIVIDUALS WITH DIZZINESS

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A dissertation submitted in part fulfillment for the degree of Master of Science (Audiology) University of Mysore, Mysore

> ALL INDIA INSTITUTE OF SPEECH & HEARING MANSAGANGOTHRI, MYSORE-570006 APRIL 2006.

Dedicated to My family For their support and affection &

....

My guide For his unmatched guidance

# CERTIFICATE

This is to certify that this dissertation entitled "VESTIBUALAR EVOKED MYOGENIC POTENTIAL IN NORMALS AND IN INDIVIDUALS WITH DIZZINESS" is a bonafide work in part fulfillment for the degree of Master of science (Audiology) of the student Registration no: A0490003.This has been carried under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any diploma or degree.

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Mysore April 2006

# CERTIFICATE

This is to certify that this dissertation entitled "VESTIBUALAR EVOKED MYOGENIC POTENTIAL IN NORMALS AND IN INDIVIDUALS WITH DIZZINESS" has been prepared under my supervision & guidance. It is also certified that this dissertation has not been submitted earlier to any other university for the award of any diploma or degree.

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# DECLERATION

This is to certify that this master's dissertation entitled "VESTIBUALAR EVOKED MYOGENIC POTENTIAL IN NORMALS AND IN INDIVIDUALS WITH DIZZINESS" is the result of my own study and has not been submitted earlier to any other university for that award of any degree or diploma.

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#### INTRODUCTION

Vestibular evoked myogenic potentials (VEMPs) were first described by Bickford, Jacobson, and Cody (1964), and recently have been proposed as a reliable clinical test of saccular or inferior vestibular nerve function (Cloebatch, 2001). VEMPs are short latency electromyogram (EMG) that are evoked by higher-level acoustic stimuli and are recorded from surface electrodes over the tonically contracted sternocleidomastiod (SCM) muscle. The neurophysiological and clinical data indicate that, the VEMPs are mediated by a pathway that includes the saccular macula, inferior vestibular nerve, the lateral vestibular nucleus, the lateral vestibulospinal tract, and the motorneurons of the ipsilateral SCM muscle (Halmagyi & Curthoys, 2000).

Geisler, Frishkopf, and Rosenblith (1958) recorded short latency responses to auditory clicks at the inion, these responses were thought to be of cortical origin. Bickford, Jacobson, and Cody (1964) describe the characteristics of averaged inion responses to clicks and concluded that the responses were vestibular in origin. Cody and Bickford (1969), and Townsend and Cody (1971) provided further evidence suggesting that these responses arouse from activation of the vestibular end organ, specifically the saccule.

In 1994, Colebatch, Halmagyi, and Skuse established a reliable procedure to record the myogenic potentials evoked by the clicks. These authors revised previous recording procedures by putting surface electrodes on the sternocleidomastoid (SCM) muscles, rather than placing them at the inoin.

Normal VEMP responses are characterized by biphasic (positive – negative) waves. In a majority of studies, the peaks and troughs are usually labeled with the mean latency in milliseconds preceded by the lower case letters 'p' (for positive) or 'n ' (for negative), as proposed by Yoshie and Okodaira (1969) to distinguish them from neurally generated evoked potentials. The first positive- negative complex is often labeled as p13-

n23. Robertson and Ireland (1995) found the second wave complex (n34-p44) to be present in 68% of their participants.

The VEMP amplitudes are large and vary from a few microvolts, depending on the muscle tension and the intensity of stimuli. (Cheng & Murofushi, 2001a, 2001b; Colebatch, Halmaygi, & Skuse 1994; Li, Houlden, & Tomlinson, 1999, Ochi, Ohashi, & Nishino, 2001, Pyykko, Aalto, Gronfors, Starck, and Ishizaki, 1995; Version, Colnaghi, Callieco, & Cosi, 2001; Wu & Murofushi, 1999; Wu, Young & Murofushi, 1999).

Dizziness was defined as a subjective complaint encompassing a variety of sensation, including a sense of rotation or motion of the surrounding environments (classic vertigo), a sense of spinning inside one's body (internal vertigo), impending fainting (presyncopal lightheadedness) or imbalance, disequilibrium or related symptoms (Ex. Vague lightheadedness', giddiness, swimming sensation) (Sloane, Hartman, & Mitchell, 1994). A few people describe their balance problem by using the word vertigo, which comes from the Latin verb "to turn". They often say that they or their surrounding are turning or spinning. Vertigo is frequently due to an inner ear problem.

Heide, Freitage, Wollenberg, Iro, Schimrigk, and Dillmann (1999) recorded clickevoked myogenic potential (CEMP) in 40 patients with acute vertigo of vestibular origin and the results compared with standard caloric reaction (CR). In comparison with CR, CEMP showed a sensitivity of 59% and specificity 100% for peripheral vestibular disorders. Different results of CR and CEMP may be due to this difference between target organs stimulated and may be important for prognostic value. Ribeiro, Almeida, Caovilla, and Gananca (2005) investigated that VEMPs were altered in 35% of the affected ears and in 25% of the asymptomatic ears in Meniere's disease. Study concluded that VEMPs could present abnormalities in the affected and asymptomatic ears in patients with diagnosis of unilaterally defined Meniere's disease. VEMPs in patients with meiners disease and reported that 54% of the patients had no VEMPs when clicks were used as stimuli. (De Waele, Huy, Diard, Freyss, & Vidal 1999). In patients with superior canal dehiscence (SCD) VEMPs showed abnormally large responses with low thresholds, particularly in the frequency range of 500-1000Hz on the affected side. (Brantberg, Bergenius & Tribukait 1999). Abnormal VEMPs were found in 2 patients with vestibular schwannoma while ABR data were normal (Matsuzaki, Murofushi, and Mizuno, 1999). Hence, the VEMP has the clinical potential to differentiate different vestibular pathology.

#### **Purpose of the study:**

The purpose of this study was to establish the norms for two age group 21-30 and 31-40 years with two stimuli, click and short duration tone burst, and to observe how the VEMP responses related to different type or symptoms related to dizziness.

#### Need of the study:

- It has been reported in literature that VEMP can be false positive or false negative. Click sensitive vestibular hair cell might differ from short duration tone burst (STB) sensitive vestibular hair cell. Therefore, it is better to use two kinds of stimuli to confirm the result of sound evoked potential on SCM (Murofushi, Matsuzaki, & Chih-Hsiu; 1999).
- 2. VEMP is the recent tool in the field of Audiology to find out the cause for vertigo. However, there are a few studies on this to know the effectiveness of VEMP to identify the lesion. Hence, many more such findings in different clinical population might highlight its effectiveness. It is also necessary to have norms to compare the results obtained in clinical population. So there is need to establish the norms for two stimuli click and short duration tone burst, to know which should be the better stimuli to evoked VEMP response.
- Cheng, Huang and Young (2003) reported click stimulation produces 98% VEMPs response, where as 88% revealed positive short tone burst-evoked VEMPs. In contrast Murofushi, Matsuzaki, and Wu (1999) reported that in

subject with vestibulocochlear disorders revealing same VEMP response in 88% of the subjects. So there is need to check the specificity of VEMP response.

- 4. Muscle artifact can contaminate the VEMP response. However, latency of muscle artifact is approximately 50 msec, which is different from VEMP latency. However, one must clear how frequent one can set false positive response and ways to avoid or cross check the false positive response.
- 5. There is no study, which finds one to one correlation between the type or symptoms related to dizziness and VEMP response. Thus, an attempt has to be made to find out the relationship between VEPM response and type of symptoms related to dizziness.
- 6. VEMP provide important diagnostic information as set the functioning of vestibular system can be simultaneously recorded without additional preparing on whom ABR is administered (Debatisse, Pralong, Guerit, and Bisdorff, 2005). Hence, more extensive such studies are required in clinical population.

### Aim of the study:

Aim of the study is to find out:

- 1. Age related changes of VEMP response in normal-hearing subjects without dizziness.
- 2. Effect of Intensity on VEMPs response in healthier subjects and subjects with dizziness for short duration tone burst (STB).
- 3. Comparison of VEMP response obtained in control and experimental group.
- 4. Specificity of VEMP responses in subject with normal hearing without dizziness.
- 5. The present study also aims to investigate the association of VEMP responses with different symptoms of dizziness.

# **REVIEW OF LITERATURE**

Vestibular evoked myogenic potentials (VEMP) are responses from the otolithic organs the saccule and the utricle to the high intensity sound stimulation. These responses can be acquired from the anterior neck muscles, specifically from the sternocleidomastoid (SCM) muscles. There is initially biphasic positive response (p13-n23) recorded from the averaged EMG which occurs at short latency and ipsilateral to the stimulated ear (Colebatch, Halmagyi & Skuse, 1994)

VEMP has been used as a clinical tool, which provides additional information about disturbances of vestibular function as a result of their dependence upon different vestibular receptors.

- 1. Neck muscles via the medial vestibulospinal tract (MVST).
- The leg muscles via the lateral vestibulospinal tract (LVST). (Colebatch & Halmagyi 1994; Wilson & Boyle, 1995; Murofushi, Halmagyi, Yavor, & Colebatch, 1996; Uchino & Sato, 1997; Kushiro & Zakir, 2000)

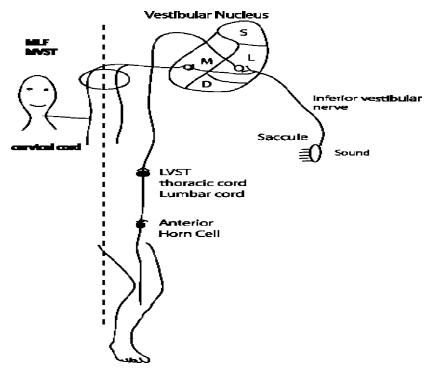


Figure: -1 showing pathway of Vestibular Evoked Myogenic Potential.

VEMP which arises from tonic contraction of the muscle (Welgampola & Colebatch, 2005) are proposed to be generated via a dysynaptic pathway, beginning in the saccular macula to the inferior vestibular nerve, lateral vestibular nucleus, medial vestibulospinal tract, and finally to the motor neurons of the SCM muscle (Kushiro, Zakir, & Ogawa, 1999; Uchino, Sato, & Sasaki, 1997).

#### **Recording methods of vemps:**

#### VEMPs with clicks and short tone burst:

Intense clicks of about 95 to 100dBnHL above normal hearing level are required to evoke VEMPs and are at the limit of what is considered safe but are generally well tolerated. Stimuli of 95dBnHL and 0.1-millisecond duration are used in routine clinical tests. The presence of tinnitus is a relative contradiction to click and tone burst VEMP testing and an alternate stimulus should be considered in this condition. An intact middle ear conductive apparatus is needed to convey the click to the end organ and the response is abolished or attenuated in conductive hearing loss with air-bone gaps as small as 8.75dBHL. (Bath, Harris, & Ewan, 1999)

The amplitude of the p13-n23 response is largely determined by click intensity and the level of tonic SCM contraction (Lim, Clouston, Sheean, & Yannikas, 1995). Ideally rectified EMG is averaged or alternatively feedback given to subjects to control for the levels of tonic muscle activation. Subjects must activate their SCMs, for example by lying semi-recumbent and lifting their heads, so that adequate levels of tonic neck activation are maintained during the recording (correspondingly to a mean rectified EMG of about  $60\mu\nu$ ). Alternate methods of bilateral activation by pushing the head forward against the resistance of a padded bar while sitting upright, causing isometric contraction of both SCMs, are less comfortable and cannot be sustained for prolonged periods. Unilateral activation by rotating the head against resistance permits recording from only a single SCM.

#### VEMPs evoked by bone- conducted stimuli:

Skull taps and bone-conducted tones are stimuli that by pass the middle ear. A forehead tap delivered at  $F_{pz}$  (international 10-20 system) via a tendon hammer, evokes a vestibular dependent short latency p13n23 response in both SCMs. The tap also evokes a second negativity (n2), which can sometimes be difficult to separate from n13 and thus produces unambiguous analysis in some normal subjects. (Halmagyi, Yavor, & Colebatch, 1995).

Tap-evoked VEMPs owing to the magnitude of the stimulus are 1.5 to 3 times as large as those evoked by clicks. These are relatively preserved in older subjects in whom stimulus thresholds are likely to be high (Welgampola & Calebatch, 2001).

A bone- conducted tone burst delivered over the mastoid process via a B71 clinical vibrator (radio ear corporation, Philadelphia, PA), routinely used in audiometric testing, evokes VEMPs despite conductive hearing losses (Sheykholeslami, Murofushi, Kermany, & Kara, 2000; Welgampola. Rosengren, Halmagyi, & Colebatch, 2003). Optimum stimulation is delivered with the conductor placed 3 x 2cm posteriosuperior to the external acoustic meats, using frequencies of 200 to 250 Hz (Welgampola, Rosengren, Halmagyi, & Colebatch 2003; Sheykholeslami, Murofushi, Kermany, & Kaga, 2000). VEMPs are often bilateral as the stimulus is transmitted via bone and activate end organs on both sides. The ipsilateral VEMP is about 1.5 times larger and occurs approximately 1 millisecond earlier. Rarely larger responses have been recorded contra lateral to the stimulated ear.

#### VEMPs evoked by galvanic simulation:

A short duration (2 millisecond) pulsed current delivered via electrodes attached to the mastoid processes evokes a p13n23 response on the side ipsilateral to cathodal stimulation. Similar to that evoked by sound stimuli of 4mA/2msec as used for clinical testing are well tolerated by patients. Such a current in close proximately to the recording

site causes a large stimulus artifact and specific subtraction techniques are required to recover the response of interest (Watson & Colebatch 1998). DC stimulation acts proximal to the end organ at the terminal part of the primary vestibular afferent, the spike trigger zone. Cathode currents increase and anodal currents decrease spontaneous firing rates (Goldberg, Fernandez, & Smith, 1982). A monaural cathodal stimulus evokes an ipsilateral p13n23 response and a contra lateral n12p20 crossed neural response in all subjects. The greater frequency of a crossed neural response, when compared with clicks may indicate activation of utricular afferents.

This technique should help distinguish between end organ (labyrinthine lesions and more proximal (retrolabrinthine) lesions. In confirmation of this all 10 subjects with Meniere's disease and endolymphatichydrops had preserved galvanic VEMPs. Where as 16 of 18 subjects with cerebellopontine angle tumors had reduced or absent VEMP responses (Murofushi, Takegoshi, Ohki, & Ozeki, 2002).

#### **Stimulus related factor:**

*Frequency Effect:* 

Murofushi, Matsuzaki, and Chih-Hsiu (1999) observed larger VEMP amplitudes with 500Hz tone bursts than with 1000Hz and 2000Hz tone bursts. Welgampola and Colebatch (2001) reported largest VEMP amplitudes at 500Hz and 1000Hz. Todd, Cody, and Banks (2000) recorded VEMPs with frequencies ranging from 100 to 3200Hz and demonstrated that the VEMP has well defined frequency tuning with a maximum response amplitude ranging from 200 to 400Hz, although stimulus frequencies between 400 and 800Hz were not used. The VEMP frequency response in humans is consistent with neurophysiological findings in cats that show the acoustically responsible afferent fibers in the inferior vestibular nerve have broad, V-shaped tuning curves with frequencies between 500 and 1000Hz (McCue & Guinan, 1995).

Tone-evoked VEMP amplitudes were larger than click evoked amplitudes when comparisons were made at equal peak SPLs. The magnitude of the amplitude differences between tones evoked and clicks evoked VEMPs increased as tone burst frequency decreased. The observed amplitude differences however may be due to differences in stimulus spectrum level. When comparisons are made at equal peak SPLs, the click has a lower spectrum level than the tone bursts due to its wider bandwidth (Akin, Murnane, & Profitt, 2003).

Welgampola and Colebatch (2001) had reported no significant effect of stimulus frequency on VEMP latency. Akin et al. (2003) reported that VEMP latency decreases as the stimulus frequency increases. Murofushi et al. (1999) had used 0.5, 1 and 2 KHz short tone bursts to evoke the VEMPs. Short tone bursts of 0.5 KHz evoked the largest responses while STB (short tone burst) of 2 KHz evoked the smallest.

Cheng, Huang, and Young (2003) had investigated click VEMPs and found that they had a higher response rate, shorter latency and larger amplitude than STB VEMPs. These findings suggest that click is superior to short tone burst to trigger VEMPs. Because click VEMPs have a shorter p<sup>13</sup>latency than STB-VEMPs it is possibly because the clicks reach maximum sound intensity earlier than STBs by 1m sec.

### Intensity:

VEMP amplitude increased as a function click level increased (Akin et al 2003). Colebatch et al (1994); Lim et al. (1995). Ochi, Ohashi, and Nishino (2001) had also reported that as the level of the click and tone burst increased there was a corresponding increase in level of the VEMP amplitude, However VEMP latency did not vary as a function of click level.

#### Effect of stimuli duration:

Huang, Su, and Cheng (2005) had investigated effect of click duration on VEMPs in 18 healthy volunteers (10males, 8 females) age range 22-40 years. Four click durations (0.1, 0.2, 0.5, and 1.0ms) were used in a random order to elicit VEMP responses (0.1-, 0.2-, 0.5- and 1.0- VEMP respectively). Click stimulation of 34 ears (94%) produced 0.1 VEMP responses, whereas positive 0.2, 0.5, and 1.0 VEMP responses observed in 36 ears (100%). The latencies of peaks p13and n23 were significantly prolonged between successive stimulus durations from 0.1 to 1.0ms, in contrast to the p13-n23 intervals. The relative amplitude was significantly increased between successive durations from 0.1 to 0.5ms, but there was no significant difference between 0.5 and 0.1ms. Hence 0.5 VEMP demonstrates more prominent waveform morphology than either the 0.1 or 0.2 VEMPs.

Four kinds of rise/fall time STB stimulation patterns in a random order were used to test 22 ears using changing rise/fall times (0.3, 1.3 and 10ms). VEMP responses (p13/n23) triggered by these patterns were clearly observed in all 22 ears. When the rise/fall time was prolonged from 0.3 to 10ms, the p13 latency was prolonged in parallel. There was a similar trend for the n23 latency, although a significant difference was not attained between 0.3 ms and 1ms rise/fall times. Considering the p13 and n23 latencies for the 4 rise/fall times, the variances were smallest for the 1msec stimulation, meaning that it caused the smallest inter aural latency differences. The amplitude or relative amplitude in the individual ear tested was lowest for the 10ms stimulation, being comparable among the other 3 rise/fall times. So the 1msec rise/fall time was a remarkable stimulation pattern because its VEMP responses were simultaneously more constant and conspicuous (Cheng and Murofushi, 2001).

Cheng and Murofushi (2001) inspected four different plateau times (1, 2, 5 and 10msec) were used in a random order to test 26 normal ears. VEMP responses (p13/n23) triggered by the tone bursts were clearly observed in all ears. When the plateau time was increased in order from 1 to 10msec, the latencies p13, n23) and interval (p13, n23) were also increased in parallel although significant differences were not observed between some plateau times. The amplitude or relative amplitude in individual ears was lowest for the 1msec plateau time, while it was comparable for the other three plateau times. So ideally they had recommended 1 msec rise/fall time and plateau time 2msec wee evoking more constant VEMP responses.

#### Repetition Rate:

Wu and Murofushi (1999) analyzed effect on VEMPs at five different click stimulation rates (1Hz, 5Hz, 10Hz, 15Hz & 20Hz) in random order. VEMP responses were apparent in all 24 ears stimulated with 1Hz, 5Hz, and 10Hz. One ear was void of response at 15Hz stimulation and 9 ears at 20Hz stimulation. The relative amplitude or the rank of amplitude in individual ears was higher at 1Hz and 5Hz stimulation; progressively decreasing as the stimulation rate increased. Comparisons of p13 & n23 latencies showed no difference among five stimulation rates; but variance was greatest at 20Hz stimulation and smallest at 1Hz VEMPs generated. At lower stimulation rates VEMP seemed to be more marked and constant.

Ozeki, Iwasaki, and Murofushi (2005) had evaluated the influence of stimulation rate of galvanic on the galvanic evoked vestibulocollic reflexes and to propose the optimal stimulation rate for clinical use. Both ears of 30 healthy adults were tested at 5 different galvanic stimulation rates (1, 3, 5, 7 and 9Hz) in a random order. Responses were evident in all 60 ears only at 5 Hz; some ears showed no response at the repetition rate. The relative amplitudes in individual ears were higher at 1, 3, 5 Hz than at 7 & 9Hz. Comparison of the latencies of p13g and n23g showed no significant difference among the five stimulation rates.

#### Stimulation mode:

Wang and Young (2003) compared binaural acoustic stimulation (B-VEMP) versus monaural acoustic stimulation (M-VEMP) in 17 healthy volunteers. 14 subjects demonstrated both B-VEMPs and M-VEMPs without significant difference in the latencies of p13 and n23. When using interaural amplitude difference (IAD) ratio for interpreting amplitude, B-VEMPs can produce information equivalent to M-VEMPs in terms of response rate, latencies and IAD ratio in healthy subjects. So B-VEMPs provide neither different information nor less variability, as compared with M-VEMPs. In addition B-VEMPs can offer information on unilateral inner ear (saccular) pathology

similar to that by M-VEMPs and had suggested that bilateral stimulation might be better option when testing old or disable patients.

There was no significant left-right difference in amplitude under binaural stimulation, while binaural stimulation tends to produce greater amplitude when compared to monaural stimulation (Ferber-Viart, Duclaux, Colleaux, & Dubreuil, 1997).

#### Subject related factor:

Effect of age on VEMPs:

Su, Haung, Young, and Cheng. (2004) had investigated affect of age on VEMPs. Group I included patients aged<20years, Group II subjects age ranged from 21 to 40 years, Group III subjects were 41 to 60 years and group IV included subjects older than 60 years. Results showed that VEMP response rate from groups I to IV was 98%, 96%, 90%, and 60% respectively, with a significant difference only between group IV and other groups. The amplitude was negatively correlated with age in contrast to the n23 latency, correlating positively with age; both reaching a significant difference. Although the p13 latency had a trend to prolong as age increased, no significant correlation was noticed with age. Hence as age increased over 60 years, the VEMP response rate decreased dramatically, while age increased, the VEMP amplitude decreased in comparison to n23 latency prolonged. These findings might suggest that aging could deteriorate the saccular and corresponding neural functions.

Sheykholesami et al. (2005) had recorded VEMPs induced by air and bone conducted auditory stimuli were recorded from the sternocleidomastoid muscles of 12 normal neonates and 12 neonates with various clinical findings (bilateral Artesia of EAC, Treacher Collins syndrome etc.). They showed that with the exception of one patient with hearing loss, reproducible biphasic VEMPs were recorded with the short tone burst sounds. The overall morphology of the neonatal VEMPs is quite similar to that of adults. The major neonatal differences are shorter latency of the n23 peak and of higher amplitude variability.

The click and galvanic evoked responses were present bilaterally in all subjects below 60 years of age. Average click evoked response amplitude decreased with age, with a pronounced decline of 25-30% per decade from the 6<sup>th</sup> decade. The average click thresholds increased from 85dBnHL in the third decade to 96.5dBnHL in the 8<sup>th</sup> and 9<sup>th</sup> decade for average galvanic evoked Vestibulocollic reflex (VCR) amplitude which decreased sharply from the seventh decade. Tap evoked reflex amplitudes showed a milder decrease. When side to side differences in amplitude were expressed as asymmetry ratios (AR) in subjects below the age of 60 years, values of up to 35 and 46% were obtained for click amplitudes correlated and un corrected tap response amplitudes, and up to 41 and 55% for corrected and uncorrected galvanic evoked responses. In conclusions click and galvanic evoked VCR amplitudes decrease rapidly there after while tap evoked responses are less affected. These changes are probably due to morphological changes in the vestibular system occurring with aging (Welgampola and Colebatch, 2001).

Age related morphological changes affecting the vestibular system from the end organs to the central nuclei are well documented. The vestibular epithelium shows hair cells loss of 6% per decade between the ages of 40 and 90 years (Rosenhall, 1973).

Bergstrom (1973) reported a decrease in the number of vestibular nerve fibers by 3.5% per decade over a similar age range. A decrease with age in the number of cell bodies in scarpa's ganglion (Richter, 1980) and loss of neurons in the vestibular nuclear complex (Lopez et al., 1997) have also been reported. Johnson and Hawkins (1972) reported a loss of otoconia from the age of 30 onwards, affecting the saccule more severely than the utricle.

#### Effect of muscle tension:

Bickford et al. (1964) noticed that muscle tension was involved in the presence of the response. Increased tension in the neck muscle produced increases in amplitudes; while the intensity of stimuli remained unchanged. Colebatch et al. (1994) monitored electromyography (EMG) activity with an oscilloscope and quantified the activity with mathematical analysis. In all their participants, there was a linear relationship between the amplitude of the response and the mean level of EMG activity. This finding was confirmed by later studies and is considered as one of the unique features of VEMPs.

Many procedures have been used to activate the neck muscle. Bickford et al. (1964) applied different loads to a plastic loop and pulley that changed the traction of neck muscle. Colebatch et al. (1994) asked their participants to press against a padded bar to activate SCM muscles.

In studies by Welgampola and Colebatch (2001) and Robertson and Ireland (1995) participants were placed in a supine position and asked to elevate their head to activate the SCM muscles on both sides simultaneously. Ochi et al. (2001) & Todd et al. (2000) used a sitting position and instructed participants to turn their head away from the stimulated ear. Although different procedures have been used to activate the neck muscles, the published studies provide consistent findings that the muscle tension not the head position itself, influences the presence and the amplitude of the response (Colebatch et al 1994; Ferber-viart, Dubreuil, & Duclaux 1999; Li et al., 1999; Ochi et al., 2001).

#### Response Laterality:

Bickford et al (1964) and Townsend and Cody (1971), symmetric responses from both sides were reported. In contrast, Colebatch et al (1994) reported that the response was always larger on the ipsilateral SCM muscles when monaural stimuli were presented. Robertson and Ireland (1995) also studied the laterality at VEMPs. They obtained symmetric responses from SCM muscles to clicks presented unilaterally to 7 normal participants. Ferber-Viart et al. (1997) demonstrated that responses tended to be greater in SCM muscles contra lateral to the side of stimulation. While these studies indicate that VEMPs are bilateral in nature. Li, Houlden, and Tomlinson (1999) concluded that the response was purely ipsilateral. Akin and Murnane (2001) also demonstrated that VEMPs were ipsilateral responses to the stimulation side.

#### **VEMPs in clinical population:**

Vestibular neuritis and differential diagnosis:

The peripheral vestibular system includes five end organs: three (lateral, superior and posterior) semicircular canals, the saccule and the utricle. The canals respond to angular acceleration and the otoliths (the saccule and the utricle) sense linear acceleration. The lateral and superior canals are innervated by the superior vestibular nerve, while the posterior canal is innervated by the inferior vestibular nerve. Moreover the macula of the utricle is innervated by the superior vestibular nerve and the majority of the macula of the saccule is innervated by the inferior vestibular nerve. Distribution of any one or more of these organs could lead to dizziness or vertigo. The pathologies rebated to the abnormal vestibular dysfunction are sometimes difficult to identify due to the insensitivity of current diagnostic tools. Although caloric test is helpful to identify dysfunction of the lateral semicircular canal, easy and reliable procedures to evaluate other peripheral vestibular organs are needed. (Zhou & Cox, 2004).

Heide, Freitage, Wollenberg, Iro, Schimrigk, and Dillman (1999) investigated VEMPs in the differential diagnosis of actuate vertigo. These authors evaluated those patients who had normal VEMPs with abnormal caloric tests and their study yielded the following findings:

- (a) All patients with BPPV had normal VEMPs
- (b) All patients with psychogenic vertigo had normal VEMPs.

- (c) In the 17 patients who had abnormal VEMPs, 5 had no VEMPs in either ear, while caloric testing revealed only unilateral loss.
- (d) More than 5 weeks after the onset of vertigo VEMPs had reappeared in 2 patients with acute vestibulopathy, while in the patients who lost vestibular function due to trauma, VEMPs had not returned more than 9 months after accident.

From this above results suggests that VEMP testing was useful in the diagnosis of acute vertigo regarding the location and nature of the disorder.

Murofushi, Halmagi, Yavor, and Colebatch (1996) had taken 47 patients (34 men and 13 women) with acute vestibular neurolabyrinthitis, 10 of whom had then developed posterior semicircular canal type BPPV. Results show that first positive - negative peak of amplitude was ipsilaterally present on stimulation of the unaffected side in all patients. It was absent on the affected side in 16 patients (34%). The absence or presence of P13n23 was independent of the results of caloric tests, pure tone audiometry and auditory brain stem responses. Typically posterior semicircular canal BPPV developed in 10 of the 47 patients after the acute attack of vestibular neurolabyrinthitis always on the same side as the neurolabyrinthitis. The p13n23 potentials were preserved or stimulation of the affected ear in all 10 patients with BPPV.

Halmagyi and Colebatch, (1995) reported in their study that patients who did not have caloric responses on the affected sides indicating dysfunction of the lateral semicircular canal. Result showed VEMPs were normal in 6 patients, reduced in 5 patients and absent in 11 patients. So results not only suggested that VEMPs were not of lateral canal origin but also revealed different pathologies involved in vestibular neuritis.

Halmagyi, Aw, Karlberg, Curthoys, and Todd (2002) had reported 2 patients with acute vertigo but normal lateral semicircular canal function as indicated by the caloric test. It was reported that these 2 patients had selective inferior vestibular neuritis since VEMPs were absent on the affected side for both cases.

#### Vestibular Hypersensitivity disorder:

Colebatch, Rothwell, Bronstein, and Ludman (1994) studied VEMPs in a patient with unilateral tullio phenomenon. They found that the responses elicited from the symptomatic side were larger in amplitude and had abnormally low thresholds but retained normal waveform configuration. They concluded that VEMPs were indicative of a pathological increase in the normal vestibular sensitivity to sound.

Study on VEMPs and high resolution CT on 4 patients with the tullio phenomenon indicated that the threshold of click evoked VEMPs were low for all affected ears (fair at 65dB, one at 35dBnHL) and normal (70-90dBnHL) for three unaffected ears. (Watson, Halmagyi, & Colebatch, 2000)

In 3 patients with superior canal dehiscence (SCD) VEMPs showed abnormally large responses with low thresholds, particularly in the frequency range of 500-1000Hz on the affected side. (Brantberg, Bergenius, & Tribukait 1999). Brantberg, Bergenius, Mendel, Witt, tribukait, and Yagg (2001) studied 8 patients with SCD. In all patients, VEMPs were present with extremely low thresholds and abnormally large amplitudes on the affected side. In contrast, 4 of the 8 patients have normal hearing and 6 patients had normal findings with caloric test.

Streubel, Cremer, Carey, Weg, and Minor (2001) evaluated 10 patients with SCD in 8 patients without prior middle ear disease, the VEMP threshold from the affected side was  $72 \pm 8$ dBnHL, compared to the threshold from normal participants of  $96 \pm 4$ dBnHL. In the 2 remaining patients with conductive hearing loss VEMPs were present from the affected side. Given that VEMPs should not be expected in ears with conductive hearing loss. The Streubel et al., (2001) findings are compelling with regard to the sensitivity of VEMPs in diagnosing SCD in a variety of different hearing conditions.

#### Vestibular schwannoma:

Murofushi, Matsuzaki, and mizuno (1998) reported abnormal VEMPs in 80% of 17 patients with vestibular schwannoma. 15 out of the 17 had no VEMPs, while the remaining 2 had significantly decreased amplitudes. Abnormal VEMPs were found in 2 patients with vestibular schwannoma while ABR data were normal. (Matsuzaki, Murofushi, & Mizuno, 1999). Ochi et al. (2001) also reported 3 vestibular schwannoma cases with abnormal interval differences of thresholds and abnormal P13n23 amplitude ratios between left and right sides.

VEMP results were not always correlated with the nerve where the tumor was located. Moreover no correlation was found between the VEMPs and tumor size (Tsutsumi, Tsunoda, Noguchi, & Komatsuzuki, 2000).

#### Meniere's disease (Endolymphatic hydrops):

Robertson and Ireland (1995) reported that VEMPs were absent in all 3 of their patients with Meniere's disease (MD). VEMPs in patients with MD showed that 54% of the patients had no VEMPs when clicks were used as stimuli (De Waele, Huy, Diard, Freyss, & Vidal 1999). Shojaku, Takemori, Kobayashi, and Watanabe (2001) reported similar results in which 8 out of 15 patients with MD had abnormal VEMP amplitude.

Ohki, Matsuzaki, Sugasawa, and Murofushi (2002) reported a very interesting finding: absence of or abnormal VEMPs in contra lateral ears that may have delayed endolymphatic hydrops.

VEMPs were altered in 35% of the affected ears and in 25% of the asymptomatic ears. The alterations were absence of responses in 7 cases, and increase in interaural amplitude difference ratios in one case. From this study concluded that VEMPs could present abnormalities in the affected and asymptomatic ears in patients with unilaterally defined MD (Ribeiro, Almeida, Caovilla, & Gananca, 2005).

Seo, Node, Yukimasa, and Sakagami (2003) had investigated whether endolymphatic hydrops in Meniere's disease could be diagnosed comparing VEMP before and after furosomide administration (F-VEMP). Results showed that the amplitude of the p13-n23 biphasic loading. So F-VEMP test may be useful in the diagnosis of endolymphatic hydrops.

Murofushi, Matsuzuki, and Takegoshi (2001) had taken 6 normal volunteers and 17 patients with unilateral Meniere's disease. VEMPs were recorded before and after administration of glycerol (1.3 g/kg body weight). He concluded VEMPs in some patients with unilateral Meniere's disease were improved by oral administration of glycerol. This result suggests that abnormal VEMPs in patients with unilateral Meniere's disease could result from endolymphatic hydrops.

Interaural amplitude difference (IAD) ratio of VEMPs correlates with the stage of Meniere's disease and can be used as another aid to assess the stage of Meniere's disease. (Young, Huang, and Cheng, 2003)

Murofushi, Shimizu, Takegoshi, and Cheng (2001) had taken 134 patients (61 men and 73 women aged 0-75 years). Of whom, 43 patients with Meniere's disease, 62 acoustic neuroma patients, 23 vestibular neuritis and 6 multiple sclerosis and also 18 healthy volunteers (13 men 65 women aged 25-38 years) were enrolled. Results showed VEMPs were absent or decreased in 51% of patients with Meniere's disease (n=22); 39 with vestibular neuritis (n = 9), 77% with acoustic neuroma (n=28), and 25 % with multiple sclerosis (3 of 12 sides of 6 patients). Concerning prolonged latencies of VEMP suggest lesions in the retro labyrinthine especially in the vestibulo spinal tract.

#### Multiple sclerosis:

The latencies of a vestibulospinal reflex can be prolonged in multiple sclerosis (MS). The VEMP delay could be attributed to demyelination either of primary afferent axons at the root entry zone or secondary vestibulo spinal tract axons rather than to lesion involving vestibular nucleus. Measurement of VEMPs could be helpful in detecting sub clinical vestibulospinal lesions in suspected multiple sclerosis (Shimizu, Murofushi, & Sakurai, 2000).

Alpini, Pugnetti, Caputo, Cornedio, Capobianco, and Cesarani (2004) took 40 patients who were diagnosed with multiple sclerosis and they suggested, the abnormal VEMPs indicated brainstem dysfunction in 4 patients (10%) with normal MRI and no specific clinical signs. So VEMPs is also important in diagnosing the multiple sclerosis.

#### Spinocerebellar degeneration:

Takegoshi and Murafushi (2000) had recorded VEMPs in 16 patients with spinocerebellar degeneration including olivo-ponto-cerebellar ataxia (OPCA) in 10, cortical cerebellar atrophy (CCP) in 3 and machado-joseph disease (MJD) in 3. The results revealed VEMPs in patients with OPCA and CCA types of spinocerebellar degeneration were definitely abnormal in patients with MJD. It has been reported that the peripheral nervous system is frequently affected in patients with MJD. It is more likely that the losses of the vestibulo colic and vestibulo ocular reflex in MJD. It seems more likely that the losses of the vestibulo-colic and vestibulo-ocular reflex in MJD patients might be due to the degeneration of the peripheral vestibular system.

#### Conductive hearing loss:

Interference of sound transmission due to some disorders such as chronic otitis media (COM) may lead to absent VEMPs (Young, Wu & Wu 2002). Tone stimuli rarely elicit VEMP responses in patients with conductive hearing loss (Halmagyi, Calebatch, & Curthoys, 1994).

Yang and young (2003) had compared the tone burst and tapping evocation of myogenic potentials in patients with chronic otitis media have taken 22 ears with conductive hearing loss due to chronic otitis media. Results showed that 13 (59%) of the 22 ears showed positive VEMPs using the tone burst method whereas 20 ears (91%) displayed positive VEMPs by the tapping method. So they concluded that while stimulating, sound is attenuated by middle ear pathology, VEMPs are expected to be poorly elicited under such conditions. Myogenic potentials may be evoked with the tapping method to elicit the absent VEMPs that results from middle ear or inner ear pathology.

Bath, Harris, Ewan, and Yardely (1999) found click evoked, restores present in less than 10% of their group of patients with conductive hearing loss, compared with 97% of those without conductive hearing loss.

As conduction across the middle ears ossicular chain is defective, VEMPs are attenuated or absent in subjects with Otosclerosis (Halmagyi, Calebatch, and Corthoys, 1994; Ochi, Ohashi, & Kinoshita 2002). Attenuation of the VEMP occurs early; therefore, apparent conductive hearing loss without abolition of VEMPs warrants careful investigation for an alternate diagnosis such as SCD with enhanced bone conduction VEMP testing may be potential value in monitoring the efficiency of stakes mobilization procedures.

#### Sudden deafness:

VEMPs were evoked by short tone burst, in 20 patients with unilateral idiopathic sudden deafness (Wu & Young, 2002). The results of the deaf ears were compared with those of the contra lateral healthy ears and the normal control ears. The relations between VEMPs and the hearing level or caloric response were then investigated. All the twenty deaf ears displayed normal biphasic VEMPs. The mean latency of P<sup>13</sup> and n<sup>23</sup>, as well as mean amplitude was not significantly different of either the contra lateral healthy ears or the normal central ears. Neither the hearing level nor the caloric response correlated to the VEMPs. In other words, the transudation pathway of the VEMPs is unrelated to the hearing level.

#### Gentamycin therapy:

DeWaele, Meguenni, and Freyss (2002) found that the VEMPs can be used to monitor the effects of low close intra tympanic gentamycin injections used to achieve chemical labyrinthectomy, a procedure used to control debilitating vertigo in Meniere's disease and other peripheral vestibulopathies.

### Auditory Neuropathy:

Sheykholeslami, Kaga, Murofushi, and Hughes (2000) had taken 3 auditory neuropathy patients. These patients also complained of balance disorders. Tests of battery were administered, audiometric tests (pure-tone audiometry and speech discrimination tests), Otoacoustic emissions, auditory-evoked brainstem responses and vestibular function tests (clinical tests of balance, electronystagmography, damped rotation tests and VEMPs). They concluded that, in patients with isolated auditory neuropathy, the vestibular branch of the 8<sup>th</sup> cranial nerve and its innervated structures may also be affected. Thus they had also suggested that use of the term "cochlear neuropathy" to characterize those patients with involvement of only the auditory branch of the VIIIth cranial nerve and its innervations.

Sheykholeslami, Schmerber, Kermeny, and Kaga (2005) had recorded VEMP using tone burst in a case with bilateral auditory neuropathy (AN). There were no response on left ear stimulation and a biphasic response with normal latency and amplitude on right-ear stimulation.

So above review suggested that the VEMP has the potential to and the intermedian to audiologist to identify the pathological condition in the vestibular system associating with different disorder. VEMP results are also more levels to alter by several factors. Thus, it is essential to carry out intense study in different clinical population and using different stimuli to understand the physiological aspects of VEMP and also to identify the lesion the vestibular system.

# **METHOD**

The present study aimed at investigating vestibular evoked myogenic potential in individual with or without dizziness for both click and short duration tone burst.

## Subject:

The subjects were devided into two groups, control group and experimental group. Control group was devided in to two subgroups on the basis of age, Group A (21-30year) and Group B (31-40year). 30 subjects consist of (15 male & 15 female) were participated in each Group. Control group was devided in two groups because Johnson and Hawkins (1972) reported a loss of otoconia from the age of 30 onwards, affecting the saccule more severely than the utricle. The experimental group includes 25 subjects heaving age range from 20-40 years.

### **Group: Control group**

Selection criteria:

- 1. All the subjects had hearing sensitivity within 15dBHL at frequencies from 250 to 8000Hz.
- 2. 'A' type tympanogram with normal reflexes in both ears.
- 3. They did not have any history or presence of any otological problem (like ear discharge, ear ache etc)
- 4. No history or presence of neurological symptoms.
- 5. Uncomfortable levels (UCL) for speech for all the subjects were greater than 105dBHL.

#### GroupII: Experimental group

Selection criteria:

1. The subjects were having either normal hearing or sensory neural hearing loss with air-bone gap not exceeding 10dBHL.

- 2. Immittance measurements showed 'A' type tympanogram with presence, elevated or absence of reflexes in both ears.
- 3. None of them reported to have hyper or hypotension or spondylitis.
- 4. Did not have any evidence of space occupied lesion (decided based on auditory brainstem response results and neurologist report).
- 5. All the subjects had to have complain of dizziness.
- 6. They had UCL (uncomfortable level) greater than 105dBHL.

# Instrumentation:

- 1. Calibrated diagnostic audiometer was used to estimate the pure-tone threshold and UCL for speech for all the subjects for both air and bone conduction.
- 2. Calibrated middle ear analyzer GSI-Tympstar was used for tympanometry and reflexometry.
- 3. IHS Smart EP version: 3140 (Intelligent hearing systems, Florida, USA) was used to record and analyze ABRs and VEMP. Eartone 3A insert earphones were used to deliver the stimuli.

# **Test environment:**

All the tests were carried out in sound treated room.

# **Procedure:**

- Pure tone threshold was obtained using modified version of Hughson and westlak procedure (Carhart & Jerger, 1959, cited in silman &Silverman, 1991) across octave frequencies from 250 to 8000Hz for air conduction and from 250 to 4000Hz for bone conduction.
- To determine the uncomfortable loudness level (UCL) of the subjects, the speech material was presented through the headphone (TDH-39) at different intensities using ascending method. The UCL for speech was defined the

hearing level at which the subjects considers speech material to be uncomfortably loud.

- Tympanometry and Reflexometry were carried out using 226Hz probe tone to know the status of the middle ear for all subjects. The subject were made to sit comfortably and asked not to sallow during the testing period. Initially tympanometry and then acoustic reflex threshold were checked for all the subjects.
- A part of dizziness questioner a checklist described by Maryland Hearing and Balance centre were administered. It contains 6 sections. 2<sup>nd</sup> section questioner were administered which contains many symptoms associated with dizziness. The questionnaire is enclosed in appendix: 1.
- ABR recording: ABR was administered on experimental group to rule out space occupied lesion. Following parameter were used for ABR measurement.

Band pass filter	100-3000 Hz
Montage	Cz/A1 & Cz/A2
Notch	Off
Repetition rate	11.1 & 90.1
No. of channel	2
Gain	1,00000
Artifact	On
Stimulus	Click
Polarity	Rarefaction
Intensity	80 or 90dBnHL

Table 1: Shows the parameters used for ABR recording

Having inter-peak latency within normal range and good ABR morphology for both at 11.1 and 90.1 were considered as devoid of space occupied lesion.

VEMP recording: The subjects were seated upright position and instructed to turn their heads to opposite side of the test ear to activate unilaterally the sternocleidomastoid muscle (SCM). They were also asked to close their eyes. Instruction was given to avoid extraneous movements of head, neck and jaw while recording the VEMP. The button electrodes were used with the following electrode montage.

Electrode montage	➢ Non-inverting electrode (+): -mid
	point of the sternocleidomastoid
	muscle of the side being
	stimulated.
	<ul> <li>Inverting electrode (-): -</li> <li>sternoclavicular junction.</li> </ul>
	➢ Ground electrode: - forehead.

Table 2: Shown the Electrode montage for VEMP recording.

Before placing the electrodes, the sites were cleaned using skin preparation paste and electrodes were placed with the ten-20 conduction paste to increase the conductivity. The electrode impedance was checked and it was ensured that the impedance at each electrode site was less than 5 Kohm and inter electrode impedance was with in 3 Kohm.VEMPs was recorded in normal hearing subjects and subjects with symptoms of dizziness using the following parameters:

Analysis time	120msec
Filter setting	High pass: - 30Hz
	Low pass: - 1500Hz
Amplification	30,000
Type of stimulus	500Hz tone burst with 2-cycle rise/fall time
	and 0.1msec. Click.
Rate	5/sec
Polarity	Rarefaction
Total number of stimulus	250

Table 3: The parameters used to record VEMP

Two types of stimuli, short duration tone burst and click were used. Tone burst VEMPs were recorded at two intensity (99dBnHL & 105dBnHL) and 99dBnHL for click stimulus. VEMP was also recorded at 70dBnHL to check for muscle artifact. For each subject VEMP latency and peak-to-peak amplitude of p13 and n23 was recorded.

### Analysis:

The data collected were subjected to statistical analysis. Pair t-test was carried out to compare the latencies and peak to peak amplitude within group obtained at 2 different intensity level and independent t-test was carried out between control and experimental group to observe the significant difference if any and also between the two subgroup of control group to compare the latencies and peak to peak amplitude to see the age related changes.

### RESULTS

The objective of the present study was to observe age related changes in STB and click evoked VEMP responses and establish norms. Attempt was also made to investigate whether there is any difference in VEMP results obtained in individual with out dizziness and with dizziness and the relationship between the type of symptoms and VEMP results. Data obtained from both control and experimental group have been explained below.

### 1. Age related changes on VEMPs in subject with normal hearing without dizziness:

To investigate age related changes, two age groups (21-30 & 31-40years) population not having dizziness were taken. Evoked VEMP latency and peak to peak amplitude of p13 and n23 were noted for both tone burst and click. Independent sample t-test was administered to see the significant difference between the two groups for two different intensity levels (99dBnHL & 105 dBnHL) of STB in subject with normal hearing. It is evident in Table 4 that the p13 and n23 latency and peak-to-peak amplitude are approximately the same. The variability in latency and amplitude for both the peaks were also very negligible. Independent sample t-test revealed no significant difference in VEMPs responses for both latency and amplitude across the group except at the amplitude obtained at 105dBnHL for STB stimuli.

Intensity		Group	Mean	Std. Deviation	"t" value
105(STB)	P13	A	13.6893	1.0178	1.021
100(012)	1 10	В	13.4920	1.0281	1.021
99(STB)	P13	A	13.6759	1.0572	.982
00(012)	1 10	В	13.4725	1.1333	.002
99(C)	P13	A	11.4161	.9385	1.212
33(0)	110	В	11.6639	1.2091	1.212
105(STB)	N23	A	21.5821	1.8508	1.076
100(012)	1120	В	21.9779	2.0372	1.070
99(STB)	N23	A	21.1074	1.8578	1.191
00(012)	1120	В	21.5866	2.3694	1.101
99(C)	N23	A	18.9130	2.1468	1.261
00(0)	1120	В	19.4546	2.3929	1.201
105(STB)	PP	A	36.6729	18.8967	2.190*
100(012)		В	29.5971	15.0742	2.100
99(STB)	PP	A	26.8239	14.8326	.780
33(312)		В	24.5361	16.1985	
99(C)	PP	A	22.2064	11.8209	.428
33(0)		В	21.3050	10.4510	20

Table 4: Depicts the Mean, SD and "t" values of the VEMP responses (latency & peak to peak amplitude) in two age groups (21-30 & 31-40years) in normal hearing individual without dizziness.

\* Significant at the 0.05 levels

STB: short duration tone burst

C: click

P13 and n23: latency

PP: peak to peak amplitude.

Table 5: Depicts the Mean, SD and range of VEMP latency and peak-to-peak amplitude	
obtained in control group (Group A & Group B together).	

VEMP	Intensity (dBnHL)	Range	Mean	Std. Deviation
parameters				
P13	105(STB)	11.90-17.00	13.6020	1.0301
P13	99(STB)	11.20-17.40	13.5712	1.0913
P13	99(click)	9.80-15.20	11.5450	1.0758
N23	105(STB)	16.20-26.80	21.8014	1.9184
N23	99(STB)	16.20-26.00	21.3528	2.1244
N23	99(click)	14.00-27.00	19.2032	2.2640
PP	105(STB)	7.17-105.20	32.6154	17.3838
PP	99(STB)	7.20-90.00	25.6209	15.4465
PP	99(click)	7.80-65.00	21.6777	11.0327

STB: short duration tone burst

P13 and n23: latency

PP: peak to peak amplitude

The latency, peak-to-peak amplitude value obtained in two subgroup of control group did not differ significantly. Thus, data obtained in two subgroups were combined for the development of norms. It is evident from the table 5 that the mean p13 and n23 latency for clicks at 99dBnHL are shorter compared to STB at 105dBnHL and 99dBnHL. Click evoked VEMPs had shorter peak-to-peak amplitude than STB evoked VEMPs. For developing the norms, responses of "mean  $\pm 2$  SD" were derived at 95% confidence levels in combined age groups. In table 5 combined mean, standard deviation and range are given.

### 1. Effect of Intensity on VEMPs in control and experimental group for STB.

VEMPs were recorded at 99dBnHL and 105dBnHL for STB for both control and experimental group to obtained effect of intensity on VEMP. Independent sample t-test was administered to see the significant difference between two intensity levels i.e.

99dBnHL and 105dBnHL for STB stimuli in control group. It can be seen in table 6; the mean p13 latency is same at 105dBnHL and 99dBnHL. For n23 latency there is slight difference in mean values, and peak-to-peak amplitude at 105dBnHL when compared to 99dBnHL. Standard deviation is less for latency p13 and n23, however the variance of peak-to-peak amplitude is more for both 99dBnHL and 105dBnHL. From the "t" values of independent sample t- test of statistical analysis, it can be inferred that there is no significant difference in VEMPs for p13 latency. Statistically significant difference in VEMPs for n23 latency and peak-to-peak amplitude between 99dBnHL and 105dBnHL were obtained.

Table 6: Depicts the Mean, SD and "t" value of VEMPs at 105dBnHL and 99dBnHL obtained in control and in experimental group for STB.

			Control			Experimental		
Pai		Intensity	Mean	Standard	t-value	Mean	Standard	t-value
r		(dBnHL)		deviation			deviation	
1	P13	105	13.5792	1.0258		14.552	1.6165	
					.117	1		1.660
		99	13.5712	1.0913		14.286	1.7690	
						4		
2	N23	105	21.7873	1.9404		22.554	1.5381	
					4.015*	3		1.354
		99	21.3528	2.1244		22.192	2.0544	
						9		
3	рр	105	33.1250	17.3052		32.302	14.0870	
					6.923*	5		6.608*
		99	25.6209	15.4465		22.482	10.1787	
						1		

\*Significant at the 0.05 levels

STB: short duration tone burst

C: click

P13 and n23: latency

PP: peak to peak amplitude

In experimental group it can be seen in table 6 that, the mean p13 and n23 latency are approximately same at both intensity levels. Peak to peak amplitude is larger at 105dBnHL compared to 99dBnHL. The variability of p13 and n23 latencies was less. However, peak-to-peak amplitude variability is more at both the intensity levels (99dBnHL & 105dBnHL). From the "t" values of independent sample t-test of statistical significance, it can be inferred that there is no significant difference in VEMPs response at p13 and n23 latency. However, there is significant difference in VEMP results for peak-to-peak amplitude.

## **3.** Comparison of VEMP responses obtained in control and experimental group and between STB and click evoked VEMP.

To compare VEMPs obtained in control group and experimental group for two different stimuli (STB & Clicks). Independent sample t-test was administered to see the significant difference between control and experimental group.

It is evident from table 7 that there is a mean difference for p13 and n23 latency and peak-to-peak amplitude of control group than that of experimental group. Comparison to normal groups the experimental groups had prolonged mean latency for p13 and n23 latency. Peak to peak amplitude were slightly reduced in experimental group as compared to control group at both 105dBnHL and 99dBnHL for STB and 99dBnHL for click. The standard deviation values reveal less variability. From the "t" values of independent sample t- test of statistical significance, it can be inferred that there is significant difference in VEMPs response in normal hearing subjects without dizziness and subjects with dizziness for latencies P13 and n23 for STB at two different levels (99dBnHL & 105dBnHL), as well as for click at 99dBnHL. There is no significant difference for peak-to-peak amplitude at both intensity levels (105dBnHL & 99dBnHL) for STB and as well as click at 99dBnHL in both control and experimental group.

Table 7: Depicts the Mean, and SD of latency and peak to peak amplitude of VEMP and "t" value for both control and experimental group at different intensities for two stimuli (STB & click)

Intensity		GROUP	Mean	Std.	t-value
(dBnHL)				Deviation	
105(STB)	P13	Control	13.6020	1.0301	-4.938*
100(01D)	110	Experimental	14.9183	2.2091	
99(STB)	P13	Control	13.5712	1.0913	-2.706*
33(010)	115	Experimental	14.2864	1.7690	
99(C)	P13	Control	11.5450	1.0758	-3.401*
33(0)	110	Experimental	12.5273	2.3632	
105(STB)	N23	Control	21.8014	1.9184	-2.277*
105(010)	1125	Experimental	22.6367	1.9385	
99(STB)	N23	Control	21.3528	2.1244	-1.885
99(010)	FB) N23	Experimental	22.1929	2.0544	
		Control	19.2032	2.2640	-2.574*
99(C)	N23	Experimental	20.4836	3.2569	
		Control	32.6154	17.3838	1.088
105(STB)	PP	Experimental	29.1194	14.8982	
00(STD)	PP	Control	25.6209	15.4465	1.020
99(STB)	FF	Experimental	22.4821	10.1787	
00(C)	PP	Control	21.6777	11.0327	1.178
99(C)	ГГ	Experimental	19.2255	8.5066	

\* Significant at the 0.05 levels

STB: short duration tone burst

C: click

P13 and n23: latency

PP: peak to peak amplitude

Comparisons were made at 99dBnHL between two stimuli (STB & click) in subject with normal hearing without dizziness and subjects with dizziness. It shows that click is having better latency (p13 & n23) as compared to STB. And peak to peak amplitude is better for STB as compared to clicks in normal hearing subject.

The mean latency is prolonged and peak-to-peak amplitudes reduced in experimental group as compared to control group.

Table 8: Shows the significant difference between click and STB at 99dBnHL in control and experimental group.

	VEMP parameters	Intensity (dBnHL)	Control group	Experimental group
			t- value	t- value
Pair 1	P13	99(STB)-99Click)	21.766*	11.098*
Pair 2	n23	99(STB)-99Click	12.131*	3.882*
Pair 3	PP	99(STB)-99Click	4.165*	1.723

\* Significant at the 0.05 levels

STB: - short duration tone burst

C: - click

P13 and n23:- latency

PP: - peak to peak amplitude

Pair t-tests were administered to see the significance difference between two stimuli (STB & click) at 99dBnHL for control as well as for experimental group. It is evident from the table 8 that there is significant difference in p13 and n23 latency in control and experimental group between two stimuli STB and click. However there is a no significant difference in VEMP results for peak-to-peak amplitude for experimental group.

# 4: Descriptive analysis of VEMPs response obtained in control and experimental group.

To find the response rate of VEMPs of two age (21-30 & 31-40 years) groups were combined at different level 99 dBnHL and 105 dBnHL for STB and for click 99 dBnHL. However, 70dBnHL were also taken to check the presence of false positive VEMP response.

It is evident from the table 9, percentage of VEMP responses elicited in control group is higher for tone burst at 105dBnHL compared to 99dBnHL for STB and click stimuli. The

elicited response rate was higher for click compare to STB at 99dBnHL. However, the difference in response elicited by 105dBnHL STB and 99dBnHL STB or 99dBnHL click were negligible.

Table 9: Shows the number of ears and percentage of VEMP responses elicited in normal hearing subjects without dizziness

Stimuli	Intensity level	Total no. Of	Present VEMP	Response rate
	(dBnHL)	ear.	response	(%)
Short duration	105	120	116	96.66
tone burst	99	120	114	94.16
Click	99	120	115	95

VEMPs were recorded at 70dBnHL for both click and STB to check for false positive responses. It was observed that almost all the ears VEMP was absent in which VEMPs were recorded at higher intensity level. These ears showed noisy VEMP responses at 70dBnHL with amplitude of  $5\mu v$  without replicability. Thus, it suggests that VEMP recorded from all these subjects were not due to muscle artifact.

In experimental group, 30 ears were having either absent or abnormal VEMPs at 99dBnHL out of 50 ears. The same results were consistent for both click and STB i.e. when VEMP was absent or abnormal it was absent or abnormal for both STB and click.

Table10: Shows the number of ears and percentage of VEMP responses elicited in subject with dizziness

Stimulus	Intensity lev	vel	Total No. of ear	Present VEMP	Percentage (%)
	(dBnHL)			response (No.	of VEMPs
				of ear)	
Short tone burst	105		50	23	46
	99		50	18	36
Click	99		50	30	40

It is evident from the table 10, eliciting VEMP response was higher at the 105dBnHL compared to 99dBnHL for STB stimuli in subject with dizziness. Click is having slightly higher response rate as compared to STB at 99dBnHL. However, when comparison was made at 105dBnHl for STB and 99dBnHL for click, it was observed that STB is having higher response rate in subject with dizziness.

### 6: Symptoms related VEMPs in subject with dizziness.

Table 11: Shows the normal and abnormal VEMP response in subject with dizziness with respect to two major symptoms.

Symptoms	Absent or	abnormal VEMP	response in	Normal	VEMP re	sponse in	
	subject with	n dizziness		subject with dizziness			
	No. of	Total No. of	% (having	No. of	Total	%(present	
	subject(	subjects(showed	absent or	subject	No. of	VEMP)	
	had	having absent or	abnormal)	(present	subjects		
	absent or	abnormal)		VEMP)	( present		
	abnormal)				VEMP)		
Objects	13	17	76.47	3	8	37.5	
spinning							
or turning							
around							
you.							
Sensation	4	17	23.52	5	8	62.50	
that you							
are turning							
or							
spinning							
inside							

A part of dizziness questioner Maryland hearing and balance centre were administered in subjects with dizziness to find the symptoms related VEMPs. There were two major symptoms observed in subject with dizziness like "objects spinning or turning around you and Sensation that you are turning or spinning inside". Either one of the symptoms always present in subject with dizziness among all the symptoms were taken into consideration. It is evident from table 11, that, subjects who reported symptoms of "objects spinning or turning around you" showed either absent or abnormal VEMP response in 76.47% of subjects. However, subject who had symptoms of "Sensation that you are turning or spinning inside" showed either absent or abnormal VEMP response in 23.52% of subjects.

As it was observed that subject reported having symptoms of "Sensation that you are turning or spinning inside" 62.50% of subject was have normal VEMP response and 37.50% of the subject have present normal VEMP who complaint of symptoms "objects spinning or turning around you".

Symptoms	No. of	subject	Total	No. of	%	of
	having ab	sent or	subject	having	subjects	
	prolonged V	<b>VEMPs</b>	absent c	or abnormal	having	
			VEMP		abnormal	l
					VEMPs	
Lightheadedness or	3		17		17.64	
swimming sensation in						
the head.						
Blacking out or loss of	4		17		23.35	
consciousness.						
Tendency to fall.	6		17		35.29	
Objects spinning or	13		17		76.64	
turning around you.						
Sensation that you are	4		17		23.52	
turning or spinning inside.						
Loss of balance when	8		17		47.05	
walking.						
Headache	5		17		29.41	
Pressure in the head	4		17		23.35	
Nausea or vomiting.	6		17		35.29	

Table 12: Descriptive analysis of VEMPs based on symptoms

It has been observed that the subject who had symptoms of "objects spinning or turning around you" most often had other symptoms associated with nausea or vomiting, tendency to fall and loss of balance when walking. Hence, all these symptoms also showed higher percentage of either absent or abnormal VEMP response. One of the other symptoms "loss of balance when walking" had 32% absent or abnormal VEMPs response. When VEMP response was absent most often, the symptoms "Sensation that you are turning or spinning inside" is associated with headache and pressure in the head. When VEMP response was present most often, the symptoms "Sensation that you are turning or spinning inside" is associated with Blacking out or loss of consciousness and Lightheadedness or swimming sensation in the head. Hence, it can be concluded that one can expect absent or abnormal VEMP responses when the individual has symptoms of "objects spinning or turning around you", which might indicate abnormality in saccular pathway.

### DISCUSSION

#### 1. Age related changes on VEMPs in subject normal hearing without dizziness.

The results of the present study was shown that there is no significant difference in VEMPs in two age groups (21-30 years & 31-40 years) for p13 and n23 latency and peak to peak amplitude, except for peak to peak amplitude at 105dBnHL for STB stimuli.

The similar finding was also reported by Su, Haung, young and Cheng (2004). They also did not find any significant difference in the age range from 21-40 years and as age increase over 60 years, the response rate decreased dramatically with increasing in age. Welgampola and Colebatch (2001) suggested that the average click- evoked responses amplitudes decreased with age, with a pronounced decline of 25-30% per decade from the  $6^{th}$  decade. It is clear from the both the studies that there is significant difference in amplitude as the age increase over 60 years. These changes are probably due to morphological changes in the vestibular system occurring and corresponding change in neural function. Hence, significant changes could not observe in present study.

In the present study there was significant difference in peak-to-peak amplitude at 105dBnHL for STB in two age groups. This might be due to chance factor or may be due to inability to keep the tonic muscle contraction constant.

Thus, the present study suggests that there will not be any significant changes in VEMP test results in normal individual with in age between 21 to 40 years who do not have symptoms of dizziness.

## 2. Effect of Intensity on VEMP responses in control and experimental group for STB.

In subject with normal hearing without dizziness and with dizziness there was no change in p13 and n23 latency as the intensity increase from 99dBnHL to 105dBnHL. However, significant change is obtained for n23 latency in normal hearing subject

without dizziness. And there was significant difference in peak-to-peak amplitude at 99dBnHL and 105dBnHL intensity level for STB in subject with normal hearing without dizziness and with dizziness subject.

In the present study there is a significant difference for n23 latency at 99dBnHL and 105dBnHL in subject with normal hearing without dizziness. Significant difference in n23 latency could be due to chance factor.

This study is also supports the results obtained by Akin, Murnane, and Profitt (2003), Colebatch, Halmagyi, and Skuse (1994) Lim, Closton, Sheean, and Yiannikas (1995) and Ochi, Ohashi and Nishino (2001). They had reported as the level of the click increased there is a corresponding increase in the level of the VEMP amplitude, however the VEMP latency p13 and n23 did not vary as a function of intensity level after the change in intensity level.

Thus, the present study suggesting that at the higher level from 99dBnHL to 105dBnHL the saccule pathway reaches the plateaus, which could have resulted in no change in p13 and n23 latency.

## **3.** Comparison of VEMP response obtained in control and experimental group also and between the STB and click evoked VEMPs.

The present study is in contrary to the study by Cheng et al, 2003 where they reported that the peak-to-peak amplitude for click evoked VEMP were larger than the tone burst VEMP. The difference between the two studies may be due to the difference in contraction of the sternocleidomastoid muscle of the subjects.

In the present study there was a significant difference between control group and experimental group for mean latency and the amplitude. The mean latency was prolonged in the experimental group as compared to control group for both STB and click evoked VEMP. This might be due to the lesion in the saccular or in the saccular pathway, resulted in abnormal VEMP for both STB and click evoked-VEMP.

Ribirio et al. (2005) reported prolonged latency and abnormal VEMPs (asymmetrical or long latency) in some of the Meniere's disease patient's. Prolonged latency and abnormal VEMPs have been reported in about 25% of the persons diagnosed with vestibular neuritis also (Murofushi, Halmagyi, Yavor, & Colebatch, 1996).

In the present study there is a significant difference between the latency of tone burst and click evoked VEMP. The latencies with Tone burst VEMP are longer than the click evoked VEMP. The findings of the present study are consistent with that of Cheng, Huang, and Young (2003). The delay in latencies may be attributed to the different firings pattern of the vestibular neurons to the tone burst. It has been reported that the primary vestibular neurons might have double or triple firing to one tone burst hence, the delayed latency of Tone burst evoked-VEMP might be due to the second or third spikes (Cheng & Murofushi, 2001b).

Further Tone burst VEMP amplitudes were larger than that of Click evoked-VEMP when comparisons were made at equal SPL. The observed differences between the STB VEMP and Click evoked-VEMP may be due to the differences in stimulus spectrum level. When comparisons are made at equal peak SPL, the click has a lower spectrum level than the tone bursts due to its wider bandwidth (Akin, Murnane, and Proffittm2003).

## 4. Descriptive analysis of VEMP results obtained in subject without dizziness and subject with dizziness.

In the present study STB-VEMPs at 105dBnHL had higher response rate 96.66% as compared to 99dBnHL. The response rate is higher for click (95%) in normal individual without dizziness when VEMP results were compared at same intensity level.

Subject with dizziness also showed similar finding at the same intensity level response rate is higher for click compared to STB stimuli.

Cheng, Huang and young (2003) had reported click stimulation produced 98% VEMPs response and STB-VEMP shown 88% positive response at 95dBnHL. From this study it can be concluded that the sensitivity of VEMPs is higher for click than the STB

Previous report demonstrates that 34 affected ears of vestibulocochlear disorders revealing same VEMP response rate 88% with STBs and with clicks (Murofushi, Matsuzaki, & Wu, 1999).

Thus, it can be concluded that at the higher intensity level in control as well as in experimental group the efficacy to elicit the VEMP response is higher than at VEMP threshold level. As the VEMP response elicilating rate at the higher intensity level is approximately same response rate for both stimuli STB and click thus at the higher intensity either STB or click stimuli can be used to elicitate the VEMP response. However, the maximum level at which click can be presented is less in compared to STB. Thus, STB can be used to elicit VEMP due to its dynamic range.

In the present study as it was observed that some of the subject was having noisy VEMP response or very less amplitude which cannot be taken as response. Hence, it suggests that to check the VEMP response it is necessary to check with lower intensity where the VEMP response usually absent to ensure that the response were obtained is not a muscle artifact.

#### **5.** Symptoms related VEMPs response in subject with dizziness

As exposed in table 9, subject with "objects spinning or turning around you" symptoms showed absent or abnormal VEMP response most of the time. However, this can be conclude that this symptom produces realistic VEMP. Interestingly point is that most of the subject who reported tendency to fall, nausea or vomiting and loss of balance

when walking have also reported of "objects spinning or turning around you", which are more commonly associated with Meniere's disease.

In Meinere's disease, nausea or vomiting is a major complaint. Ribeiro, Almeida, Caovilla, and Ganan, (2005) reported altered VEMP in 35% of the affected ears and in 25% of the asymptomatic ears in Meinere's diseases. Absent of biphasic VEMP wave in between 40 to 50% of Menieres diseases reported by Waele, Huy, Diard, Fressy, and Vidal (1999), Murofushi, Matsuzaki, and Takeghoshi (2001b), Seo, Node, Yukimasa, and Sakagami (2003), Shojaku, Takemori, Kobayashi, and Wantanable (2001) and Young, Haung, and Cheng (2003).

The individual with Meniere's disease have associated symptom like nausea or vomiting. In present study also it found that there is high percentage of absent or abnormal VEMP in subject with symptoms of "nausea or vomiting". Which is almost always associated with symptom of "objects spinning or turning around you".

From the present study it can be suspected that if the dizziness patients show the symptoms of "objects spinning or turning around you, tendency to fall, loss of balance when walking and nausea or vomiting" can have the lesion in the saccular pathway.

One of the other symptom "sensations that you are turning or spinning inside" has less percentage of abnormal VEMP response. And who had complained of "sensation that you are turning or spinning inside" most often this symptoms was associated with symptoms lightheadedness or swimming sensation in the head and pressure in the head. Thus, it can be concluded that individual having such symptoms may not have any saccular impairment.

### SUMMARY AND CONCLUSION

VEMPs are short latency electromyogram (EMG) that are evoked by higher-level acoustic stimuli and are recorded from surface electrodes over the tonically contracted sternocleidomastiod (SCM) muscle. It can be affected by several factors like stimulus use, intensity, tension of the muscles, age etc. This has got potential in diagnostic implication. Ribeiro, Almeida, Caovilla and Ganan, (2005) found in VEMPs were altered in 35% of the affected ears and in 25% of the asymptomatic ears in Meniere's diseases. No significant differences on VEMPs were seen in the age range from 21-40 years and as the age increases over 60 years, the response rate decreased and amplitude decrease. (Su, Haung, Young & Cheng, 2004 and Welgampola & Colebatch, 2001). Cheng, Huang and young (2003) observed the response rate of VEMPs is higher for click than the STB. Hence, attempt has to be made to establish norms as this is still in infantile stage in the field of Audiology. There is hardly any study, which tried to correlate the type symptoms of dizziness and VEMP results.

Thus, the present study was taken up to investigate

1. Age related changes of VEMP response in normal-hearing volunteers for both click and short tone burst.

2. Effect of Intensity on VEMPs responses.

3. To observe the VEMP response in subject with dizziness.

4. Specificity of VEMP in subject with normal hearing without dizziness.

5. The present study also aims in investing the association of VEMP responses with symptoms of dizziness.

For this purpose VEMPs was recorded for click and STB across ages (21-30 & 31-40 years) for 30 normal hearing subject without dizziness and 25 subjects with dizziness across intensity level for STB 99dBnHL and 105dBnHL and 99dBnHl for clicks. Pair t-test and independent sample t-test was carried out to analyze the data collected.

Results of the study indicate that:

- 1. There is no significant difference in VEMPs response at two age groups i.e. between 21 to 30 and 31 to 40 years for both stimuli across all the intensity levels.
- 2. As the intensity level of STB was increased from 99dBnHL to 105dBnHL there was increase in peak-to-peak amplitude, however there is no change in p13 and n23 latency. It was observed in both control and experimental group.
- 3. As observed in experimental group the latency of p13 and n23 were prolonged and peak-to-peak amplitude reduced compared to control group.
- 4. It was also observed that click is having shorter latency and shorter amplitude as compared to STB.
- 5. At the higher intensity levels the VEMPs response rate is higher as compared to threshold level of VEMPs.
- 6. 30 No. of ears had absent or abnormal VEMP response out of 50 ears at 99dBnHL.
- 7. 94% of the subject with normal without dizziness had VEMPs.
- Subject who complain "objects spinning or turning around you" tendency to fall, loss of balance when walking and nausea or vomiting, the incidence of abnormal VEMPs were more.

Thus, it can be concluded that there will not be any change in VEMPs in age from 21 to 40 year. Click evoked VEMPs likely to have shorter latency and reduced amplitude than STB evoked VEMPs at same dBnHL. However, the maximum limit can be present for STB is higher than the click. Thus, STB can be used at maximum intensity to check for VEMPs as the percentage of VEMP response increased from 94% to approximately 97% from 99dBnHL to 105dBnHl in normal. It can also be concluded that abnormal VEMPs can associated with symptom "objects spinning or turning around you, tendency to fall, loss of balance when walking and nausea or vomiting". Thus, can be concluded that the subject who complain these symptoms are likely to have saccular pathway lesion.

### Implication

- 1) Norms established could be used to compare the data obtained in clinical population.
- 2) The present study suggest there will not be any change in VEMP results age between 21 to 40 year through Johnson and Hawkin (1972) reported that there could be loss of otoconia from the age of 30 years onward.
- It also highlights the symptoms are more likely to be associated with abnormal VEMP which in turn can suggest the possible site of lesion.

#### REFERENCES

- Akin, F. M., & Murnane, O. D. (2001). Vestibular evoked myogenic potentials: Preliminary report: *Journal of the American Academy of Audiology*, *12*, 445-452.
- Akin, F. M., Murnane, O. D, & Proffitt, T. M. (2003). The effects of click and tone burst stimulus parameters on the Vestibular evoked myogenic potential. *Journal of the American Academy of Audiology*, 14,500-509.
- Alpini, D., Pugnetti, L., Caputo, D., Cornelio, F., Capobianco, S., & Cesarani, A. (2004). Vestibular evoked myogenic potentials in Multiple Sclerosis- clinical and imaging correlations. *Multiple Sclerosis*, 10 (30), 316-321.
- Bath, A. P., Harris, N., Mc Ewan, J., & Yardley, M. P. (1999). Effect of conductive hearing loss on the Vestibulocollic reflex: *Clinical Otolaryngology*, *24*, 181-183.
- Bergstrom, B. (1973). Morphology of the vestibular nerve: the number of myelinated vestibular nerve fibers at various ages. *Acta Otolaryngologica*, *76*,173-179.
- Bickford, R. G., Jacobson, J. L., & Cody, D. T. R. (1964). Nature of average evoked potentials to sound & other stimuli in man. *Annals of the New York Academy of Sciences*, 112, 204-218.
- Brantberg, K., Bergenius, J., Mendel, L., Witt, H., Tribukait, A., & Ygge. (2001). Symptoms, findings, and treatment in patients with dehiscence of the semicircular canal. *Acta Oto-laryngologica (stockh)*, *121*, 68-75.
- Brantberg, K., Bergenius, J., & Tribukait, A. (1999). Vestibular evoked myogenic potentials in patients with dehiscence of the superior semicircular canal. *Acta Otolaryngologica* (stockh), *119*, 633-640.

Cody, D. T. R., & Bickford, R. G. (1969). Average evoked myogenic responses in normal man. *Laryngoscope*, 79, 400-446.

- Cheng, P. W., Haung, T. S., & Young, Y. H. (2003). The influence of clicks versus short tone bursts on the vestibular evoked myogenic potentials. *Ear and Hearing*, 24(3), 129-197.
- Cheng, P. W., & Murofushi, T. (2001a). The effect of Rise/ fall time on Vestibular evoked myogenic potential triggered by short tone bursts. *Acta Otolaryngologica 121*, 696-699.
- Cheng, P. W., & Murofushi, T. (2001b). The effect of plateau time on Vestibular evoked myogenic potential triggered by tone bursts. *Acta Otolaryngologica*, 121, 935-938.
- Colebatch, J. C., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click – evoked vestibulocollic reflex. *Journal of Neurology Neurosurgery and Psychiatry*, 57, 190-197.
- Colebatch, J. C., Rothwell, J. C., Bronstein, A., & Ludman, H. (1994). Click-evoked vestibular activation in the Tullio-phenomenon. *Journal of Neurology Neurosurgery and Psychiatry*, 57, 1538-1540.
- Colebatch, J.C. (2001). Vestibular evoked myogenic potentials. *Current opinion in Neurology*, 14, 21-26.
- Debatisse, D., Pralong, E., Guerit, J. M., & Bisdorff, A. (2005). Recording click-evoked myogenic potentials (CEMPs) with a setup for brainstem auditory evoked potentials (BAEPs). *Clinical Neurophysiology*, 35(4), 109-117.

- De waele. C., Hay, P. T., Diard, J. P., Freyss, X., & Vidal, P. P., (1999). Saccular dysfunction in Meniere's disease. *The American Journal of Otology*, 20, 223-232.
- De Waele, C., Meguenni, R., Freyss, G., Zamith, F., Bellalimat, N., Vidal, P. P., Tran, Ba Huy P. T. (2002). intratympanic gentamicin injections for Meniere's disease: Vestibular hair cell impairment & regeneration. *Neurology 59*, 1442-1444.
- Dizziness questionnaire, Maryland Hearing and Balance center. Retrived on September, 2004.www.umm.edu/otolaryngology/dizziness\_quest.doc
- Ferber- Viart, C., Dubreuil, C., & Duclaux, R. (1999). Vestibular evoked myogenic potentials in human: A Review. Acta Otolaryngologica, 119, 6-15.
- Ferber- Viart, C., Duclaux, R. Colleaux, B., & Dubreuil, C. (1997). Myogenic evoked potentials in normal subjects: A comparison between responses obtained from sternomastoid & trapezius muscles. *Acta Otolaryngologica*, 117, 472-481.
- Fujikawa, S., & Starr, A. (2000). Vestibular neuropathy accompanying auditory and peripheral neuropathies. Archive Otolaryngology Head Neck Surgery, 126, 1453-1456.
- Geisler, C. D., Frishkopf, L. S., & Rosenblith, W. A. (1958). Extracranial responses to acoustic clicks in man. *Science*, *128*, 1210-1211.
- Goldberg, J. M., Fernandez, C., & Smith, C. E. ((1982). Responses of vestibular-nerve afferents in the squirrel monkey to extremely applied galvanic currents. *Brain Research*, 252, 150-160.
- Halmagyi, G. M., Aw, S. T., Karlberg, M., Curthoys, I.S., & Todd, M. J. (2002). Inferior vestibular neuritis. *Annals of the New York Academy of sciences*, 956, 306-313.

- Halmagyi, G. M., Colebatch, J. G., & Curthoys, I. S. (1994). New tests of vestibular function. *Baillieres clinical neurology*, 3, 485-500.
- Halmagyi, G. M., & Colebatch, J. G. (1995). Vestibular evoked myogenic potentials in the sternomastiod muscle are not of lateral canal origin. *Acta Otolaryngologica*, (suppl.520), 1-3.
- Halmagyi, G. M., & Curthoys, I. (2000). Clinical testing of otolith functions. New York Academy of Sciences.871, 195-204. Retrieved January 30,2006 from http://www.annalsnyas.org/cgi/content/abstract/871/1/195
- Halmagyi, G. M., Yavor, R. A & Colebatch, J. G. (1995). Tapping the head activates the vestibular system: a new user for the clinical reflex hammer. *Neurology*, 45, 1927-1929.
- Haung, T. W., Su, H. C., & Cheng, P. W. (2005). Effect of click duration on Vestibular evoked myogenic potentials. *Acta Otolaryngologica*, 125, 141-144.
- Heide, G., Freitage, S., Wollenberg, I., Iro, H., Schimrig, K., & Dillmann, U. (1999).
  Click evoked myogenic potentials in the differential diagnosis of acute vertigo. *Journal of Neurology, Neurosurgery, and psychiatry*, 66, 787-790.
- Johnsson L. G, Hawkins J. E. (1972). Sensory and neural degeneration with aging as seen in the micro dissection of the human inner ear. Annal Otology Rhinology Laryngology, 81, 179-193.
- Kushiro, K., Zakir. M., Ogawa, Y., Sato, H., & Uchino, Y. (2000). Saccular and utricular inputs to single vestibular neurons in cats. *Experimental Brain Research*, 131(4), 406-415.

- Li, M. W., Houlden, D., & Tomlinson, R. D. (1999). Click evoked EMG responses in sternocleidomastoid muscles characteristics in normal subjects. *Journal of Vestibular Research*, 9, 327-334.
- Lim, C. L., Clouston, P., Sheean, G., & Yeannikas, C. (1995). The influence of voluntary EMG activity & click intensity on the vestibular click evoked myogenic potential. *Muscle Nerve*, 18, 1210-1213.
- Lopez, I., Honrubia, V., Baloh, R. W., (1997). Aging and the human vestibular nucleus. *Journal of vestibular Research*, 7, 77-85.
- Matsuzaki, M., Murofushi, T., & Mizuno, M. (1999). Vestibular evoked myogenic potentials in acoustic tumor patients with normal auditory brainstem responses. *European Archives of Otorhinolaryngologica*, 256, 1-4.
- Matsuzaki. M., & Murofushi, T. (2001). Vestibular evoked myogenic potentials in patients with Idiopathic bilateral vestibulopathy. Report of three cases. *Journal of Otorhinolaryngologica*, 63, 349-352.
- Mc cue, M. P., & Guinan, J. J. (1995). Spontaneous activity & frequency selectivity of acoustically responsive vestibular afferents in the cats. *Journal of Neurophysiology*. 74, 1563-1572.
- Murofushi, T., Matsuzaki. M., & Takegoshi. H. (2001). Glycerol affects vestibular evoked myogenic potentials in Meniere's disease. *Auris Nasus Larynx*, 28(3), 205-208.
- Murofushi, T., Halmagyi, G. M., Yavor, R. A. & Colebatch, J. G (1996). Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis: An indicator of inferior vestibular nerve involvement. *Archives Otolaryngology Head Neck Surgery*, 122, 845-848.

- Murofushi, T., Matsuzaki. M., & Mizuno, M. (1998). Vestibular evoked myogenic potentials in patients with acoustic neuromas. Archives Otolaryngology Head Neck Surgery, 124, 509-512.
- Murofushi, T., Matsuzaki. M., & Wu, C. H. (1999). Short tone burst evoked myogenic potentials on the sternocleidomastoid muscle: Are these potentials also of vestibular origin? Arch Otolaryngology Head Neck Surgery, 125, 660-664.
- Murofushi, T., Shemizu, K., Takegoshi, H., & Cheng, P. W. (2001). Diagnostic value of prolonged latencies in the Vestibular evoked myogenic potential. Archives Otolaryngology Head Neck Surgery, 127, 1069-1072.
- Murofushi, T., Takegoshi. H., Ohki, M., & Ozeki, H. (2002). Galvanic evoked myogenic responses in patients with an absence of click-evoked vestibulocollic reflexes. *Clinical Neurophysiology*, 113, 305-309.
- Ochi, K., Ohashi, T., & Kinoshita, H. (2002). Acoustic tensor tympanic response & vestibular evoked myogenic potential. *Laryngoscope*, *112*, 2225-2229.
- Ochi, K., Ohashi, T., & Nishino, H. (2001). Variance of vestibular evoked myogenic potentials. *Laryngoscope*, *111*, 522-527.
- Ohki, M., Matsuzaki, M., Sugasawa, K., & Murofushi, T. (2002). Vestibular evoked myogenic potentials in patients with contralateral delayed endolymphatic hydrops. *European Archives of Otorhinolaryngologica*, 259, 24-26.
- Ozeki, H., Iwasaki, S., & Murofushi, T. (2005). Effect of stimulation repetation rate on galvanic-evoked vestibulo-collic reflexes. *Acta Otolaryngologica*, *125*, 159-162.

- Ribeiro, S., Almeida, R. R., Caovilla, H. H., & Gananca, M. M. (2005). Vestibular evoked myogenic potentials in affected and asymptomatic ears in unilateral Meniere's disease. *Revista Braseleira DE Otorrinolaringologia*, 7, 60-66.
- Richter E. (1980). Quantitative study of human scarpa's ganglion and vestibular sensory epithelia. *Acta Oto-laryngologica*, 90: 199-208.
- Robertson, D. D., & Ireland, D. J. (1995). Vestibular evoked myogenic potentials. *The Journal of Otolaryngologica*, 24, 3-8.
- Rosenhall, U. (1973). Degenerative patterns in the aging human vestibular neuro epithelial. *Acta Otolaryngologica*, *76*, 208-220.
- Seo, T., Node, M., Yukimasa, A., & Sakagame, M. (2003). Furosemide loading vestibular evoked myogenic potential for unilateral Meniere's disease. *Otology & Neurotology*, 24 (2), 283-288.
- Sheykholeslami, K., Kaga, K., Murofushi, T., and Hughes, D. W. (2000). Vestibular function in auditory neuropathy. *Acta Otolaryngologica*, *120*, 849-854.
- Sheykholeslami, K., Kermany, H. M., & Kaga, K. (2001). Frequency sensitivity range of the saccule to bone- conducted stimuli measured by Vestibular evoked myogenic potential. *Hearing Research*, 5, 58-62.
- Sheykholeslami K, Megerian C. A, Arnold J. E., Kaga K. (2005). Vestibular evoked myogenic potential in infancy and early childhood. *Laryngoscope*, 115 (8), 1440-1444.
- Sheykholeslami, K., Murofushi, T., Kermany, H, M., & Kaga, K. (2001). Bone conduction evoked myogenic potentials from sternocleidomastoid muscle. *Acta Otolaryngologica*, 120, 731-734.

- Sheykholeslami, K., Schmerber, S., Kermany M. H., & Kaga, K. (2005). Sacculo-collic pathway dysfunction accompanying auditory neuropathy: case report. *Acta Otolaryngologica*, 125, 786-791.
- Shimizu, K., Murofushi, T., Sakurai, M., & Halmagyi, M. (2000). Vestibular evoked myogenic potential in multiple sclerosis. *Journal of Neurology Neurosurgery* psychiatry, 69, 276-277.
- Shojaku, H., Takemori, S., Kobayashi, K., Watanabe, Y. (2001). Clinical usefulness of glycerol vestibular evoked myogenic potentials: preliminary report. Acta OtoLaryngologica (suppl.545), 65-68.
- Streubel, S. O., Cremer, P. D., Carey, J. P., Weg, N., & Minor, L. B. (2001). Vestibular evoked myogenic potential in the diagnosis of superior canal dehiscence syndrome. *Acta Otolaryngologica* (suppl. 545), 41-49.
- Su, H. C., Huang, T. W., Young, Y. H., Cheng, P. W. (2004). Aging effect on vestibular evoked myogenic potential. *Otology & Neurotology*, 25(6), 977-980.
- Takegoshi, H., & Murofushi, T. ((2000). vestibular evoked myogenic potentials in patients with spinocerebellar degeneration. *Acta Otolaryngologica*, *120*, 821-824.
- Toddy, N. P. M., Cody, F. W. J., & Banks, J. R. (2000). A saccular origin of frequency tuning in myogenic vestibular evoked potentials? Implication for human responses to loud sounds. *Hearing Research*, 141, 180-188.
- Townsend, G. L., & Cody, D. T. R. (1971). The averaged inion response evoked by acoustic stimulation: its relation to the saccule. *Annals of Otology, Rhinology & Laryngology*, 80, 121-131.

- Tsutsumi, T., Tsunoda, A., Noguchi, Y., & komatsuzaki, A. (2000). Prediction of the nerves of origin of vestibular schwannomas with vestibular evoked myogenic potentials. *The American Journal of Otology*, *21*, 712-715.
- Uchino, Y., Sato, H., Sasaki, M., Imagawa, M., & Ikegami, H. (1997). "Sacculocollic reflex arcs in cats" *journal of neurophysiology*, 77(6), 3003-3012.
- Wang, S.J., & Young, Y.H. (2003). Vestibular evoked myogenic potentials using simultaneous binaural acoustic stimulation. *Hearing Research*, 185(1-2), 43-48.
- Watson, S.R., Colebatch, J.G. (1998) Vestibulocollic reflex evoked by short-duration galvanic Stimulation in man. *Journal of physiology*, *513*, 587-597.
- Watson, S.R.D; Halmagyi, G.M; & Colebatch, J.G. (2000). Vestibular hypersensitivity to sound (Tullio phenomenon) structural & functional assessment. *Neurology*, 54, 722-728.
- Welgampola, M.S., & Colebatch, J.G. (2001). Characteristic of tone burst-evoked myogenic potentials in the sternocleidomastoid muscles. *Otology Neurotology*, 22, 796-802.
- Welgampola M. S., Colebatch J. G. (2001) Vestibulocallic reflexes: normal Values and the effect of age. *Clinical Neurophysiology*, 112, 1971-1979.
- Welgampola, M. S., Rosengren, S. M., Halmagyi, G. M., & Colebatch, J. G. (2003). Vestibular activation by bone conducted sound. *Journal of neurology Neurosurgery Psychiatry*, 74, 771-778.
- Wilson, V.J., R.Boyle et al. (1995). "The vestibulocollic Reflex" *Journal of research* 5(3), 147-70.

- Wu, C.H., & Murofushi, T. (1999). The effect of click repetition rate on vestibular evoked myogenic potential. Acta OtoLaryngologica (stockh), 199, 29-32.
- Wu, C.C., & Young, Y.H. (2002). Vestibular evoked myogenic potentials are intact after sudden deafness. *Ear and Hearing*, 23, 235-238.
- Yang, T. L., & Young, Y. H. (2003). Comparison of tone burst & tapping evocation of myogenic potentials in patients with chronic otitis media. *Ear and Hearing*. 24, 191-194.
- Young, Y. H., Huang, T. W., Cheng, P. W. (2003). Assessing the stage of Meniere's disease using Vestibular evoked myogenic potentials. Archive of Otolaryngology head Neck Surgery, 129, 815-818.
- Young, Y. H., Wu C. C., & Wu, C. H. Augmentation of Vestibular evoked myogenic potential an indication for distended saccular hydrops. *Laryngoscope*, 112, 509-512.
- Zhou, G., Cox, L. C. (2004). VEMPS: History & overview. American Journal of Audiology, 13, 135-143.

### APPENDIX

2nd Section of the dizziness questionnaire of Maryland Hearing and Balance Center were administered.

When you are "dizzy" do you experience any of the following sensations? You may circle as many yes responses as necessary.

head.

Yes	No	1.	Lightheadedness or swimming sensation in the
Yes	No	2.	Blacking out or loss of consciousness.
Yes	No	3.	Tendency to fall.
Yes	No	4.	Objects spinning or turning around you.
Yes	No	5.	Sensation that you are turning or spinning inside.
Yes	No.	6.	Loss of balance when walking
Yes	No	7.	Headache
Yes	No	8.	Pressure in the head.
Yes	No	9.	Nausea or vomiting