EFFECT OF DANCE TRAINING ON VESTIBULAR EVOKED MYOGENIC POTENTIALS

Swathi V M

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University of Mysore, Mysore

ALL INDIA INSTITUTE OF SPEECH AND HEARING,

MANASANGOTHRI, MYSORE – 570006

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CERTIFICATE

This is to certify that this dissertation entitled "*Effect of dance training on vestibular evoked myogenic potentials*" is the bonafide work submitted in part fulfillment for the Degree of Master of Science (Audiology) of the student with Registration No. : 09AUD034. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Dr. S.R. Savithri

Director

All India Institute of Speech and Hearing,

Mysore

Manasagangothri, Mysore-570 006

June, 2011

CERTIFICATE

This is to certify that the dissertation entitled *"Effect of dance training on vestibular evoked myogenic potentials"* has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any Diploma or Degree.

Sujeet Kumar Sinha Guide

Lecturer in Audiology,

Department of Audiology,

All India Institute of Speech and Hearing,

Mysore

June, 2011

Manasagangothri, Mysore - 570 006.

DECLARATION

This dissertation entitled "*Effect of dance training on vestibular evoked myogenic potentials*" is the result of my own study under the guidance of Mr. Sujeet Kumar Sinha, Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysore, June, 2011 Registration No.09AUD034

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CHAPTER-1

Introduction

Diagnostic testing of the vestibular system is an essential component of treating patients with balance dysfunction. Until recently, testing methods primarily evaluated the integrity of the horizontal semicircular canal, which is only a portion of the vestibular system. Recent advances in technology have afforded clinicians the ability to assess otolith function through Vestibular Evoked Myogenic Potential testing. This newly developed procedure augments the management of dizzy patients by increasing specificity when investigating the site of lesion.

The Vestibular evoked myogenic potentials (VEMP) is a biphasic inhibitory response elicited by loud clicks or tone bursts recorded from the tonically contracted sternocleidomastoid muscle, being the only resource available to assess the function of the saccule and the inferior vestibular nerve (Colebatch, Halmagyi & Skuse, 1994; McCue & Guinan,1995; Ferber-Viart, Duclaux, Colleaux, & Dubreuil,1997; Li, Houlden, & Tomlinson, 1999; Chen, Young & Wu, 2000; Ochi, Ohashi, & Nishino, 2001; Welgampola & Colebatch, 2001; Al- Abdulhadi, Zeitouni, Al-Sebeih, & Katsarkas, 2002; Clarke, Schonfeld, & Helling, 2003; Akin, Murnane & Proffitt, 2003). The afferent pathway of the VEMP, which is mediated by saccule (the receptor), is responsible for linear acceleration and deceleration. VEMP response consists of an initial positive peak (P13, or p1) in the ipsilateral SCM followed by successive negative and positive peaks (N23, P34, and N44). VEMP has been utilized for the diagnosis of various disorders such as, Meniere's disease (Murofushi, Shimizu, Takegoshi & Cheng, 2001; Iwasaki, Takai, Ito, & Murofushi, 2005), Acoustic neuromas (Murofushi, Matsuzaki & Mizuno, 1998; Murofushi et al.2001; Streubel, Cremer, Carey, Weg & Minor 2001; Suzuki, Yamada, Inoue, Kashio, Saito & Nakanishi, 2008) Superior canal Dehiscence syndrome (Brantberg, Bergenius & Tribukait,1999), Vestibular neuritis (Ochi, Ohashi & Watanabe, 2003), Vertigo (Yang, Kim, Lee & Lee, 2008), Noise induced hearing loss (Fakharnia, Sheibanizadeh, Jafari & Hoseini, 2009), Auditory neuropathy / audiovestibular neuropathy (Kumar, Sinha, Singh, Bharti & Barman, 2007), and in other disorders such as cerebellopontine angle tumor (Iwasakiet al., 2005), Multiple sclerosis, (Murofushi et al., 2001).

Plasticity of the auditory system is not a new entity. Various studies have reported a change in the response of the auditory system after training (Tremblay 2005; Christopher et al., 2006; Kacelnik, Nodal, Parsons & King, 2006). Similarly a remarkable plasticity has been noted in the vestibular system throughout life. Behavioural analyses of vestibular plasticity have focused primarily on the vestibulo-ocular reflex (VOR), which enables retinal images to remain stable during head motion by driving compensatory eye movements. Powerful forms of motor learning occur in the VOR whenever images move persistently on the retina during head movements (Gittis & Sascha, 2006). Learning in the VOR causes adaptive changes in the strength and/or timing of eye movements and can be quantified as changes in gain (ratio of eye speed to head speed) and phase (timing relationship between eye and head movement). There are various reports which suggest that dance based training improves the balance function in young as well as adult subjects (Federici, Bellagamba & Rocchi 2005; Krampe, et al., 2010).

In a recent study by Lavon et al., (2010), it has been shown that there is plasticity not only in the VOR system but also the utricle and the saccule. Lavon et al., (2010) recorded the vestibular evoked myogenic potential (VEMP) response to evaluate saccular function in 12 professional divers shortly after a dive and after an interval of at least 24 hours. The control group consisted of 12 matched non-divers. Wave latencies and amplitudes, asymmetry ratio, and the response threshold were compared between the groups. Results revealed a statistically significant shortening of N23-wave latency was in the divers compared with the control group.

Dance is generally recommended to maintain good dexterity and coordination, fluid movements of the joints, muscle tone and trophism. In dance, movement of the head and trunk and the shifting of the centre of gravity in every direction from the axis of support allow the development of all those factors which contribute to the maintenance of balance, such as coordination and joint mobility. It is well known that the best defence mechanism against injuries and risk of fall is well toned, strong flexible body. Appropriate alignment and range of motion of large joints are required for dance activity; in the same way, dance exercises represent a potentials relevant support in both increasing balance and decreasing the risk of falls and injuries.

Along with improving the muscle tonicity and other joint movements the dance can also improve the responses of the vestibular systems. During the dance exercises the body requires more balance, it is possible that the neuronal discharge may increase from the vestibular system in order to balance the body. In this process the vestibular system in dancers may be more responsive and thus the dancers may have a better balance system.

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Need of the study:

- Vestibular evoked myogenic potentials has been utilised as a good tool to assess the sacculocollic pathways. Therefore there is a need to study the vestibular evoked myogenic potentials as a tool to assess the plasticity in the sacculocollic pathway.
- Neurophysiologic evidences show that auditory training results in plasticity of the auditory system. However there is a dearth of information regarding the effect of such training on vestibular system, there is a need to study whether the training improves the neurophysiologic responses from the utricle and saccule.
- There are studies from different care centres for older individuals evidenced that dance based therapy would improve balance and gait (Krampe, et al., 2010).
 However these studies are subjective studies. There is need of an objective study to evaluate such changes occurring in the vestibular system

Objectives of the study:

- 1. To find out the effect of dance training on Vestibular Evoked Myogenic Potential (VEMP).
- To find out whether the sacculocollic pathway plasticity results in better latency or better amplitude.

CHAPTER-2

Review of Literature

Vestibular evoked myogenic potential (VEMP) is an inhibitory potential recorded from the sternocleidomastoid muscle (SCM) in response to loud sounds. VEMP testing provide a useful, noninvasive method for assessment of otolith function and the functional integrity of the inferior vestibular nerve (Colebatch, Halmagyi & Skuse, 1994; McCue & Guinan,1995; Ferber-Viart, Duclaux, Colleaux & Dubreuil,1997; Li, Houlden & Tomlinson, 1999; Chen, Young & Wu, 2000; Ochi, Ohashi & Nishino, 2001; Welgampola & Colebatch, 2001; Al- Abdulhadi, Zeitouni, Al-Sebeih & Katsarkas, 2002; Akin, Murnane & Proffitt, 2003; Clarke, Schonfeld & Helling, 2003).

VEMPs are believed to be a good indicator of saccular and inferior vestibular nerve function in clinical evaluations. When compared with the most commonly ordered clinical vestibular tests (e.g. electronystagmogram and rotary chair) that evaluate the pathway between the horizontal semicircular canal and the oculomotor nuclei (via the vestibulo-ocular reflex or VOR), this electrophysiological test is specific to otolith (saccule) and vestibulospinal reflex function. The VEMP pathway has been speculated to include the saccule, inferior vestibular nerve, vestibular nucleus, and medial and lateral vestibulospinal tract to the ipsilateral sternocleidomastoid muscle (Halmgyi & Curthoys, 2000). Thus, VEMPs indirectly measure vestibular function through a vestibulocollic reflex.

The neurophysiologic and clinical data indicate that the VEMP's are mediated by a pathway that includes the saccular macula, inferior vestibular nerve, the medial vestibular

nucleus, the medial vestibulospinal tract, and the motor neurons of the ipsilateral SCM muscle (Halmagyi & Curthoys, 2000).

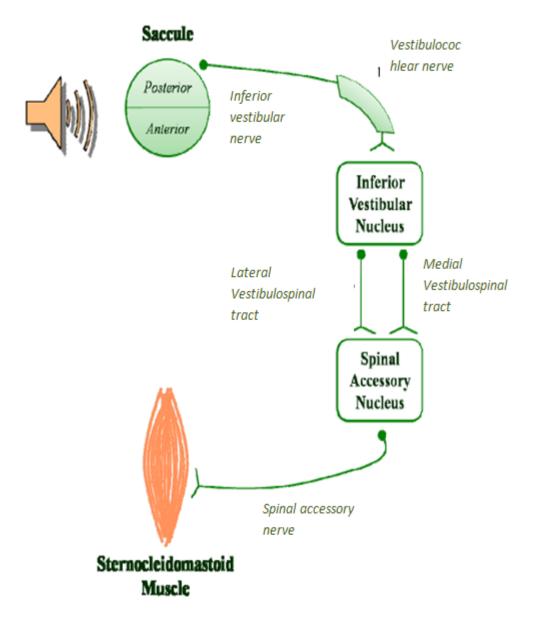


Figure- 2.1 Neural pathway for VEMP

Clinical applications of Vestibular Evoked Myogenic Potentials

Meniere's disease

In Meniere's disease VEMP is either absent or if present the amplitude is decreased without affecting the latency. Murofushi, Shimizu, Takegoshi and Cheng (2001), reported that fifteen of the 43 subjects had an absence of p13-n23 on the affected side, 7 had decreased responses, and 21 showed normal responses. No prolongation of latency of p13 & n23 was observed in the same study. Another study done by Iwasaki, Takai, Ito and Murofushi (2005) also reported abnormal VEMP responses with normal caloric test responses in 12 patients with Meniere's disease.

The interaural amplitude difference (IAD) ratio of VEMP serves for assessing the stage of Meniere's disease. Young, Huang and Cheng (2003) reported that The 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. Forty patients with unilateral definite Meniere's disease were included for the study among 40 patients, six ears were classified as stage I where VEMPs were normal in 5 and augmented in 1, with IAD ratio of -0.02 ± 0.20 , Twelve ears were classified as stage II where VEMPs were normal in 5 and augmented in 7, augmented in 2, depressed in 1, and absent in 2, with an IAD ratio of -0.12 ± 0.39 , Seventeen ears were classified as stage III where VEMPs were normal in 10, depressed in 4, and absent in 3, with an IAD ratio of -0.30 ± 0.30 , Five ears were classified as stage IV where VEMPs were normal in 2, depressed in 1, and absent in 2, with an IAD ratio of -0.54 ± 0.43 . A comparison of the IAD ratio and the stage of Meniere's demonstrated a significant relationship.

There are studies which report that the VEMP threshold is increased in Meniere's disease. Rauch et al., (2004) observed threshold shifts at all frequencies (250, 500, 1,000,

2,000, and 4,000 Hz.) in affected ears in 34 unilateral Meniere's patients when compared with 14 normal individuals. Authors concluded that alterations in vestibular evoked myogenic potential threshold support the hypothesis of altered saccular motion mechanics arising from hydropic distention.

Alterations in the tuning of VEMP response in Meniere's patients when compared to normal subjects have been reported. Rauch et al., (2004) reported largest and most consistent VEMP responses in 14 normal subjects and unaffected ears of Meniere's subjects when compared to 34 affected Meniere's ears i.e. normal subjects show a frequencydependent vestibular evoked myogenic potential threshold, with best response ("frequency tuning") at 500 Hz relative to the VEMP response at 250, 1,000, 2,000, and 4,000 Hz.) Authors concluded that alterations in vestibular evoked myogenic potential less apperent tuning in meniere's patients supports the hypothesis of altered mechanical resonance of saccular due to hydropic distention.

Acoustic Neuromas

VEMP could be useful in classifying acoustic neuromas according to the involvement of inferior or the superior vestibular nerves. Murofushi, Matsuzaki and Mizuno (1998) reported that out of 21 patients (8 men, 13 women), surgically and histopathologically confirmed acoustic neuromas, VEMP was absent on the affected side in 15 patients (71%) and significantly decreased in amplitude in 2 patients (9%). Thus, 17 (80%) of the 21 patients showed abnormal VEMPs. Three patients had abnormal VEMPs although they had normal caloric responses. Three patients had abnormal caloric responses although they had normal VEMPs. It was suggested that VEMP is able to classify acoustic neuromas based on the involvement of the inferior or the superior vestibular nerves. Absence or decreased amplitude of VEMP has been reported in patients with acoustic neuromas. Study done by Murofushi et al., (2001), showed that thirty-nine of the 62 patients showed absence of responses on the affected side, 9 showed decreased responses, and 14 showed normal responses. In other words, 48 patients (77%) showed abnormal amplitudes. Concerning p13latencies, 4 (17%) of the 23 patients with large tumors responses showed prolongation of the latency.

It has also been reported that P13 and N23 latencies of VEMP are significantly prolonged in subjects with unilateral vestibular schwannoma. Suzuki et al., (2008) recorded VEMP in 130 subjects and found that patients with unilateral vestibular schwannoma of 10 to 19 mm or of the intermediate type pathologically diagnosed by surgery, P13 and N23 latencies of VEMP are significantly prolonged compared to that of the normal opposite ear. The authors concluded that Prolonged VEMP latencies seem to be not only caused by tumor compression to the brainstem or vestibular spinal tract but also by tumor compression isolated to the inferior vestibular nerve.

Superior canal Dehiscence syndrome (SCDS)

There are studies which report a good sensitivity and specificity in the diagnosis of superior canal dehiscence syndrome (SCDS). Zhou, Gopen and Poe (2007) reported 26 subjects with dehiscence confirmed radiologically. Among these subjects, 25 had SCDS (9 had bilateral), and 1 had unilateral posterior SCDS 35 to 77 years, with an average age range of 51 ± 9 years showed that VEMP resulted in 91.4% sensitivity and 95.8% specificity. Authors concluded that vestibular evoked myogenic potential is highly sensitive and specific for SCD, possibly better than high resolution CT.

An abnormally low threshold of VEMP has been reported in patients with Superior canal Dehiscence syndrome, especially in low frequencies. Brantberg, Bergenius and Tribukait (1999) reported abnormally large sound-induced vestibular-evoked myogenic potentials (VEMP), i.e. the short latency sternomastoid muscle response especially in the frequency range 0.5–1 kHz in 3 patients with SCDS. The VEMP also had a low threshold, especially in the same frequency range. They suggested vestibular hypersensitivity to sounds was in patients with dehiscence of bone overlying the superior semicircular canal.

An abnormally low threshold in ten subjects with SCDS was also obtained by Streubel, Cremer, Carey, Weg and Minor (2001). The authors report that VEMP threshold from the dehiscent ears measured 72 ± 8 dB NHL (normal hearing level) whereas the threshold from normal control subjects was 96 ± 5 dB NHL. The diagnosis had been confirmed in each case by evoked eye movements and by high-resolution CT scans of the temporal bones that showed a dehiscence overlying the affected superior canal.

Vestibular neuritis

In vestibular neuritis, there are equivocal findings regarding absence or presence of VEMP. Murofushi et al., (2001), reported that nine (39%) of the 23 patients with vestibular neuritis had an absence of p13- n23 on the affected side, and 14 had normal responses. Concerning latencies, only 1 of the 14 patients showed prolonged p13 and none of the patients with vestibular neuritis showed prolongation of N23 latency. The authors concluded that patients with vestibular neuritis may have complete damage or no damage to the inferior vestibular nerve. Second, damage only to the vestibular nerve may be insufficient for VEMP latency prolongation beyond the normal range.

VEMP can also be used to know about the status of the inferior vestibular nerve in patients with unilateral vestibular neuritis. Ochi, Ohashi and Watanabe (2003) reported that two subjects who exhibited no VEMP response even at the highest stimulus intensity used when initially tested, one of subject of two presented recovery in neither the VEMP nor the caloric testing for 14 months after the onset. In contrast, the other patient showed a gradual recovery of VEMP during the follow-up period.

Interpretation of VEMP results in vestibular neuritis should be done cautiously in aged patients. Study done by Hong, Yeo, Kim and Cha (2008) showed that, of the134 patients with vestibular neuritis, 49 (36.6%) showed abnormal VEMP response when interpreted within the age-related normal range. They concluded that Interpretations of VEMP findings need to take into account age-related changes in the VEMP response.

Vertigo

Abnormal VEMP was observed in six patients of sudden deafness with vertigo as reported in the study by Iwasaki, Takai, Ito and Murofushi (2005). Main Outcome Measures in the study was amplitudes and latencies of the first positive-negative peak of the VEMP (p13-n23) were prolonged.

Prolongation of P13 & N23 latencies has been reported in patients with vertigo. Yang, Kim, Lee and Lee (2008) observed that VEMP latencies are increased in 41 BPPV patients, which signify neuronal degenerative changes in the macula of the saccule. The authors also reported absence of VEMP When disease progress showed a chronic and resistive course. The authors propose that VEMP could be a useful method to determine a clinical prognosis of patients with BPPV. Another study done by Hong et al., (2008) showed that sixteen (25.8%) of the 62 patients with BPPV also showed abnormal VEMP rates, when interpreted within the agerelated normal range. They concluded that Interpretations of VEMP findings need to take into account age-related changes in the VEMP response.

Noise induced hearing loss (NIHL)

Prolonged latency of VEMP response has been reported in patients with NIHL. Fakharnia, Sheibanizadeh, Jafari and Hoseini (2009), compared vestibular evoked myogenic potential findings in noise induced hearing loss patients and healthy individuals. Thirty male subjects with noise-induced hearing loss and thirty male matched controls were studied. Difference in mean latencies of p13 was significant between the two groups however the difference in n23 latency was significant only in the right ear. There was no significant difference between groups in p13-n23 amplitude. Authors concluded that inferior vestibular nerve is the susceptible part in individuals with NIHL.

Prolonged VEMP latencies and reduced peak to peak amplitude has been reported in NIHL subjects. Study done by Kumar, Vivarthini and Bhat (2010), observed VEMP in thirty NIHL subjects (55 ears). Out of the 55 ears, VEMP was absent in 16 (29.0%) ears, latency of P13 & N23 was prolonged and the peak to peak amplitude was reduced in 19 (34.6%) ears, VEMP results were normal in 20 (36.4%) ears. Authors concluded that strong possibility of vestibular system disturbances in subjects with chronic noise exposure, which can be identified by VEMP responses indicating saccular pathway abnormality.

Auditory neuropathy / Audiovestibular neuropathy

Absence of VEMP (i.e., no stimulation of saccule-collic pathway) in patients with auditory neuropathy has also been observed. Single case study done by Sheykhholeslami, Schmerber, Kyerman and Kaga (2005), reported sacculo-collic pathway dysfunction accompanying auditory neuropathy on left-ear stimulation and a biphasic response with normal latency and amplitude on right-ear stimulation in 21 year old female .

Kumar, Sinha, Bharti, Singh and Barman (2007), described VEMP in 10 subjects with auditory neuropathy, they concluded that 80% of the ears with auditory neuropathy showed abnormal VEMP results (i.e., prolonged latencies and reduced amplitude) giving an indication of high incidence of vestibular involvement in the auditory neuropathy population.

Study done by Sazgar, Yazdani, Rezazadeh and Yazdi (2010) found that out of the 8 patients (16 ears), normal VEMP response was detected in 3 ears (1 in right and 2 in left ears). There were unrepeatable VEMP waves in four ears and absent VEMPs in nine ears. The results demonstrated a centrally compensated decrease in the response of the vestibular end organs, which was associated with hearing loss. These findings imply that a subclinical well-compensated malfunction of the vestibular system is associated with the auditory destruction.

Masuda and Kaga (2011) studied Influence of aging on auditory and vestibular functions in three patients with auditory neuropathy. VEMP was one of the tests to check vestibular functions in these patients. VEMPs in 2 patients was present at p13 and n23 only on the right side and were absent on the other side. In patient 3 the VEMP response was

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absent on both sides. Authors concluded that vestibular function markedly declined with aging.

Other vestibular disorders

Among 12 sides of the 6 patients with multiple sclerosis, 3 sides showed absence of responses. All of the other sides (9 sides) showed prolongation of p13, and 4 showed prolongation of n23 in a study done by Murofushi et al., (2001). In conclusion, prolonged VEMP latencies, especially prolonged p13, would strongly suggest lesions in the vestibulospinal tract, although they are not pathognomonic for multiple sclerosis.

Abnormal VEMP responses in terms of the amplitudes and latencies of the first positive-negative peak of the VEMP (p13-n23) with normal caloric test responses was also observed in 2 patients with multiple sclerosis, same results were also obtained in 4 patients with cerebellopontine angle tumor(Iwasaki et al., 2005).

Study done by Baier, Stieber and Dieterich (2009), reported that VEMP amplitudes were significantly and bilaterally reduced compared to those of controls, but no difference was seen in the latencies in 63 patients with vestibular migraine (median age 47 years; range 24–70 years) and compared with those of 63 sex- and age-matched healthy controls.

Comparison of vestibular evoked myogenic potentials in 21 patients with multiple sclerosis and 20 normal individuals, Showed that VEMP responses were abnormal in 47.6% of patients. P13 latency and p13-n23 peak to peak amplitude in both ears showed significant difference between the two groups (p<0.05). P13 latency was delayed in 14 ears (of nine patients). VEMPs were unilaterally absent in two patients (Toufan, Jalaei Nafisi, Sheibanizade & Keyhani, 2010).

Plasticity in vestibular system

Auditory system is shaped by experience and training. Training induces neurophysiologic changes and plasticity in auditory system (Kacelnik, Nodal, Parsons & King, 2006). There are evidences from electrophysiological and behavioral methods that functional modifications occur with training (sensory experience) in normal hearing individuals, hearing loss patients, hearing aid users and cochlear implanted subjects. Also evidences from functional and structural magnetic resonance imaging revealed neurophysiologic changes in auditory system after training (Tremblay 2005; Kacelnik, et al., 2006; Christopher et al., 2006). Not only speech stimulus, but music also brings about functional and structural organization of the brain. Musicians have a variety of perceptual and cortical specializations compared non-musicians.

Motor skills are not innate but are acquired and perfected by a repetitive process of trial and error on the basis of sensory feedback. They can be considered a simple form of learning, generally referred to as "motor learning." (Green., 2000).

A mechanism of plasticity such as long-term potentiation (LTP) and long-term depression (LTD) at the cellular level and changes in the neural response with well controled behaviors has been studied. (Ito1989; Artola & Singer 1993; Bliss & Collingridge 1993; Bear & Malenka 1994; Daniel, Levens & Crepel, 1998). Vestibulo-ocular reflex (VOR) has been greatly studied for motor learning due to its relative simplicity compared with other motor control systems and the precision with which the behavioral performance of the reflex may be measured.

Modification of reflex performance was first demonstrated by Gonshor and Melvill Jones (1976a). Adaptation of the VOR gain in a variety of species, including humans has been observed by many authors through various experiments (Gauthier & Robinson 1975; Gonshor & Melvill Jones 1976a, 1976b). Also VOR adaptation has been reported in monkeys (Miles & Fuller 1974; rabbits (Ito et al. 1974; Collewijn & Grootendorst 1979), cats (Gonshor and Melvill Jones, 1976; Robinson 1976), and goldfish (Schairer & Bennett 1981; Pastor et al. 1992).

In addition to various experiments, many studies have also observed adaptation of VOR to injured states (e.g., unilateral or bilateral damage to the vestibular organs) in an effort to restore functional visual–vestibular oculomotor performance (Paige 1983; Curthoys & Halmagyi 1995; Dieringer, 1995).

Dance based therapy improves balance and gait in young and elderly individuals. Study done by Federici, Bellagamba & Rocchi (2005) showed that dance based training for three months improves balance in 20 adult and young old subjects of age range 58 to 68 yr when they compared with age matched 20 control groups using four balance tests such as Tinetti, Romberg, improved Romberg and sit up and go. Authors concluded that physical activity such as dancing will improve balance and hence it can be a useful tool in reducing the risk of falling in the elderly.

Similarly pilot study done by Alpert et al., (2009) also reported that modified jazz dance improves balance in 15 jazz classes in 13 older women of mean age of 68 years by using Sensory organization test (SOT) for balance measurements (using the NeuroCom Smart Balance Master). Post hoc analyses using paired t tests with a Bonferroni correction indicated significant increases in balance from baseline to final measurements. This finding may have significant implications for fall prevention in the postmenopausal population.

Another study done by Krampe et al., (2010) reported that dance-based therapy of six weeks affects the balance/gait of community-based frail seniors which included 11 participants. Functional Reach and Timed Get Up and Go tests were used at baseline, 6 weeks after the start of the intervention, and 6 weeks post intervention to estimate effect of dance training on balance. Authors concluded that dance based therapy improve fall risk and reduce falls.

Vestibular habituation in 13 ballet dancers was reported by Osterhammel, Terkildsen and Zilstorff (1968). Ballet dancers were examined for postrotatory responses using high velocity rotation and electronystagmography. Dancers with intense nystagmus were immune to vertigo like those with slight nystagmus. In some of the subjects the nystagmus tended to perpetuate for long periods even after the termination of the activity so authors concluded that vestibule-ocular responses following rotation is influenced by so many factors that it becomes a very unreliable measure for balancing proficiency in dancers.

Lavon, et al., (2010), found out decreased latency of the n23 wave of the VEMP response in 12 professional divers when compared with control group which consisted of 12 matched non divers, but no significant difference were found between the study and control groups in wave amplitude, asymmetry ratio and the response threshold. Authors concluded that this change may reflect adaptation of sacculo-collic reflex to the underwater environment, where there is reduction in the linear velocity and acceleration stimuli.

To summarise the review, it can be seen that VEMP has been reported to be an useful tool in the diagnosis of several vestibular disorders. There are studies which have reported an improvement in the balance of the subjects after the training. Utility of VEMP however has not been reported in studying the plasticity of the sacculocollic pathway.

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CHAPTER-3

Method

The present study was conducted with an aim of investigating plasticity of utricle and saccule using vestibular evoked myogenic potential in dancers.

Participants

Two groups of subjects participated for the study:

- Experimental group: 20 professional dancers (40 ears) in the age range of 16 to 40 years (mean age of 29.25 years) participated in the study. The experimental group was further subdivided into two groups.
 - a. Professional dancers who have received training in Kathak dance [Total 10 subjects, (7 females, 3males)]
 - b. Professional dancers who have received training in Salsa dance [Total 10 subjects, (8 females, 2 males)]

Only those dancers were included who have received the training and are continuing with the dance practice were subjected for the study.

 Control group: 15 individuals (30 ears), included 9 females and 6 males in the age range of 16 to 40 years (mean age of 24.46 years) who have not got any professional training in the dance participated in the study.

Participant Selection criteria for Experimental group:

- 1. All the participants had at least one year of dance experience/regular practice of dancing. The criteria for one year of dance practice is based on the studies which has shown that dance based training improves that balance in young adult subjects in three months (Federici, Bellagamba & Rocchi, 2005).
- Bilateral hearing sensitivity within normal limits (i.e., pure tone average of 500Hz, 1 kHz and 2 kHz should be less than ≤15 dB HL).
- No history or presence of any otological problems such as ear discharge, ear pain, itching, tinnitus etc.
- 4. No history or presence of any history of neuromuscular problem.
- 5. No history or presence of intake of drugs that may lead to vestibulotoxicity.
- 6. No history or presence of symptoms of vestibular problems such as vertigo, giddiness, nausea, blurring of vision.
- 7. No other associated neurological problems.

Participant Selection criteria for the Control group:

- 1. All the participants did not have any formal dance experience/regular practice of dancing or any training taken for the dance.
- Bilateral hearing sensitivity within normal limits (i.e., pure tone average of 500Hz, 1 kHz and 2 kHz should be less than ≤15 dB HL).
- 3. No history or presence of any otological problems such as ear discharge, ear pain, itching, tinnitus etc.

- 4. No history or presence of any history of neuromuscular problem.
- 5. No history or presence of intake of drugs that may lead to vestibulotoxicity.
- No history or presence of symptoms of vestibular problems such as vertigo, giddiness, nausea, blurring of vision.
- 7. No other associated neurological problems.

Instrumentation

- A calibrated two channel diagnostic audiometer (Orbiter 922) with TDH-39 supra aural ear phone with MX-41AR cushions, for air conduction testing and B-71 Radio ear bone vibrator, for bone conduction testing. OB922 was also used for obtaining the UCL for all the individuals.
- Calibrated Middle ear analyser (GSI- Tympstar), for tympanometry and reflexometry.
- Intelligent hearing system [I. H. S, Smart EP (4 .00 USBez) with ER-3A Insert ear phone] was used for testing of Vestibular Evoked Myogenic Potentials.

Test environment – The entire test was conducted in acoustically treated room with permissible noise levels as per ANSI S 3.1 (1991) standards.

Procedure

1. A detailed case history was obtained regarding the condition of the hearing system from all the participants.

2. Pure tone thresholds were obtained by using modified Hughson – Westlake procedure (Carhart & Jerger, 1959), for octave frequencies from 250Hz to 8 KHz for air conduction stimuli and from 250Hz to 4 KHz for bone conduction stimuli.

3. For Tympanometry 226Hz probe tone was used. Ipsilateral and contralateral reflexes at 500Hz, 1000Hz, 2000Hz and 4000Hz were obtained for both the ears.

4. UCL for all the participants was obtained by presenting speech stimuli at 100dB, Participants were instructed to respond to the stimuli by saying whether it is comfortable or not.

5. Rectified VEMPs was recorded for both the groups by an averaging of the acoustically evoked electromyogram of the sternocleidomastoid muscle. Subjects were instructed to turn their neck towards the non- stimulation ear side i.e., to rotate towards the contralateral side of the testing ear. A visual feedback was given to the participants in order to monitor their sternocleidomastoid muscle tension. The muscle tension was monitored with EMG level feedback system provided by I.H.S system. The EMG level was maintained between 100% to 200 %($50\mu\nu$ to $100\mu\nu$) for all the participants. The site of the electrode placement was prepared with skin preparation gel, silver chloride disc electrodes with conducting gel was used. Absolute electrode impedances and Inter electrode impedances of less than 5 K Ω and less than 2 K Ω respectively was maintained. Subjects were made to sit in upright position and also were instructed to tense the sternocleidomastoid muscle during runs of acoustic stimulation and relax between runs.

6. VEMP was recorded with the following protocol:

Table 3.1

Recording protocol for VEMP

| Stimulus parameters | | Acquisition parameters | | |
|---------------------|----------------------------|---------------------------------|--|--|
| Transducer | Insert ear phones ER-3A | Amplification | 5000 | |
| Type of stimulus | 500Hz tone burst | Analysis window | -10 to 70ms | |
| Intensity | 95 dBnHL | Filters30 - 1500 Hz | | |
| Polarity | Rarefaction | Electrode montage | | |
| No of sweeps | 200 | Non- Inverting (+) electrode | Upper half of sternocleidomastoid muscle | |
| Repetition Rate | 5.1/sec | Inverting (-) electrode | Strenoclavicular junction | |
| Notch filter | Off | Ground electrode | Forehead | |

500Hz tone burst stimulus was selected based on the earlier studies which show a better amplitude and response rate with 500Hz tone burst (Kumar, Sinha, Bharati & Barman, 2006; Kumar, Sinha, Bharti & Barman, 2011).

Analysis

VEMP was recorded for both the ears for all the subjects. The responses were morphologically analyzed to interpret the VEMP findings. Two recordings were obtained for the same ear to ensure reliability of the waveform. The first positive peak and the first negative peak of the biphasic wave with the latency of 13ms and 23ms was considered as p13and n23 respectively, peak to peak amplitude was calculated in order to obtain amplitude of p13-n23 complex.

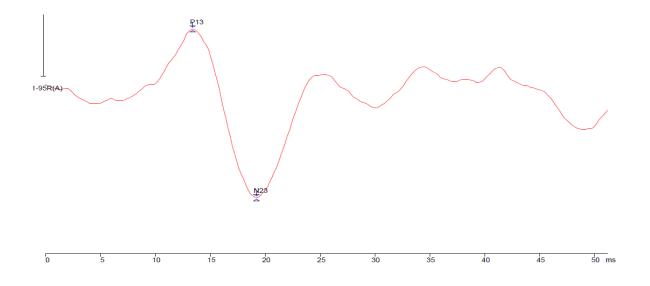


Figure 3.1 wave form of VEMP response showing P13 and N23.

- 1. Latency of P13 was analysed for the control and experimental group.
- 2. Latency of N23 was analysed for the control and experimental group.
- 3. Amplitude of P13 was analysed for the control and experimental group
- 4. Amplitude of N23 was analysed for the control and experimental group
- 5. Amplitude of P13-N23 complex was compared across the two groups.

CHAPTER -4

Results and Discussion

The latency and amplitude of different peaks of vestibular evoked myogenic potentials were analyzed for the two groups i.e. the experimental group and the control group. Latency of P13 peak, N23, amplitude of P13, N23 peak, amplitude of P13, N3 and peak to peak amplitude of P13-N23 complex were compared across the two groups.

The following statistical analyses were carried out across the group of subjects:

- 1) Descriptive statistics was done to find out the mean and standard deviation of
 - a) Latency of P13 & N23 peaks for the control and the combined data of the two subgroups of the experimental group.
 - b) Latency of P13 and N23 peaks for the two subgroups (i.e Kathak and Salsa dance groups) of the experimental group separately.
 - c) Amplitude of P13, N23 peaks and the combined data of the two subgroups of the experimental group.
 - d) Amplitude of P13, N23 peaks for the two subgroups (i.e Kathak and Salsa dance groups) of the experimental group separately.
- 2) Independent sample 'T' test was done to find out the group differences for

a). Latency of P13 & N23 peaks for combined data of the two subgroups of the experimental group.

b). Amplitude of P13, N23 peaks and peak to peak amplitude of P13-N23 complex for the control group and the two subgroups of the experimental group.

- Multiple analyses of variance (MANOVA) was done to find out significant differences for
 - a) Latency of P13 & N23 peaks for the non dancers and the two subgroups (i.e.
 Kathak and Salsa dance groups) of the experimental group separately.
 - b) Amplitude of P13, N23 peaks and peak to peak amplitude of P13-N23 complex for the non dancers and two subgroups (i.e. Kathak and Salsa dance groups) of the experimental group separately.
- 4) Duncan's post hoc analysis to understand the group differences.

Latency of P13 and N23 peaks

Vestibular evoked myogenic potentials could be recorded for all the subjects in both the control and the experimental group. P13 &N23 peaks in the waveform were visualized and analyzed for the control (non dancers) group and the experimental (dancers) group. Figure 1 shows recorded waveforms from the control (non dancers group and the experimental (dancers) group. As it can be seen from figure 1 that the latency of P13 &N23 peaks are almost similar for control (non dancers) and the experimental group (salsa and kathak dancer group).

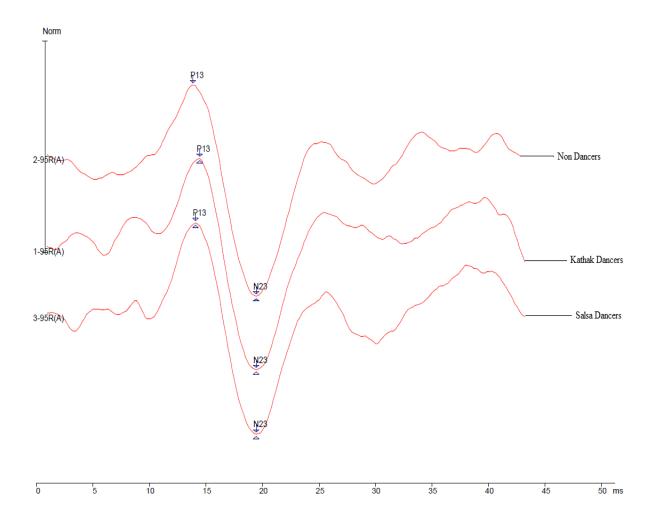


Figure 4. 1- Waveform of VEMP response showing P13 & N23 latency in non dancers and dancers.

To understand the group differences between the experimental and the control group, the data of two subgroups of the experimental group (i.e., salsa and kathak dancer group) were combined and the mean and standard deviation (S.D) were computed for the control group and the combined data of the experimental group. The mean values for the latency of P13 & N23 for both the control and the combined data of the experimental group are given in the table -4.1.

Table- 4.1

Mean and standard deviation (S.D) values of latency (msec) of control group and the experimental group.

| Parameters | P13 latency | | N23 latency | |
|--------------|-------------|------------|-------------|------------|
| Groups | Mean(msec) | S.D (msec) | Mean(msec) | S.D (msec) |
| Control | 15.84 | 2.34 | 21.39 | 2.28 |
| Experimental | 15.43 | 1.77 | 21.15 | 1.61 |

As we can see from table 4.1 that the mean latency for the control group and the combined data of the experimental group for the P13 peak and N23 peak are almost similar. However it can be noted from table 4.1 that the standard deviation in the experimental group is lesser than the control group. The same can be seen in figure 4.2

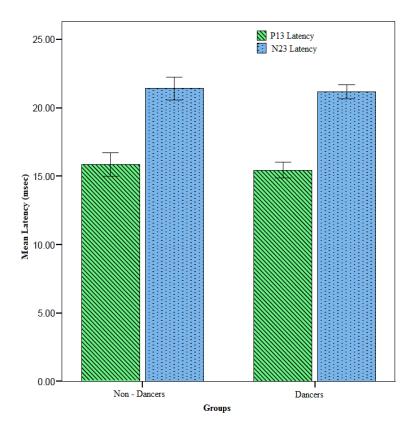


Figure 4.2- Bar graph showing latency of P13 & N23 peaks for the Control group (Non-Dancers) and the experimental group (Dancers)

To understand the significant differences in the mean values for the latency of P13 & N23 peaks, for the combined data of the experimental and the control group, Independent sample T test was done. Independent sample T test failed to show any significant differences for the latency of P13, [t (68) = 0.835, p>0.05] & N23, [t (68) = 0.516, p>0.05] between dancers and the non dancers group.

The experimental group had two subgroups (i.e. salsa and Kathak group) one of the subgroup involved Indian style of dance form and the other subgroup involved western style of dance form. To understand the differences between latency of the control group and the two subgroups of the experimental group separately, mean and S.D were calculated for each group separately.

Table-4.2

Mean and S.D of P13 and N23 latencies of VEMP responses in control group and experimental group (Kathak dancers and Salsa dancers).

| Parameters | P13 lat | tency | N23 latency | |
|----------------|-------------|------------|-------------|------------|
| Groups | Mean (msec) | S.D (msec) | Mean (msec) | S.D (msec) |
| Non dancers | 15.84 | 2.34 | 21.39 | 2.28 |
| Kathak dancers | 15.33 | 1.46 | 20.53 | 1.26 |
| Salsa dancers | 15.53 | 2.07 | 21.78 | 1.71 |
| | | | | |

As it can be seen from the table -4.2 that mean latency value for P13 & N23 peaks are almost similar for the control group and the two subgroups of the experimental group. It can also be seen from table that the standard deviation is again less for the latency of both P13 as well as N23 peaks for the two subgroups of the experimental group. The same can be seen from figure 4.3

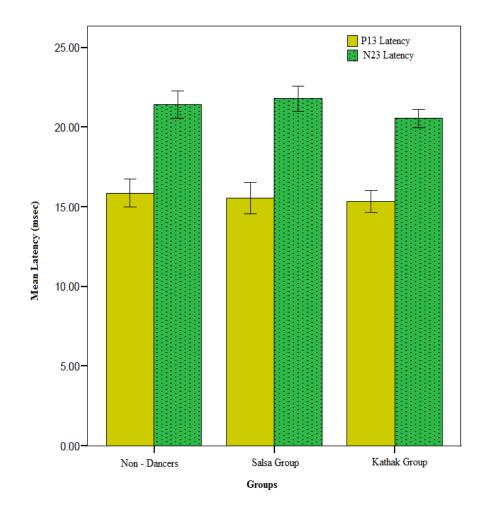


Figure 4.3- Bar graph showing latency of P13 & N23 peaks for the three groups.

To further understand the differences between the control and the experimental subgroups groups i.e., the non dancers, dancer kathak group and dancer salsa group, multiple analyses of variance (MANOVA) was done. Multiple analyses of variance failed to show any significant differences between the non dancers, dancer kathak group and dancer salsa group for latency of P13 [F (2, 67) = 0.39, P>0.05] and also for the latency of N23 [F (2, 67) = 2.34, P>0.05].

The mean values for the latency for both the experimental and control group was approximately 15.5 msec for P13 latency and for the N23 peaks the latency varied between 20.53 msec to 21.5 msec. Previous studies have reported a mean latency between 13 msec to 14.5 msec for the P13 latency whereas, for the N23 the mean value for the latency reported was around 21 to 23 msec (Wang & Young, 2003; Wang &Young 2006; Isaradisaikul et al. 2008). However, Manasa (2009) and Vijayshankar (2008) have reported a similar latency as obtained in the present study. The protocol used and the instrumentation used in the present study is same as that of used by Manasa (2009) and Vijayshankar (2008), whereas other studies have used a different protocol and different instrument to record the VEMP. The difference in the latency of VEMP from the study by Wang and Young, (2003), Isaradisaikul et al., (2008) could be due to the fact that the instrumentations used and the calibration differences in recording may be different from the present study.

There was no difference in the latency of P13 peaks or N23 peaks among the control versus experimental groups. Various studies have reported that latency parameter of VEMP is relatively less subject to undergo changes than amplitude and threshold of VEMP response (Faith et al., 2004). Faith et al., (2004) demonstrated that there was no effect of any of the stimulus parameters (i.e., stimulus level, stimulus frequency, and tonic EMG level) on latency of P13 & N23 of VEMP response.

Other studies which involved the study of degeneration process of the sacculocollic pathways have also reported no significant change in the latency parameters compared to the amplitude parameters (Welgampola & Colebatch, 2001; Sun Kyu Lee, et al., 2007; Kumar et al., 2007). Even the data which represents the different pathological conditions have reported no change in latency (Murofushi et al. 2001; Young, Huang & Cheng, 2006; Robert, Todd &

Daniel, et al., 2009). Thus, the no change in latency between the control group and experimental group could be due to the fact that the latency of the VEMP may not show a significant change in the latency parameters.

Amplitude of P13, N23, and P13-N23 complex

Amplitude of P13, N23 peak, amplitude of P13, N3 and peak to peak amplitude of P13-N23 complex in the waveform analyzed for the non dancers group and the two dancers group.

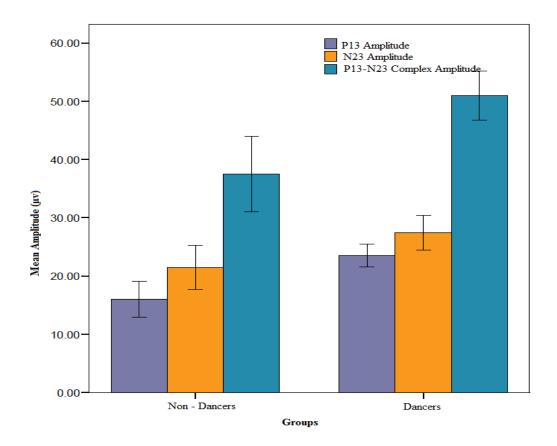
First the data of the two subgroups of the experimental group (i.e., salsa and kathak dance groups) were combined. This was done in order to understand the overall difference between the amplitude of the P13 peak, N23 peak and the P13-N23 complex for the control and the experimental group. Mean and standard deviation for amplitude of P13, N23 peak and peak to peak amplitude of P13-N23 complex of VEMP responses for experimental and control groups were computed and the details are given in table - 4.3.

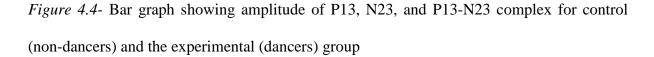
Table-4.3

Mean and S.D values of amplitude of P13, N23 and peak to peak amplitude of P13-N23 Complex of dancers and the non dancers group.

| Parameters | P13 Amplitude | | N23 Am | plitude | P13-N23 Amplitude | |
|--------------|---------------|---------|----------|---------|-------------------|---------|
| Groups | Mean(µv) | S.D(µv) | Mean(µv) | S.D(µv) | Mean(µv) | S.D(µv) |
| Control | 16.02 | 8.38 | 21.46 | 10.10 | 37.47 | 17.36 |
| Experimental | 23.51 | 6.18 | 27.42 | 9.19 | 50.96 | 13.21 |

It can be seen from table 4.3 that the amplitude of the VEMP responses in the experimental group for P13, N23 and also for the P13-N23 complex is much higher compared to the control group. Additionally it can also be seen that the standard deviation for the experimental group is again low compared to the control group. The same can be seen in the figure 4.4 also





To find out the significant differences in the mean values for amplitude of P13, N23 and peak to peak amplitude of P13-N23 complex for the control and the experimental group, independent sample T test was done. Independent sample T test showed significant differences for the amplitude of P13 peak [t (68) = 4.309, p<0.05],

N23 peak, [t (68) = 2.572, p<0.05] and peak to peak amplitude of P13-N23 complex [t (68) = -3.694, p<0.05] between the control and the experimental groups.

To understand the differences for the amplitude of the control group and the two subgroups of the experimental group separately, mean and S.D were calculated for each group separately. Mean and standard deviation values for amplitude of P13, N23 peak and peak to peak amplitude of P13-N23 complex for the control group (non-dancer) and the two subgroups of the experimental group (Kathak and the salsa group) are given in the table - 4.4.

Table- 4.4

Mean and S.D of P13 and N23 latencies of VEMP responses in non dancers and dancers (kathak dancers and salsa dancers).

| | Parameters | P13 Amplitude | | N23 Amplitude | | P13-N23 | |
|---------|------------|---------------|---------|---------------|---------|-----------|---------|
| | | | | | | Amplitude | |
| Groups | | Mean(µv) | S.D(µv) | Mean(µv) | S.D(µv) | Mean(µv) | S.D(µv) |
| Control | Non | 16.02 | 8.38 | 21.46 | 10.10 | 37.47 | 17.36 |
| | dancers | | | | | | |
| N | Kathak | 25.72 | 5.14 | 28.28 | 11.37 | 53.99 | 14.96 |
| Experi- | dancers | | | | | | |
| mental | } | | | | | | |
| | Salsa | 21.31 | 6.46 | 26.61 | 6.53 | 47.92 | 10.71 |
| , | dancers | | | | | | |

It can be seen from table - 4.4 that mean amplitude value for amplitude of P13, N3 and peak to peak amplitude of P13-N23 complex are higher for the two subgroups of the experimental group (Kathak and salsa dance group) when compared to the control group. It can also be seen from table that the mean amplitude value of P13, N23 and P13-N23 complex is higher for Kathak dancers compared to the salsa dancers and non dancers. The same can be seen from figure 4.5 also.

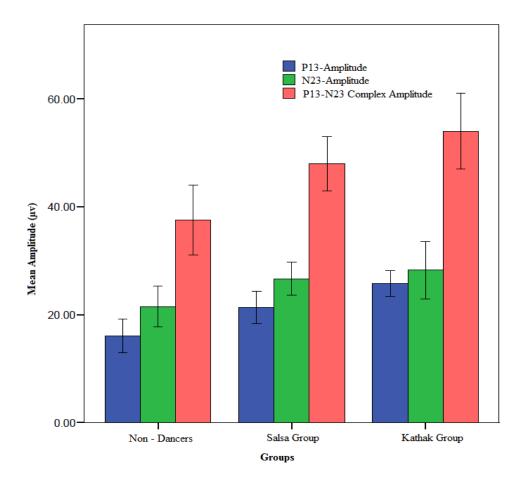


Figure 4.5- Bar graph showing amplitude of P13, N23 and P13-N23 complex peaks for the three groups

To further understand the differences between groups i.e., the control group and the two subgroups of the experimental group (dancer kathak group and dancer salsa group), multiple analyses of variance (MANOVA) was done. Multiple analyses of variance showed significant differences between the non dancers, dancer kathak group and dancer salsa group for the amplitude of P13 [F (2, 67) = 11.63, P<0.05], amplitude of N23 [F (2, 67) = 3.41, P<0.05] and also amplitude of P13-N23 complex [F (2, 67) = 7.69, P<0.05].

Ducan's post hoc analysis was done to see which of the groups were significantly different. Results of the Duncan's post Hoc test are given in table 4.5 below-

Table-4.5

Duncan's post Hoc analysis for the amplitude of the VEMP parameters

| | P13 | | N23 | | P13-N23 | |
|---------|--------|--------|--------|--------|---------|--------|
| | Kathak | Salsa | Kathak | Salsa | Kathak | Salsa |
| Control | P<0.05 | P<0.05 | P<0.05 | P<0.05 | P<0.05 | P<0.05 |
| Kathak | | P<0.05 | | P>0.05 | | P<0.05 |

To summarise the results, there was no significant difference in terms of P13 and N23 latency of VEMP response in both the control and the experimental group. However, there was significant difference in the amplitude of P13, N23 peaks & peak to peak amplitude of P13-N23 complex of VEMP response in both non dancer group and dancer group i.e., amplitude was significantly higher in the experimental group compared to the control group.

There were statistically significant differences in the amplitude of P13, N23 and peak to peak amplitude of P13-N23 complex of VEMP responses between experimental and control groups. Increased P13, N23 amplitude and peak to peak amplitude of P13-N23 complex of VEMP responses were seen in experimental group than control group. Mean amplitude value of P13, N23 and peak to peak amplitude of P13-N23 complex for the experimental and control group was 23.51 (6.18), 27.42 (9.91) and 50.96 (13.21) & 16.02 (8.38), 21.46 (10.10) and 50.96 (13.21) respectively. The amplitude obtained for the control group is almost similar to reported by Manasa (2009) and Vijay Shankar (2008).

Most of the studies reported that amplitude parameter of VEMP is relatively more subject to undergo changes than the latency of VEMP response. Pathological studies have also reported that the amplitude of VEMP is reduced or abnormally high (i,e., amplitude is more prone to undergo changes than latency) such as meniere's disease (Murofushi et al., 2001; Iwasaki, Takai, Ito & Murofushi, 2005), acoustic neuromas (Murofushi, Matsuzaki & Mizuno, 1998; Murofushi, Shimizu, Takegoshi & Cheng, 2001), vestibular neuritis (Murofushi et al , 2001), vertigo (Iwasaki, Takai, Ito and Murofushi, 2005). Many behavioral studies reported that dance based training improves that balance in young and adult subjects in three months (Federici, Bellagamba & Rocchi, 2005; Alpert et al, 2009; Krampe et al, 2010). Thus, the differences in amplitude of the P13, N23 and P13-N23 could be because that the amplitude changes in the experimental group would have been more compared to the latency of the VEMP.

The results obtained here are just a preliminary report which indicates possible sacculocollic pathway plasticity because of the regular practice of the dance. The significant improvement in amplitude of the VEMP responses could be due to the fact that the dance

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requires more balance activity and thus would have resulted in a more responsive vestibular system in the dancers compared to the non-dancers. The plasticity in the vestibular system would have occurred in different anatomical structure which can be measured with the other techniques such as Electronystagmography. Here in the present study only otolith organs were assessed. Similar to the plasticity in other vestibular structures, the plasticity of the otolith organs would have occurred in dancers. Thus, the otolith organs would have become more responsive and an improved functioning of these structures would have resulted in improved amplitude responses of the vestibular evoked myogenic potentials. One more thing to be noted in the present study is that the individuals who had got training in the Indian style of dance i.e. salsa dance pattern. At this stage it is not possible to define why the amplitude of the VEMP was more for the dancers of the Indian style compared to the western dancers.

Chapter-5

Summary and Conclusion

The human vestibular endorgans consist of utricles, saccules and semicircular canals. The saccules and utricles contain otoliths that are sensitive to gravity and are slightly sensitive to sounds, as well. Vestibular evoked myogenic potential (VEMP) is a clinical test for vestibular disorders, and is deduced to be produced by the sacculo-collic reflex. VEMP is recorded from the ipsilateral tonically contracting sternocleidomastoid (SCM) muscle while monoaurally stimulating with loud short tone burst (STB) or click sounds. A successful VEMP response depends on the adequate energy transfer of sound through the middle ear, oval window, and vestibule. Then a reflex is generated by a disynaptic pathway, beginning in the saccule macula via the inferior vestibular nerve, lateral vestibular nucleus, medial vestibulospinal tract, and finally ending at the motor neurons of the SCM muscle. A typical VEMP response includes 2 biphasic waves. The first wave is believed to be generated by vestibular afferents arising from the saccule and peaks at a latency near 13 ms (p13). The trough is approximately at 23 ms (n23).

Plasticity of the auditor system is not a new entity. Various studies have reported a change in the response of the auditory system after training (Tremblay 2005; Christopher et al., 2006; Kacelnik, Nodal, Parsons & King, 2006). Similarly a remarkable plasticity has been noted in the vestibular system throughout life. There are various reports which suggest that dance based training improves the balance function in young as well as adult subjects (Federici, Bellagamba & Rocchi 2005; Krampe, et al., 2010). In a recent study by Lavon et al., (2010), it has been shown that there is plasticity not only in the VOR system but also the utricle and the saccule.

The present study was conducted with an aim of studying the plasticity in the sacculocollic pathway. To arrive at the aim two groups of subjects were selected-Control group and the Experimental group.

1. Experimental group consisted of 20 professional dancers (40 ears) in the age range of 16 to 40 years (mean age of 29.25 years). The experimental group was further subdivided into two groups.

- Professional dancers who have received training in Kathak dance [Total 10 subjects, (7 females, 3males)]
- Professional dancers who have received training in Salsa dance [Total 10 subjects, (8 females, 2 males)]
- Control group consisted of 15 individuals (30 ears), included 9 females and 6 males in the age range of 16 to 40 years (mean age of 24.46 years) who have not got any professional training in the dance participated in the study.

Subjects in both the groups had no history or any evidence of any otological or neurological problems. Additionally participants in the experimental group had minimum of one year regular dance practice. Vestibular evoked myogenic potentials were recorded in both the groups and following analysis was done-

- 1. Latency of P13 was analysed for the control and experimental group.
- 2. Latency of N23 was analysed for the control and experimental group.
- 3. Amplitude of P13 was analysed for the control and experimental group
- 4. Amplitude of N23 was analysed for the control and experimental group
- 5. Amplitude of P13-N23 complex was compared across the two groups.

To analyse the data following statistics were done-

- 1) Descriptive statistics was done to find out the mean and standard deviation of
 - a) Latency of P13 & N23 peaks for the control and the combined data of the two subgroups of the experimental group.
 - b) Latency of P13 and N23 peaks for the two subgroups (i.e Kathak and Salsa dance groups) of the experimental group separately.
 - c) Amplitude of P13, N23 peaks and P13-N23 complex of the combined data of the two subgroups of the experimental group.
 - d) Amplitude of P13, N23 peaks for the two subgroups (i.e. Kathak and Salsa dance groups) of the experimental group separately.
- 2) Independent sample 'T' test was done to find out the group differences for

a). Latency of P13 & N23 peaks for combined data of the two subgroups of the experimental group.

b). Amplitude of P13, N23 peaks and peak to peak amplitude of P13-N23 complex for the control group and the two subgroups of the experimental group.

- Multiple analyses of variance (MANOVA) was done to find out significant differences for
 - a) Latency of P13 & N23 peaks for the non dancers and the two subgroups (i.e.
 Kathak and Salsa dance groups) of the experimental group separately.

- b) Amplitude of P13, N23 peaks and peak to peak amplitude of P13-N23 complex for the non dancers and two subgroups (i.e. Kathak and Salsa dance groups) of the experimental group separately.
- 4) Duncan's post hoc analysis to understand the group differences.

Results of the study revealed the following-

- 1. The latency of the P13 and N23 peaks were similar for the control and the experimental groups.
- The amplitude of the P13 peaks and N23 peaks was more for the experimental group (i.e. dancer group) compared to the control group (non-dancers).
- 3. The amplitude of the P13-N23 complex was more for the experimental group (i.e. dancer group) compared to the control group (non-dancers).
- 4. Among the experimental group the kathak dancers had more amplitude for the P13 and N23 peaks compared to the salsa group dancers.
- 5. For the P13- N23 complex amplitude among the experimental groups, the kathak dancers had more amplitude compared to the salsa group dancers.

Better amplitude of vestibular evoked myogenic potentials in dancers was attributed to a possible better responsive sacculocollic pathway compared to non-dancers.

Conclusions:

- 1. The dance practice may improve the balance function.
- 2. Amplitude of the VEMP parameters is a better tool to study the plasticity of sacullocollic pathway compared to the latency parameters.

Implications of the Study

- 1. The study provides a thrust to long felt need for research in the field of vestibular assessment.
- 2. Present study opens a new research era in understanding the plasticity of the sacculocollic pathway. The vestibular evoked myogenic potentials may give an indication of the plasticity of the vestibular system.
- 3. Dance can be a major or can be a part (acting as additional help/support) to improve balance and gait problems.
- 4. Based on the present study some therapy techniques can be developed so that the individuals with balance problem get benefitted with the dance based therapy.
- 5. Vestibular rehabilitation therapy (VRT) exercises are typically based on principles of vestibular adaptation of semicircular canal input. If otolith organ involvement is identified, then VRT exercises designed to stimulate otolithic adaptation may be more effective for managing a patient's symptoms.

Future directions:

- 1. The study has been done with a smaller data. So, the study can be replicated with the larger data.
- 2. The study can be replicated in other populations where they requires more balancing act such as Divers and Gymnasts.
- 3. Other tests of vestibular assessment such as electronystagmography can be combined in order to understand the plasticity of the major part of the vestibular system.

References

- Akin, F. W., Murnane, O. D., & Proffitt, T. M. (2003). The effects of click and tone-burst stimulus parameters on the vestibular evoked myogenic potential (VEMP). *Journal of the American Academy of Audiology, 14,* 500–509.
- Al-Abdulhadi, K., Zeitouni, A. G., Al-Sebeih, K., & Katsarkas, A. (2002). Evaluation of vestibular evoked myogenic potentials. *The Journal of Otolaryngology*, 31, 93–96.
- Alpert, P.T., Miller, S.K., Wallmann, H., Havey, R., Cross, C., Chevalia, T., et al. (2009).
 The effect of modified jazz dance on balance, cognition, and mood in older adults.
 Journal of American Academy Nurse Practice, 21(2), 108-15.
- American National Standards Institute (1991). American National Standards for maximum permissible ambient noise levels for audiometric test room. (ANSI S3.1- 1991). New York: American National Standards Institute.
- Artola, A., & Singer, W. (1993). Long-term depression of excitatory synaptic transmission and its relationship to long-term potentiation. *Trends in Neuroscience*, 11, 480–487.
- Baier, B., Stieber N., & Dieterich, M. (2009). Vestibular-evoked myogenic potentials in vestibular migraine. *Journal of Neurology*, 52, 5132-5134
- Bear, M. F., & Malenka, R.C. (1994). Synaptic plasticity: LTP and LTD. Current Opinion in Neurobiology, 4, 389–399.
- Bliss, T. V., & Collingridge, G. L. (1993). A synaptic model of memory: long-term potentiation in the hippocampus. *Nature*, *361*, 31–39.

- Brantberg, K., Bergenius, J., & Tribukait, A. (1999). Vestibular- evoked myogenic Potentials in patients with dehiscence of the superior semicircular canal. Acta Otolaryngologica, 119, 633–640.
- Carhart, R., & Jerger, J. F. (1959). Preferred Method for Clinical Determination of Pure tone Thresholds. *Journal of Speech and Hearing Research*, 24, 330.
- Chen, C. W., Young, Y. H., & Wu, C. H. (2000). Vestibular neuritis: Three-dimensional videonystagmography and vestibular evoked myogenic potential results. *Acta Otolaryngologica*, 120, 845–848.
- Christopher, S., Bowman, G. A., Yund, W., Herron, T. J., Christina, M., Woods, D. L.et al. (2006). Perceptual training improves syllable identification in new and experienced hearing aid users. *Journal of Rehabilitation Research & Development.* 43, 537-552.
- Clarke, A. H., Schonfeld, U., & Helling, K. (2003). Unilateral examination of utricle and saccule function. *Journal of Vestibular Research*, *13*, 215–225.
- Colebatch, J. G., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click-evoked vestibulocolic reflex. *Journal of Neurology, Neurosurgery, and Psychiatry, 57,* 190–197.
- Collewijn, H., & Grootendorst, A. F. (1979). Adaptation of optokinetic and vestibuloocular reflexes to modified visual input in the rabbit. *Progressive Brain Research*, 50, 772– 781.
- Curthoys, I.S., & Halmagyi, G.M.(1995) Vestibular compensation: a review of the oculomotor, neural, and clinical consequences of unilateral vestibular loss. *Journal of Vestibular Research*, *5*, 67–107.

- Daniel, H., Levenes, C., & Crepel, F. (1998). Cellular mechanisms of cerebellar LTD. *Trends in Neuroscience*, *21*, 401–407.
- Dieringer, N. (1995) "Vestibular compensation": neural plasticity and its relations to functional recovery after labyrinthine lesions in frogs and other vertebrates. *Progressive Neurobiology*, 46, 97–129.
- Faith A.W., Murnane, O. D., Panus, P. C., Caruthers, S. K., Wilkinson, A. E., & Proffit, T. M.(2004). The influence of voluntary tonic EMG level on the vestibular evoked myogenic potential. *Journal of Rehabilitation Research & Development*, 41(3), 473–480.
- Fakharnia, F., Sheibanizadeh, A., Jafari, Z., & Hoseini, F. (2009). Comparison of vestibular evoked myogenic potential and caloric tests findings in noise induced hearing lossaffected and healthy individuals. *Audiology*, 18(1-2), 70-80.
- Fedrici, A., Bellagamba., S., & Rocchi, M. B. L. (2005). Does dance based training improves balance in adult and young old subjects? A pilot randomized controlled trial. Aging Clinical and Experimental Research, 17, 385-389.
- Ferber-Viart, C., Duclaux, R., Colleaux, B., & Dubreuil, C. (1997). Myogenic vestibularevoked potentials in normal subjects: A comparison between responses obtained from sternomastoid and trapezius muscles. *Acta Otolaryngologica*, 117, 472–481.
- Gauthier, G. M., & Robinson, D. A. (1975). Adaptation of the human vestibuloocular reflex to magnifying lenses. *Brain Research*, *92*, 331–335.
- Gittis, A., H., & Sascha, D. L. (2006).Intrinsic and synaptic plasticity in the vestibular system. *Neurobiology*, *16*, 385-390.

- Gonshor, A., & Melvill, G. (1976a). Shortterm adaptive changes in the human vestibuloocular reflex arc. *Journal of Physiology (London)*. 256, 361–379.
- Gonshor, A., & Melvill, G. (1976b). Extreme vestibuloocular adaptation induced by prolonged optical reversal of vision. *Journal of Physioliology (London), 256, 381–414.*
- Green., A. M. (2000). Visual–vestibular interaction in a bilateral model of the rotational and translational vestibulo-ocular reflexes: an investigation of viewing-context dependent reflex performance. Ph.D. Thesis, McGill University, Montreal, Canada.
- Halmagyi, G. M., & Curthoys, I. (2000). Clinical testing of otolith functions. New York Academy of Sciences, 871, 195-204.
- Hong, S. M., Yeo, S., Kim, C., & Cha, T. (2008). The results of vestibular evoked myogenic potentials, with consideration of age-related changes, in vestibular neuritis, benign paroxysmal positional vertigo, and Meniere's disease. *Acta Oto-Laryngologica*, 128, 861-865.
- Isaradisaikul, S., Strong, D. A., Moushey, J. M., Gabbard, S. A., Ackley, S. R., & Jenkins,
 H. A. (2008). Reliability of vestibular evoked myogenic potentials in healthy subjects. *Otology & Neurotolgy*, 29, 542–4.
- Ito., M. (1989). Long-term depression. Annual Review in Neuroscience, 12, 85–102.
- Iwasaki,S., Takai, Y., Ito, K., & Murofushi, T. (2005). Abnormal Vestibular Evoked Myogenic Potentials in the Presence of Normal Caloric Responses. *Otology & Neurotology*, 26, 1196–1199.
- Kacelnik, O., Nodal, F. R., Parsons, C. H., & King, A. J. (2006). Training Induced Plasticity of Auditory Localization in Adult Mammals. *PloS Biology*, 4(4), 104.

- Kumar, K., Vivarthini, C.J., & Bhat, J.S. (2010). Vestibular evoked myogenic potential in noise-induced hearing loss. *Noise and Health*, 12(48), 191-194.
- Krampe, J., Rantz, M. J., Dowell, L., Schamp, R., Skubic, M., & Abbott, C. (2010). Dancebased therapy in a program of all-inclusive care for the elderly: an integrative approach to decrease fall risk. *Journal of Nursing Administration Quarterly*, 34(2), 156-61.
- Kumar, K., Sinha, S. K., Bharti, A. K., & Barman, A. (2006). *Comparison of Vestibular* evoked myogenic potentials elicited by click & short duration one burst. Research paper presented at 2nd south zonal conference at Kerala on 14th of May 2006.
- Kumar, K., Singh, N. K., Sinha, S, K., Bharti, A., & Barman. A. (2007). Vestibular evoked myogenic potentials as a tool to assess vestibule colic pathway dysfunction in individuals with auditory neuropathy. *Asia pacific journal of Speech, Language and Hearing*, 10(3), 110-118.
- Kumar, K., Sinha, S. K., Bharti, A. K., & Barman, A. (2011). Comparison of vestibular evoked myogenic potentials elicited by click & short duration tone burst. *Journal of Laryngology and Otology*, 125, 343-347.
- Rodith, R. E., Robert, W, E., Todd, B. S., & Daniel, J.L. (2009). Cervical vestibular evoked myogenic potentials (cVEMPs) in patients with superior canal dehiscence syndrome (SCDS). *Otolaryngology- Head and Neck Surgery*, 141, 24-28.
- Lavon, H., Dror, T., Gil, K., Dov, H., & Avi, S. (2010). Vestibular Evoked Myogenic Potentials and Saccular Plasticity in Divers. Aviation, Space, and Environmental Medicine, 81(2), 103-106.

- 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.
- Manasa, M. (2009). Vestibular Evoked Myogenic Potentials (VEMP) in individuals with noise induced hearing loss (NIHL). Unpublished dissertation submitted to the University of Mysore, Mysore, India.
- Masuda, T., & Kaga, K. (2011). Influence of aging over 10 years on auditory and vestibular functions in three patients with auditory neuropathy. *Acta Oto-laryngologica*, 131, 562-568.
- McCue, M. P., & Guinan, J. J., Jr. (1995). Spontaneous activity and frequency selectivity of acoustically responsive vestibular afferents in cat. *Journal of Neurophysiology*, 72, 1563–1572.
- Milles, F. A., Fuller, J. H. (1974). Adaptive plasticity in vestibuolo-ocular reflex of monkeys. *Brain Reasearch.*, 80, 512-516.
- Murofushi, T., Matsuzaki, M., & Mizuno, M. (1998). Vestibular evoked myogenic potentials in patients with acoustic neuromas. *Archives of Otolaryngology-Head & Neck Surgery, 124,* 509–512.
- Murofushi, T., Shimizu, K., Takegoshi, H., & Cheng, P. W. (2001). Diagnostic value of prolonged latencies in the vestibular evoked myogenic potential. Archives of Otolaryngology-Head & Neck Surgery, 127, 1069–1072.
- Ochi, K., Ohashi, T., & Nishino, H. (2001). Variance of vestibular evoked myogenic potentials. *Laryngoscope*, *111*, 522–527.

- Ochi, K., Ohashi., T., & Watanabe, S. (2003). Vestibular-evoked myogenic potential in patients with unilateral vestibular neuritis: abnormal VEMP and its recovery. *The Journal of Laryngology & Otology*, *117*, 104–108.
- Osterhammel, P., Terkildsen, K., & Zilstorff, K. (1968).Vestibular habituation in ballet dancers. *Acta Oto-laryngologica*, 66, 221-228.
- Pastor, A. M., De La Cruz, R.R., & Baker, R. (1992). Characterization and adaptive modification of the goldfish vestibuloocular reflex by sinusoidal and velocity step vestibular stimulation. *Journal of Neurophysiology*, 68, 2003–2015.
- Piage, G. D. (1983). Visiual induced plasticity in human vestibuolo-ocular reflex. *Brain* Research, 80, 25-35.
- Rauch, S. D., Zhou, G., Kujawa, S. G., Guinan, J. J., & Herrmann, B. S. (2004). Vestibular evoked myogenic potentials show altered tuning in patients with Meniere's disease. *Otology & Neurotology*, 25, 333–338.
- Robinson, D. A. (1976). Adaptive gain control of vestibulo-ocular reflex by the cerebellum. *Journal of Neurophysiology*, *39*, 954–969.
- Sazgar, A. A., Yazdani, N., Rezazadeh, R., & Yazdi, A. K (2010) Vestibular evoked myogenic potential (vemp) in patients with auditory neuropathy: auditory neuropathy or audiovestibular neuropathy?. *Acta oto-laryngologica, 130*, 1130-1134.
- Schairer, J. O., & Bennett, M. V. L. (1981). Cerebellectomy in goldfish prevents adaptive gain control of the VOR without affecting the optokinetic system. In: Gualtierotti T (ed) The Vestibular System: Function and Morphology. New York: Springer-Verlag, pp. 463–477.

- Sheykhholeslami, K., Schmerber, S., Kyerman, M. H., & Kaga, K (2005). Sacculo-collic pathway dysfunction accompanying auditory neuropathy. *Acta Oto-laryngologica*, 125, 786-791.
- Streubel, S. O., Cremer, P. D., Carey, J. P., Weg, N., & Minor, L. B. (2001). Vestibularevoked myogenic potentials in the diagnosis of superior canal dehiscence syndrome. *Acta Otolaryngologica* (Suppl. 545), 41–49.
- Sun, K. L., Chang, C., Tae, S. J., Dong C, P., & Seung, G. Y. (2007). Age-related differences in parameters of vestibular evoked myogenic potentials. *Acta Otolaryngologica*, 128(1), 66-72.
- Suzuki, M., Yamada, C., Inoue, R., Kashio, A., Saito, Y., & Nakanishi, W. (2008). Analysis of Vestibular Testing in Patients with Vestibular Schwannoma Based on the Nerve of Origin, the Localization, and the Size of the Tumor. *Otology & Neurotology*, 29, 1027-1031.
- Toufan, R., Jalaei, B., Nafisi, S., Sheibanizade, A., & Keyhani, M. R (2010).Comparison of vestibular evoked Myogenic potentials in patients with multiple sclerosis and normal individuals. *Audiology*, 19(2), 9-17.
- Tremblay, K. L. (2005). Training-Related Changes in the Brain: Evidence from Human Auditory-Evoked Potentials. *Seminars in Hearing*, 28, 120-132.
- Vijay Shankar, G. (2008). Effect of mode of sternocleidomastoid (SCM) excitation on Vestibular Evoked Myogenic Potentials (VEMP). Unpublished dissertation submitted to the University of Mysore, Mysore, India.

- Wang, S. J., & Young, Y. H. (2003). Vestibular evoked myogenic potentials using simultaneous binaural acoustic stimulation. *Hearing Research*, 185, 43–8.
- Wang, C. T., & Young, Y. H. (2006). Comparison of the head elevation versus rotation methods in eliciting vestibular evoked myogenic potentials. *Ear and Hearing*, 27, 376–81.
- Welgampola, M.S., & Colebatch, J.G. (2001). Characteristics of tone burst-evoked myogenic potentials in the sternocleidomastoid muscles. *Otology & Neurotology*, 22, 796–802.
- Yang, W., Kim, S. H., Lee, J. D., & Lee, W. (2008). Clinical Significance of Vestibular Evoked Myogenic Potentials in Benign Paroxysmal Positional Vertigo. *Otology & Neurotology*, 29, 1162-1166.
- Young, Y. H., Huang, T. W., & Cheng, P. W. (2003). Assessing the stage of Meniere's disease using vestibular evoked myogenic potentials. Archives of Otolaryngology-Head & Neck Surgery, 129, 815–818.
- Zhaou, G., Gopen, Q., & Poe, D. S. (2007). Clinical and Diagnostic Characterization of Canal Dehiscence Syndrome: A Great Otologic Mimicker. *Otology & Neurotology*, 28, 920-926.