Effect of Dance Training on Vestibular Evoked Myogenic Potentials

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Abstract

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. The present study was conducted with an aim of studying plasticity of the sacculocollic pathway in professional dancers who are receiving dance training. Two groups of subjects- control group and experimental group participated in the study. Experimental group was further subdivided in to two subgroups- Professional dancers who have received training in Kathak dance, and professional dancers who have received training in salsa dance. Experimental group consisted of total 20 subjects (40 ears), 10 (20 ears) in each subgroup. Control group consisted of 15 individuals (30 ears). Results revealed an enhancement of amplitude of P13, N23 and P13-N23, in professional dancers compared to the normal control group. Results however, did not reveal any significant difference for the latency of P13, and N23 peaks in the two groups. The difference in amplitude between the two groups was attributed to the sacculocollic pathway in dancers.

Key words: *Vestibular Evoked Myogenic Potentials (VEMP), dancers, plasticity*

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 (VEMP) testing. This newly developed procedure augments the management of dizzy patients by increasing specificity when investigating the site of lesion.

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; Ferber-Viart, Duclaux, Colleaux, & Dubreuil,1997; Chen, Young, & Wu, 2000; Ochi, Ohashi, & Nishino, 2001; Welgampola, & Colebatch, 2001; 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, & N44).

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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 (Iwasaki, Takai, Ito, & Murofishi, 2005), Multiple sclerosis, (Murofushi, Shimizu, Takegoshi, & Cheng, 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 (Gittis & Sascha, 2006). 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, H., Dror, T., Gil, K., Dov, H., & Avi, S. (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. Thus, the present study was conducted with an aim of scientifically investigating plasticity of utricle and saccule using vestibular evoked myogenic potential in dancers.

Method

Participants

Two groups of subjects participated for the study: an experimental group and a control group.

The experimental group, consisted of 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, and 3 males)].
b. Professional dancers who have received training in Salsa dance [Total 10 subjects, (8 females, 2 males)] Only those dancers who have received the training and are continuing with the dance practice were subjected for the study.

The control group, on the other hand had 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). They had not got any professional training in the dance.

Participant selection criteria for Experimental group: 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). The participants hearing sensitivity was within normal limits (i.e., puretone average of 500 Hz, 1 kHz & 2 kHz was less than ≤15 dBHL). None of the participants had history or presence of any otological problems such as ear discharge, ear pain, itching, tinnitus. Additionally they did not have history or presence of any neuromuscular problem, history or presence of intake of drugs that may lead to vestibulotoxicity, and had no symptoms related to vestibular disorders.

Participant Selection criteria for the Control group: The participants in the control group did not have any formal dance experience/regular practice of dancing. The participants hearing sensitivity was within normal limits (i.e., puretone average of 500 Hz, 1 kHz and 2 kHz was less than ≤15 dBHL). None of the participants had history or presence of any otological problems such as ear discharge, ear pain, itching, and tinnitus. Also the participants did not have any history/presence of neuromuscular problem, history of intake of drugs that may lead to vestibulotoxicity, and had no symptoms related to vestibular problems.

Instrumentation

To evaluate hearing sensitivity, a calibrated two channel clinical audiometer (Orbiter-922 V-2x, G N Otometrics, Taastrum, Denmark) with TDH-39 headphones (Telephonics, 815 Broad Hollow Road, Farmingdale, New York 11735) and a B-71 bone vibrator (Radioear, KIMMETRICS, 22050 Mohawk Drive, Smithsburg, MD 21783) were used to determine the air and bone conduction thresholds. Middle-ear function was analysed using a GSITympstar system (GSI VIASYS Healthcare, Wisconsin, USA). Vestibular evoked myogenic potentials were recorded using an Intelligent Hearing Systems (Smart EP) System (Intelligent Hearing System, Florida, USA) with an Insert ER-3A earphone (Etymotic Research, Inc., 61 Martin Lane, Elk Grove Village, IL 60007, USA).

Procedure

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

Pure tone thresholds were obtained by using modified Hughson – Westlake procedure (Carhart & Jerger, 1959), for octave frequencies from 250 Hz to 8 kHz for air conduction stimuli and from 250 Hz to 4 KHz for bone conduction stimuli. For Tympanometry 226 Hz probe tone was used, ipsilateral and contralateral reflexes at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were obtained for both the ears. UCL for all the participants was obtained by presenting speech stimuli at 100 dB, Participants were instructed to respond to the stimuli by saying whether it is comfortable or not. Rectified VEMPs were recorded for both the groups by 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% (50µV to 100 µV) 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 KΩ and less than 2 KΩ 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. The VEMP was recorded with the following protocol: 500 Hz tone burst stimulus was selected based on the earlier studies which show a better amplitude and response rate with 500Hz tone burst (Kumar, Sinha, Bharti & Barman, 2006; Kumar, Sinha, Bharti & Barman, 2011).

Response 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.

Table 1: Recording protocol for VEMP

Stimulus parameters		Acquisition parameters	
Transducer	Insert ear phones ER-3A	Amplification	5000
Type of stimulus	500 Hz tone burst	Analysis window	-10 to 70ms
Intensity	95 dBnHL	Filters	30 – 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

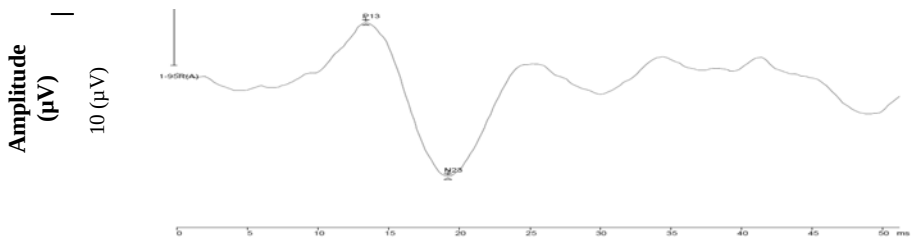

Figure 1: Wave form of VEMP response showing P13 and N23.

Results

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

Latency of P13 and N23 peak

VEMP could be recorded for all the subjects in both the control and the experimental groups. P13 and N23 peaks in the waveform were visualized and analyzed for the control (non dancers) group and the experimental (dancers) group. Figure-2 shows recorded waveforms from one of the participants each from the control (non dancers group) and the experimental (dancers) group. As it can be seen from figure-2 that the latency of P13 & N23 peaks are almost similar for control (non dancers) and the experimental group (salsa and kathak dancer group).

To understand the group differences between the experimental and the control group, the data of two subgroups of the experimental group (salsa & kathak dancer group) were combined. The mean and standard deviation (S.D) were then computed for the control group and the combined data of the experimental group. The mean values for the latency of P13 and N23 for both the control and the combined data of the experimental group are given in the Table 2.

As we can see from Table 2 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 2 that the standard deviation in the experimental group is lesser than the control group.

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 did not show any significant differences for the latency of P13, [t (68)=0.835, p>0.05] and N23, [t(68)=0.516, p>0.05] between dancers and the non dancers group.

The experimental group had two subgroups (salsa & 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. The data are given in Table 3.

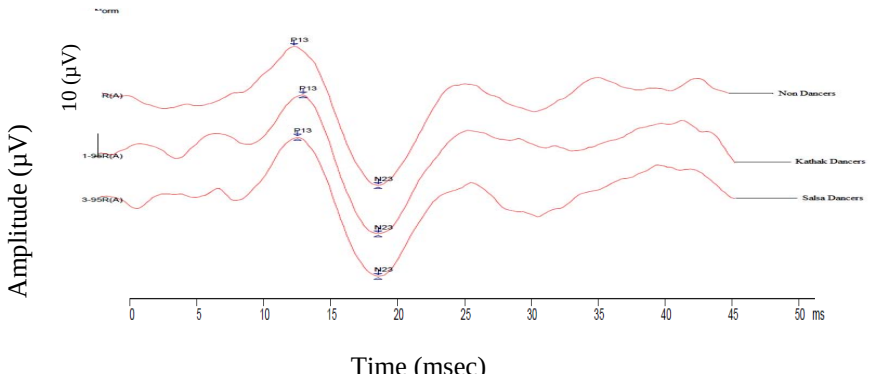

Figure 2: Waveform of VEMP response showing P13 & N23 latency in non dancers and dancers.

Table 2: Mean and standard deviation (S.D) values of latency (ms) of control group and the experimental group

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

Table 3: Mean and S.D of P13 and N23 latencies of VEMP responses in control group and experimental group (Kathak dancers and Salsa dancers)

Groups	P13 latency		N23 latency	
	Mean (ms)	S.D	Mean (ms)	S.D
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

Table 4: Mean and S.D of amplitude of P13, N3 and peak to peak amplitude of P13-N23 Complex of dancers and the non dancers group

Groups	P13 amplitude		N23 amplitude		P13-N23 amplitude	
	Mean (μV)	S.D	Mean (μV)	S.D	Mean (μV)	S.D
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

As it can be seen from the Table 3, mean latency for P13 and 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.

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 did not show significant main effect of group on latency of P13 [$F(2, 67)=0.39$, $p>0.05$] and latency of N23 [$F(2, 67)=2.34$, $p>0.05$].

Amplitude of P13, N23, and P13-N23 complex

Amplitude of P13, N23 peak 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 & 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

It can be seen from Table 4 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 control group.

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 5.

Table 5: Mean and S.D of P13 and N23 latencies of VEMP responses in non dancers and dancers (kathak dancers and salsa dancers)

		P13 Amplitude		N23 Amplitude		P13-N23 Amplitude	
Groups		Mean(µV)	S.D	Mean(µV)	S.D	Mean(µV)	S.D
Control	Non dancers	16.02	8.38	21.4	10.1	37.4	17.36
Exp	Kathak dancers	25.72	5.14	28.2	11.3	53.9	14.96
	Salsa dancers	21.31	6.46	26.6	6.53	47.9	10.71

Table 6: Duncan's pos- Hoc analysis for the amplitude of the VEMP parameters

	P13		N23		P13-N23	
	Kathak	Salsa	Kathak	Salsa	Kathak	Salsa
Control	S^*	S^*	S^*	S^*	S^*	S^*
Kathak		S^*		NS^*		S^*

S^*-significant at 0.05 level, NS^*-Not significant

It can be seen from Table-5 that mean amplitude value for amplitude of P13, N23 and peak to peak amplitude of P13-N23 complex are higher for the two subgroups of the experimental group (Kathak & 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.

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 main effect of group on the amplitude of P13 peak [$F(2, 67)=11.63$, $P<0.05$], 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 6 below:

Discussion

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.

The mean values for the latency of P13 and N23 peaks are in agreement with the previous studies in audiology department at AIISH (Vijayshankar & Basavaraj, 2008; Manasa & Barman, 2009). However, there are studies which have reported different latency values for both P13 and N23 peaks (Wang & Young, 2003; Wang &Young, 2006; Isaradisaikul, et al., 2008). Vijayshankar & Basavarj (2008); Manasa, & Barman (2009) 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 and 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, Chang, Tae, Dong, & Seung, 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; Rodith, Robert, Todd, & Daniel, 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.

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. The amplitude obtained for the control group in the present study is almost similar to amplitude values reported in earlier studies (Vijay Shankar & Basavaraj 2008; Manasa & Barman, 2009)

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, et al., 2001), vestibular neuritis (Murofushi, et al., 2001), vertigo (Iwasaki, Takai, Ito & Murofushi, 2005). Many behavioral studies reported that dance based training improves that balance in young and adult subjects within 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 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 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 form that is kathak dancer had larger amplitude values compared to the western 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.

Conclusions

In the present study, the results revealed significant difference findings in terms of amplitude of the VEMP responses between the dancers and the non-dancers. From the present study it can be concluded that the dance practice may improve the sacculocollic pathway. Amplitude of the VEMP parameters is a better tool to study the plasticity of sacculocollic pathway compared to the latency parameters.

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.

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,* 108-15.

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.

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,* 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 loss-affected and healthy individuals. *Audiology, 18,* 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 vestibular- evoked potentials in normal subjects: A comparison between responses obtained from sternomastoid and trapezius muscles. *Acta Otolaryngologica, 117,* 472–481.

Gittis, A., H., & Sascha, D. L. (2006).Intrinsic and synaptic plasticity in the vestibular system. *Neurobiology, 16,* 385-390.

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.

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,* 104.

Krampe, J., Rantz, M. J., Dowell, L., Schamp, R., Skubic, M., & Abbott, C. (2010). Dance-based therapy in a program of all-inclusive care for the elderly: an integrative approach to decrease fall risk. *Journal of Nursing Administration Quarterly, 34,* 156-61.

Kumar, K., Sinha, S, K., Singh, N. 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. (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., 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.

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,* 103-106.

Manasa, M. & Barman, A. (2009). *Vestibular Evoked Myogenic Potentials (VEMP) in individuals with noise induced hearing loss (NIHL).* Unpublished dissertation submitted to the University of Mysore, Mysore, India.

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.

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.

Streubel, S. O., Cremer, P. D., Carey, J. P., Weg, N., & Minor, L. B. (2001). Vestibular-evoked 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,* 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.

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. & Basavaraj V.(2008). *Effect of mode of sternocleidomastoid (SCM) excitation on Vestibular Evoked Myogenic Potentials (VEMP).* Unpublished Master's dissertation submitted to the University of Mysore, Mysore, India.

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.

Wang, S. J., & Young, Y. H. (2003). Vestibular evoked myogenic potentials using simultaneous binaural acoustic stimulation. *Hearing Research, 185,* 43–8.

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.