CHAPTER 1

Introduction

Sound is a form of vibrational energy that travels through a medium, usually elastic solids, liquids or gases, as an audible mechanical wave of pressure and displacement. When it impinges upon an animal’s or human’s ear, it creates a sensation of hearing, provided the auditory system is functioning efficiently. When loud enough, sound induces a reflexive contraction of the stapedial muscle within the middle ear which can be measured as an acoustic reflex (Silman & Silverman, 1991). These intense auditory signals not only stimulate the cochlea and its afferents (Borg, 1973; Lyons, 1978) but were also found to activate the otolith organs and their afferents (Colebatch & Halmagyi, 1992; Colebatch, Halmagyi, & Skuse, 1994; Ochi, Ohashi, & Nishino, 2001; Manzari, Tedesco, Burgess, & Curthoys, 2010). The activations of the otolith organs were shown to cause activations or inhibitions of the electromyographic (EMG) activity (Bickford, Jacobson, & Cody, 1964; Colebatch & Rothwell, 2004). Therefore they can be recorded as the modulations in the EMG waveform. The Vestibular evoked myogenic potential (VEMP) is one such short latency muscle potential.

VEMP is a biphasic potential that was reported to represent the response of the otolith organs to loud acoustic (Colebatch & Halmagyi, 1992; Colebatch et al., 1994), vibratory (Halmagyi, Yavor, & Colebatch, 1995; Sheykholeslami, Murofushi, Kermany, & Kaga, 2000; Rosengren, Todd, & Colebatch, 2005; Curthoys, Kim, McPhedran, & Camp, 2006; Todd, Rosengren, & Colebatch, 2009a) or galvanic (Watson & Colebatch, 1998; Monobe & Murofushi, 2004; Iwasaki et al., 2011) stimulation. It was recorded from several muscles of the body like triceps muscles (Cherchi et al., 2009), soleus muscle (Cunha, Labanca, Tavares, & Goncalves, 2014), gastrocnemius muscle (Ruddissil & Hain,
2008), masseter muscles (Deriu et al., 2007), extensor muscles of the neck (Wu, Young, & Murofushi, 1999; Sakakura, Takahashi, Takayasu, Chikamatsu, & Furuya, 2005), sternocleidomastoid muscle (Colebatch & Halmagyi, 1992; Colebatch et al., 1994) and inferior oblique muscle (Todd, Rosengren, & Colebatch, 2003; Rosengren et al., 2005; Weber, Rosengren, Michels, Sturm, Straumann, & Landau, 2012). When recorded from the sternocleidomastoid (SCM) muscle, the VEMP is popularly known as Cervical VEMP (cVEMP).

VEMP, like any other muscle in the body, can also be recorded from the inferior extra-ocular muscles below the eyes (Todd et al., 2003; Rosengren et al., 2005; Weber et al., 2012). Todd et al (2003) recorded a short latency biphasic potential, with the negative peak at 10 ms (subsequently called n1 or n10) and the positive peak at 15 ms (later termed p1 or p15), from the inferior extra-ocular muscles. They found that the threshold of this biphasic potential was comparable to that of the cVEMP and it was not deterred by the subject’s hearing acuity; nonetheless it was absent or drastically reduced in amplitude in individuals with vestibulopathies. These findings led Todd et al (2003) to conclude that this potential was of a vestibular origin. Further research in this area confirmed the vestibular origin of oVEMP and proposed its origin from the otolith-ocular pathway (Rosengren et al., 2005; Chihara, Iwasaki, Ushio, & Murofushi, 2007; Todd, Rosengren, Aw, & Colebatch, 2007; Iwasaki, et al., 2008; Govender, Rosengren, & Colebatch, 2009; Wang, Jaw, & Young, 2009). Since the origin of the responses was from the vestibular system and it was recorded from the eye muscles, this potential was subsequently termed ‘ocular vestibular evoked myogenic potential’ (oVEMP) (Rosengren et al., 2005).

The opinions in literature reports were initially divided regarding the generator end-organ for oVEMP being saccule or utricle (Rosengren et al., 2005; Todd et al., 2007;
Manzari et al., 2010). However more recent research reports are supportive of the utricle as the end organ of its origin (Curthoys, Vulovic, & Manzari, 2012). Once generated in the utricle, the impulses were reported to travel along the superior vestibular nerve, vestibular nuclei, medial longitudinal fasciculus, where they cross-over to contralateral oculomotor nuclei and travel along the ocular nerves to end-up in the inferior oblique muscle (Rosengren et al., 2005; Rosengren, Welgampola, & Colebatch, 2010; Weber et al., 2012).


Meniere’s disease (MD) is one of the pathologies that have been shown to cause a utricular pathology in addition to the better known cochlear and saccular involvement (Merchant, Adams, & Nadol, 2005). It was first described by Prosper Meniere (1861) as a syndrome consisting of continuous or intermittent head noises (tinnitus) accompanied by reduction of hearing and intermittent attacks of vertigo accompanied by nausea, vomiting, and syncope. In 1938, Yamakawa in Japan and Hallpike and Cairns in England independently and almost simultaneously reported the finding of endolymphatic hydrops in temporal bones from patients with Meniere’s syndrome. Since then, endolymphatic
hydrops has been the most widely accepted pathophysiological correlate of Meniere’s disease (Lindsay, 1942; Altmann & Fowler, 1943; Cawthorne, 1947; Day & Lindsay, 1949; Nager, 1949; Klockhoff & Lindblom, 1961; Paparella, 1991; Monsell, Balkany, Gates, Goldenberg, Meyerhoff, & House, 1995; Shulman & Goldstein, 2006). The Committee on Hearing and Equilibrium Guidelines for the Diagnosis and Evaluation of Therapy in Meniere’s disease in 1995 classified Meniere’s disease into ‘certain’, ‘definite’, ‘probable’ and ‘possible’ Meniere’s disease. This classification of Meniere’s disease is based only on the clinical symptoms, audiometry results and histopathological finding of endolymphatic hydrops. Although this classification has been almost elevated to the gold standard status owing to a lack of more efficient and objective techniques available for identifying Meniere’s disease, there is paucity of objectivity to the use of this classification. Therefore further research studies continue to sprout in search of an objective technique for its identification and diagnosis.

Benign paroxysmal positional vertigo (BPPV) is another pathology involving the utricle (Nakahara et al., 2013; Seo et al., 2013; Singh & Barman, 2015). It is mainly characterized by brief episodes of vertigo (true spinning sensation) which is often caused by head motion in the vertical or horizontal planes (McClure, 1985; Epley, 1992). These symptoms are best explained by the ‘Canalithiasis’ theory (Hall, Ruby, & McClure, 1979) which postulates that the positional vertigo and the following nystagmus is precipitated by the free-floating otolithic debris which moves within the endolymphatic fluid of the semicircular canals in response to alterations in the head position. Although the pathology could be associated with any of the three semicircular canals, the posterior/inferior semicircular canals were reported to be affected in up to 96% of cases of BPPV (Honrubia, Baloh, Harris, & Jacobson, 1999). The orientation of this canal makes it more prone to the entry of the otoconia debris (Schratzenstaller, Wagner-Manslau, Strasser, &
Arnold, 2005). The existence of the lateral and anterior canal variants were also reported, although in a much smaller proportion (Schratzenstaller et al., 2005; White, Coale, Catalano, & Oas, 2005; Escher, Ruffieux, & Maire, 2007).

Auditory neuropathy spectrum disorder (ANSD) was described as a disorder of hearing that affects the auditory nerve function, inner hair cell function, synaptic function or any combination of these, in the presence of preserved cochlear outer hair cell activity (Starr, Picton, Sininger, Hood, & Berlin, 1996; Harrison, 1998; Berlin, Morlet, & Hood, 2003; Amatuzzi, Liberman, & Northrop, 2011; Nachman, 2012). The hearing impairment in ANSD has been distinguished from other hearing losses by the hallmark of preserved otoacoustic emissions (OAEs) and/or cochlear microphonics (CM) and absent or abnormal auditory brainstem response (ABR). In 1996, Starr et al first described and coined the term ‘auditory neuropathy’ (AN) to represent this symptom complex. To date, the exact sites of lesions in ANSD are still not well understood, but investigators have found that this disorder can be caused by auditory nerve dysfunction or ‘neuropathy’ and synaptic dysfunction at the junction of the inner hair cell/auditory nerve, which can equally produce the same set of auditory test results in the affected individuals (Yasunaga et al., 1999; Rodriguez-Ballesteros et al., 2003). Therefore, a panel of experts in the year 2008 proposed the term ‘auditory neuropathy spectrum disorder’ (ANSD) as part of the Guidelines Development Conference on the Identification and Management of Infants with Auditory Neuropathy (2008) in order to accommodate these variations more efficiently.

1.1 Need for the study

As stated previously, a utricular and/or utriculo-ocular pathway involvement in various vestibular pathologies has been shown by several previous investigations mainly through the histopathological examinations conducted by post-mortem studies (O’Connor
et al., 1985; Okuno & Sando, 1987; Parnes & McClure 1992; Buckingham, 1999; von Brevern et al., 2006; McCall, Ishiyama, Lopez, Bhuta, Vetter, & Ishiyama, 2009). Although these studies have provided a great insight into the pathophysiology and knowledge about utricular and/or utriculo-ocular pathway involvement in several vestibular pathologies, these methods cannot be used for a day-to-day diagnosis of these pathologies. This resulted in continued exploration for tests that could be used to diagnose the conditions in living human beings so that the affected individuals could be extended treatment for the pathology.

1.1.1 Need for oVEMP in vestibular pathologies.

In the last couple of decades or so, a few tests were developed that lend themselves to the assessment of utricular function. These include sinusoidal off-vertical axis rotation method using the rotational chair test (Hess & Diefinger, 1990; Koizuka, Yamakawa, Naramura, & Kubo, 1995; Angelaki & Hess, 1996; Sugita-Kitajima, Azuma, Hattori, & Koizuka, 2007; Sugita-Kitajima, & Koizuka, 2014), subjective visual vertical test (Garcia & Jauregui-Renaud, 2003; Kumagami et al., 2009; Faralli et al., 2011) and head-tilting stabilometry (Inukai, Koizuka, & Takahashi, 2008; Faralli, Ricci, Ibba, Crognoletti, Longari, Frenguelli, 2009). The above studies on these tests have shown encouraging results by reporting positive findings in individuals with unilateral Meniere’s disease and unilateral benign paroxysmal positional vertigo (BPPV). Nonetheless, they have obvious drawbacks in terms of cost-effectiveness, patient comfort during testing and specificity of results across pathologies (Sugita-Kitajima et al., 2007; Bonan, Guettard, Leman, Colle, & Yelnik, 2006).

Sugita-Kitajima et al (2007) observed deviant results on the sinusoidal off-vertical axis rotation testing in several unilateral vestibular pathologies like Meniere’s disease,
neuritis/labyrinthitis, Ramsay-Hunt syndrome and delayed endolymphatic hydrops, which indicates that the results are not specific to a particular pathology rather it is indicative of the existence of utricular involvement in these pathologies. Similarly, Yelnik et al (2002) and Bonan et al (2006) reported affected results of subjective visual vertical (SVV) in patients with cerebral hemispheric lesion such as stroke, which suggests that the results of SVV are not only impacted by the presence of utricular pathology but also by more central lesions that completely spare the utricle and the utriculo-ocular pathway. Further, these tests are administered using the rotatory chair test which makes them not only uncomfortable but also more expensive. Further, the responses on these tests are not ear specific and are many times insensitive to the common vestibular lesions which are unilateral in origin (Wuyts et al., 2001; Wuyts, Hoppenbrouwers, Pauwels, & Van de Heyning, 2003).

Ocular VEMP is a relatively a new test that has been reported to assess the functioning of utricle and utricle mediated vestibulo-ocular reflex (Rosengren et al., 2005; Todd et al., 2007; Manzari et al., 2010; Curthoys et al., 2012; Weber et al., 2012). It was found to be useful in the diagnosis of several vestibular pathologies like labyrinthitis / vestibular neuritis (Moon et al. 2012a, b; Zuniga et al., 2012), superior semicircular canal dehiscence (Taylor, Bradshaw, Halmagyi, & Welgampola, 2012), Meniere’s disease (Murofushi, Nakahara, Yoshimura, & Tseda, 2011; Taylor et al., 2011) and BPPV (Nakahara et al., 2013; Seo et al., 2013; Singh & Barman, 2015) by virtue of the finding of absent or reduced response amplitudes, high inter-aural asymmetry ratio, elevated or improved thresholds and/or prolonged latencies. Additionally, the use of oVEMP ensures better comfort than the rotatory chair test and is also less expensive as it can be administered using most of the commercially available auditory evoked potential systems. These factors probably lead to these tests being relatively less popular than oVEMP, even
though oVEMP was discovered long after the utility of these above tests was already proved.

1.1.2 Need for frequency tuning of oVEMP in vestibular pathologies.

The utility of oVEMP in the diagnosis of several vestibulopathies has been shown in literature (Murofushi et al. 2011; Taylor et al., 2011, 2012; Moon et al. 2012a, b; Zuniga et al., 2012; Nakahara et al., 2013; Seo et al., 2013; Singh & Barman, 2015). These studies have mainly used amplitude, inter-aural amplitude ratio and/or response rate to arrive at the conclusion of presence of vestibulopathies. Nonetheless, most of these findings of oVEMP were not specific to any particular pathology, rather they were commonly found in most of these conditions. This continuously spurred the research community to persevere with searching for a parameter of oVEMP that had possibility of being specific to one of the above mentioned pathologies.

The search for better and more specific measures probably lead to the discovery of frequency tuning property of oVEMP. The initial studies in this regard demonstrated a shift in frequency tuning peak to 1000 Hz or beyond in the affected ears of individuals with Meniere’s disease from 500 Hz in healthy individuals (Sandhu, Low, Rea, & Saunders, 2012; Winters, Berg, Grolman, & Klis, 2012; Jerin, Berman, Krause, Ertl-Wagner, & Gurov, 2014). However, these studies only compared the results in Meniere’s disease to healthy adults but not evaluated this aspect in other vestibular pathologies. Therefore, it is not known if a shift in frequency tuning is an exclusive finding in Meniere’s disease or it could possibly be common to some of the other vestibular pathologies also. Thus, there was a need to explore this aspect in other vestibular pathologies in order to investigate whether these pathologies demonstrate similar or
dissimilar frequency tuning to that of healthy individuals and also if there is a difference in frequency tuning of oVEMP between the vestibular pathologies.

1.1.2.1 Need for the frequency tuning of oVEMP in healthy individuals.

The frequency tuning property of cVEMP in healthy individuals has been richly researched upon; however there are only a few published studies in literature that report about the frequency tuning of air-conduction tone-bursts evoked oVEMP in healthy individuals. In one of the first studies, Park and associates obtained oVEMP across the frequencies from 20 normal subjects to assess the frequency tuning properties of oVEMP in them (Park, Lee, Shin, Lee, & Park, 2010). They reported oVEMP responses to air-conducted tone-bursts to be tuned to 500 Hz frequency region, similar to that of cVEMP. Similar findings were subsequently also reported by some of the other researchers (Sandhu et al., 2012; Winters et al., 2012; Singh & Barman, 2013, 2014). However, some of the other contemporary studies tended to disagree with this by demonstrating frequency tuning at other frequencies (Todd, Rosengren, & Colebatch, 2009a; Lewis, Mustain, Xu, Eby, & Zhou, 2010; Taylor et al., 2012).

Todd et al (2009a) reported the oVEMP frequency tuning in the frequency range of 400-800 Hz; however they did not record the oVEMP at 500 Hz specifically. In contrast, Taylor et al (2012) compared the frequency tuning properties of cVEMP to oVEMP on 14 healthy individuals and reported that the two differed in terms of the peak of frequency tuning. In their study, cVEMP were reported to be closely tuned to 500 Hz frequency region where as oVEMP were tuned to 1000 Hz region in more than 50% of the healthy individuals.

Although the above mentioned studies were some of the first attempts at exploring the frequency tuning of oVEMP in healthy individuals, most of them obtained oVEMP
from less than 15 healthy individuals which is a relatively inadequate sample size. Additionally some of these studies also included only a few frequencies rather than the entire frequency set from 250 Hz to 4000 Hz (at octave and mid-octave frequencies) to study the frequency tuning properties. Although the differences in findings among the studies might be accounted by these factors, the fact remains that there is no consensus about the frequency tuning property of oVEMP in healthy individuals. The lack of consensus among the researchers regarding the frequency tuning of oVEMP in healthy individuals therefore highlights the need for resolving these inconsistencies through further studies by using larger sample size.

1.1.2.2 Need for frequency tuning of oVEMP in individuals with Meniere’s disease.

The individuals with endolymphatic hydrops, especially in the case with Meniere’s disease, were reported to have cochleo-otolithic hydrops (Rauch, Merchant, & Thedinger, 1989). Since oVEMPs also arise from the utricular afferents, it is only logical to presume that an altered motion mechanism of a distended utricle may cause a corresponding alteration of oVEMP in such clients.

The studies in the contemporary literature have reported a shift in the frequency tuning peak of oVEMP from 500 Hz in healthy individuals to 1000 Hz or beyond in individuals with Meniere’s disease (Sandhu et al., 2012; Winters et al., 2012). These findings appear promising but are not without confounds. Sandhu et al (2012) used a very small number of subjects (N = 12) whereas Winters et al (2012) did not use the mid-octave frequencies and frequencies above 1000 Hz to arrive at the conclusion. There could also be a possibility that these findings were due to variations reported in healthy individuals itself as the previous studies have shown frequency tuning at 500 Hz (Sandhu et al., 2012;
Winters et al., 2012), in the frequency range from 400-800 Hz (Todd et al., 2009a) or 1000 Hz in more than 50% of the healthy individuals (Lewis et al., 2010; Taylor et al., 2012) in the healthy individuals.

An additional confounding variable may have been age which was not controlled when matching the clinical and the control groups in most of the above studies. An unswerving observation in older adults is the considerable variation in the amount of change in the vestibular system that occurs with age (Schuknecht, Makoto, & Gacek, 1965; Johnsson, 1971; Rosenhall, 1973; Walther & Westhofen, 2007). It is not known if these well-documented neuroanatomic age-related changes occurring in the otolith organs result in altered frequency tuning, similar to that reported in patients with Meniere’s disease. However the possibility that frequency tuning could be altered as a result of the process alone was supported the findings of a recent study that reported changes in the location of the peak of the frequency tuning in middle-aged adults (age range = 40-59 years) and older adults (60 years and older) compared to the young adults (20-39 years) (Piker, Jacobson, Burkard, McCaslin, & Hood, 2013). They demonstrated shift in frequency tuning from 500 Hz or 750 Hz in most of the younger adults to 750 Hz or 1000 Hz in a large majority of older adults, with frequency tuning peak being shared between 500 Hz, 750 Hz and 1000 Hz by almost equal proportions of individuals in the middle aged group. Therefore age appears to affect the frequency tuning of oVEMP in healthy individuals. Thus, there is a need to control the effects of age from adulterating the results before concluding with any degree of conviction regarding the shift in frequency tuning in Meniere’s disease.
1.1.2.3 Need for frequency tuning of oVEMP in individuals with BPPV.

BPPV is reported to be a disease with one of the highest prevalence among the otological disorders. A population-based study performed at the Mayo Clinic in Minnesota indicated a 0.06% incidence of BPPV, with a 38% increase in incidence with each decade of life (Froehling, Silverstein, Mohr, Beatty, Oxford, & Ballard, 1991). In another study, von Brevern et al (2007) reported the prevalence of BPPV to be 11-64 per 100,000 persons and a life time prevalence of 2.4%, which confirmed a high prevalence of BPPV.

Proper diagnosis and determination of the site of pathology is imperative for selection of intervention techniques. There are various manoeuvres that can differentiate between the involvements of one semicircular canal against the others, however an objective test has been lacking. Though the studies have used cVEMPs for the evaluation of BPPV to identify posterior canal and saccular involvement and equivocal results exist (Hong, Park, Yeo, & Cha, 2008; Yang, Kim, Lee, & Lee, 2008; Longo, Onofri, Pellicciari, & Quaranta, 2012), there is dearth of studies regarding the usefulness of oVEMP in the diagnosis of BPPV. However, an understanding of the pathophysiology of the disease would be useful in explaining the need for investigating the utility of oVEMP in identification of utricular pathology in BPPV. The histopathological studies on BPPV have revealed the source of otoconia debris in the semicircular canals to be mainly the utricular macula (Parnes & McClure, 1992; Buckingham, 1999; von Brevern et al., 2006). Therefore a utricular pathology would seem evident in most of the cases of BPPV. The oVEMP being a utricular response, it could be useful in identification of utricular involvement in BPPV. This was indeed confirmed by the results of some of the recent studies which demonstrated utricular involvement in BPPV (Nakahara et al., 2013; Seo et al., 2013; Singh & Barman, 2015). While these studies reported reduced oVEMP
amplitudes in the ears with BPPV, Seo et al (2013) and Singh and Barman (2014) also reported the finding of augmented oVEMP amplitude in the affected ears of individuals with BPPV. Therefore, obtaining oVEMP only at one frequency (usually 500 Hz) and using absolute amplitude and inter-aural asymmetry ratio could possibly confuse the identification of the ear with the pathology.

The explanation for peak of the frequency tuning at a particular frequency in healthy individuals and its shift to another frequency in Meniere’s disease is primarily based on the concept of mass-spring model for resonance (Todd et al., 2000, 2009a; Sandhu et al., 2012; Winters et al., 2012; Singh & Barman, 2013, 2014). The mass is supplied by the otolithic macula, in this case utricular macula, which consists of the CACO3 crystals (otoconia). The pathology in BPPV is caused by the free-floating otoconia particles in the semicircular canal(s), which are believed to be dislodged from the utricular macula mainly (Parnes & McClure 1992; Buckingham, 1999; von Brevern et al., 2006). Since some particles move out of the utricular macula, the mass of utricular macula is likely to reduce and this would in turn impact on the resonance frequency. The change in resonance can be tapped using a possible shift in frequency tuning as tuning to a particular frequency is thought to arise out of the resonance property of the utricle (Todd et al., 2000, 2009; Singh & Barman, 2013, 2014). Hence, the logic appears to suggest that frequency tuning in BPPV might be different from that in healthy individuals. This however has not been explored previously and therefore there is a need to investigate the frequency tuning of oVEMP in individuals with BPPV.

1.1.2.4 Need for frequency tuning of oVEMP in individuals with ANSD.

The term ANSD is used to describe an unusual presentation of responses that is characterized by normal non-neural peripheral audio-vestibular functioning in presence of
abnormality in the neural auditory and/or vestibular systems. The prevalence of ANSD has been reported to vary across the globe and in different age groups. In adults, the prevalence in the Western countries was reported to range between 1.83% and 11% (Rance et al., 1999; Tang, McPherson, Yuen, Wong, & Lee, 2004). In India, Kumar and Jayaram (2006) reported an ANSD prevalence of 1 in 183 individuals with sensorineural hearing loss. Therefore, these studies confirm a high prevalence of ANSD in the West as well as in India.

Although the classical profile of a person with ANSD involves a deficiently functioning auditory nervous system in presence of normal outer hair cell activity, vestibular deficits in a large chunk of these individuals was also reported previously (Fuzikawa & Starr, 2000; Sheykholeslami et al., 2000; Wu et al., 2004; Sheykholeslami Schmerber, Kermany, & Kaga, 2005; Kumar, Sinha, Bharati, Singh, & Barman, 2007; Akdogan, Selcuk, Ozcan, & Dere, 2008; Sazgar, Yazdani, Rezazadeh, & Yazdi, 2010; Sinha, Shankar et al., 2013; Ismail, Makky, Besher, & Galhom, 2014). While most of these studies have reported regarding abnormal inferior vestibular nerve functioning in ANSD by virtue of observing absent or abnormal cervical VEMP in individuals with ANSD (Kumar et al., 2007; Sazgar et al., 2010), a few have reported abnormal superior vestibular nerve function by observing abnormal results on bithermal caloric irrigation when administering electronystagmography (Fuzikawa & Starr, 2000; Sheykholeslami et al., 2005) or videonystagmography (Ismail et al., 2014). The solitary publication on findings of oVEMP in ANSD reported absence of these potentials in 100% of the 11 subjects with ANSD in their study (Sinha et al., 2013). However, Sinha et al (2013) used a small sample size, especially for a heterogenous disorder like ANSD, and therefore the generality of their results could at best be erroneous. Nonetheless, the absence of oVEMP in individuals with ANSD appears to indicate towards a deficient utriculo-ocular pathway functioning,
which however is also common to other pathologies affecting the utricle and the utriculo-ocular pathway (Murofushi et al., 2011; Taylor et al., 2011, 2012; Moon et al., 2012a, b; Zuniga et al., 2012; Nakahara et al., 2013; Seo et al., 2013; Singh & Barman, 2015).

The postulates regarding frequency tuning of cVEMP as well as oVEMP tend to suggest that the mechanics within the peripheral vestibular system are responsible for the frequency tuning of these otolithic responses (Todd et al., 2000, 2009a). Nonetheless, the analysis of tuning within the auditory system has shown that the tuning is not only affected by the presence of pathology within the cochlea (labyrinth) but also affected by neural pathologies, especially ANSD. Vinay and Moore (2007) reported wider than normal tuning curves in the individuals with ANSD. A similar effect of ANSD might be present in the tuning curves of oVEMP; however it has never been explored. Therefore there is a need to investigate the frequency tuning of oVEMP in individuals with ANSD.

1.2 Aim

The present study aimed at characterizing the oVEMP response in healthy individuals and those with Meniere’s disease, BPPV and ANSD. Further, it aimed at examining the existence of a difference in the frequency tuning of oVEMP, if any, between the participants with normal audio-vestibular system and those having above mentioned pathologies.

1.3 Objectives

To fulfil the above aims, the study had the following objectives:

A. To examine the ear differences, if any, on the following parameters of oVEMP in the healthy individuals:
   a. Response rate
   b. Peak-to-peak amplitude
c. Threshold

B. To investigate the effect of frequency of tone-bursts on the following parameters of oVEMP in healthy individuals:
   a. Response rate
   b. Peak-to-peak amplitude
   c. Threshold

C. To study the frequency tuning of oVEMP in healthy individuals.

D. To examine the ear differences, if any, on the following parameters of oVEMP in individuals with Meniere’s disease, BPPV and ANSD and their respective age- and gender-matched comparison groups:
   a. Response rate
   b. Peak-to-peak amplitude
   c. Threshold
   d. Frequency tuning

E. To examine the effect of frequency of tone-bursts on the following parameters of oVEMP in individuals with Meniere’s disease, BPPV and ANSD and their respective age- and gender-matched comparison groups:
   a. Response rate
   b. Peak-to-peak amplitude
   c. Threshold

F. To compare each of the clinical groups (Meniere’s disease, BPPV, & ANSD) against the respective age- and gender-matched comparison groups on the following parameters of oVEMP:
   a. Response rate
   b. Peak-to-peak amplitude
1.4 Hypotheses

The study began with the null hypothesis stating that:

A. There is no significant difference between the ears on the following parameters of oVEMP in the healthy individuals and the individuals with Meniere’s disease, BPPV and ANSD:
   a. Response rate
   b. Peak-to-peak amplitude
   c. Threshold

B. There is no significant effect of frequency of tone-bursts on the following parameters of oVEMP in healthy individuals:
   a. Response rate
   b. Peak-to-peak amplitude
   c. Threshold

C. There is no frequency tuning of oVEMP in healthy individuals.

D. There is no significant difference between the ears on the following parameters of oVEMP in individuals with Meniere’s disease, BPPV and ANSD and their respective age- and gender-matched comparison groups:
   a. Response rate
b. Peak-to-peak amplitude

c. Threshold

d. Frequency tuning

E. There is no significant effect of frequency of tone-bursts on the following parameters of oVEMP in individuals with Meniere’s disease, BPPV and ANSD and their respective age- and gender-matched comparison groups:

a. Response rate

b. Peak-to-peak amplitude

c. Threshold

F. There is no significant difference between the clinical groups (Meniere’s disease, BPPV, & ANSD) and their respective age- and gender-matched comparison groups on the following parameters of oVEMP:

a. Response rate

b. Peak-to-peak amplitude

c. Threshold

G. There is no significant difference in frequency tuning of oVEMP between the clinical groups (Meniere’s disease, BPPV, & ANSD) and their respective age- and gender-matched comparison groups.

H. There is no significant difference in frequency tuning of oVEMP among the clinical groups.

I. There is no significant difference in frequency tuning of oVEMP among the comparison groups used for the three clinical groups.