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A NORMATIVE STUDY ON THE CLICK VESTIBULAR EVOKED MYOGENIC POTENTIAL

by

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THESIS APPROVAL PAGE

This is to certify that the thesis prepared by _____**Sasha T. Phillips** _____

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ABSTRACT

A NORMATIVE STUDY ON THE VESTIBULAR EVOKED MYOGENIC POTENTIAL

Sasha T. Phillips

Vestibular Evoked Myogenic Potentials (VEMPs) were recorded at various intensities to click stimuli in 16 subjects with a negative self-reported history of vestibular pathology. At most, recordings were obtained to a total of six stimulus intensities (i.e., 90, 95, 70, 75, 80 and 85 dBnHL). From the VEMP waveforms collected at 90 and 95 dBnHL several response indices (i.e., absolute latency values for p13 and n23, peak-to-peak amplitude values, and amplitude asymmetry ratios) were assessed.

Results from this study revealed no significant difference for latency, peak-to-peak amplitude, amplitude asymmetry ratio, and threshold between the left and right ears for the 90 and 95 dBnHL responses. Additionally, the tonic EMG RMS values ranged from 300 to 1250μV. All subjects were able to maintain and achieve tonic contractions within $\pm$10% of their established EMG tonic level for all stimulus intensities for each trial of the VEMP recordings. These findings provide a normative guideline for optimal stimulus and recording conditions to be used in the Speech, Language, and Hearing Center at Towson University.

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CHAPTER 1

INTRODUCTION

The Vestibular Evoked Myogenic Potential (VEMP) is a short latency myogenic potential that is evoked by high intensity acoutic stimulation. This myogenic response has been used clinically as a diagnostic tool in the assesment of saccular function within the otolith organs of the vestibular system. Currently, the primary method of assesing the integrity of the peripheral and central vestibular system is through an Electronstagmography (ENG)/Videonystagmography (VNG) examination (Wuyts, Furman, Vanspauwen, and Heyninig, 2007). One major limitation of the caloric testing portion of the ENG/VNG test battery is it only assesses the integrity of the horizontal semicircular canals (SCCs), which is only one of the five sensory structures for balance within the peripheral vestibualar system. The remaining four sensory structures of the peripheral vestibular system (i.e., the anterior SCC, the posterior SCC, the utricle, and the saccule) are not directly assesed in clinical practice. The addition of recording the VEMP response in everyday audiologic practice allows for assesment of the integrity of the saccule, as part of the vestibular test battery.

Bickford, Jabcobson, and Cody (1964) were among the first researchers to record this short latency response. It was first believed that this myogenic potential was optimally recorded with the active electrode located on the inion. However, more recently, Colebatch and Halmgyi (1992) have demonstrated that a robust VEMP response can be optimally recorded when the active electrode is placed on the sternocleidomastoid muscle (SCM). They hypothesized that there is an efferent pathway

between the saccule and the ipsilateral SCM and when the ipsilateral SCM is contracted, a myogenic response can be recorded in individuals with normal saccular functioning.

In patients with peripheral vestibular pathologies the VEMP response is either abnormal or absent. The typical abnormalites of the VEMP measurement characteristics are delayed absolute latencies for waves p13 and n23 components, reduced peak-to-peak amplitude values for the p13-n23 response, abnormal asymmetry amplitude ratios, and decreased threshold values.

CHAPTER 2

LITERATURE REVIEW

Anatomy and Physiology of the Vestibular System

There are three primary sensory systems that contribute to an individual's sense of equilibrium and balance. These are the vestibular system, the visual system, and the somatosensory system. The vestibular system, the primary contributor, is responsible for two thirds of the information related to an individual's sense of balance and equilibrium. The somatosensory system and the visual system, collectively, contribute approximately one third of the sensory information necessary for normal equilibrium and balance. The role of the central nervous system (CNS) is to synthesize information from all three of these systems and then initiate an appropriate motor response. The earlier a structure develops in an embryo, the more necessary/basic its role is in the development of the fetus. Therefore, the importance of the sensory input from the vestibular system to a human's sense of equilibrium is evident by the fact that the vestibular system is one of the first structures to develop during the embryologic period. Development of the vestibular system begins at 4 weeks gestation (Lysakowski, 2010; Bear et al. 2001).

Most anatomists and physiologists divide the vestibular system into the peripheral vestibular system and the central vestibular system. In order to understand how the VEMP is properly recorded and interpreted, it is necessary for the reader to have an understanding of the anatomy and physiology of these two sections of the vestibular system. The next section of this literature review will discuss the anatomy of the peripheral vestibular system. Following that will be a discussion of the anatomy of the central vestibular system.

Anatomy of the Peripheral Vestibular System

The peripheral vestibular system consists of two labyrinths, the bony labyrinth and the membranous labyrinth. The bony labyrinth forms the outer protective shell for the various sensory organs of balance and the membranous labyrinth is located inside the bony labyrinth and it contains the five sensory organs of balance. The bony labyrinth is filled with perilymph fluid, which contains a high concentration of Na+ ions and a low concentration of K+ ions. In contrast, the membranous labyrinth is filled with endolymph fluid which has the exact opposite ionic concentration (i.e., low concentration of Na+ ions and high concentration of K+ ions).

The five sensory organs of balance found in each ear are the three semicircular canals (SCC) and the two otolith organs. The three SCCs are: the anterior or superior SCC; the posterior or inferior SSC; and the horizontal or lateral SCC. The two otolith organs are the saccule and the utricle.

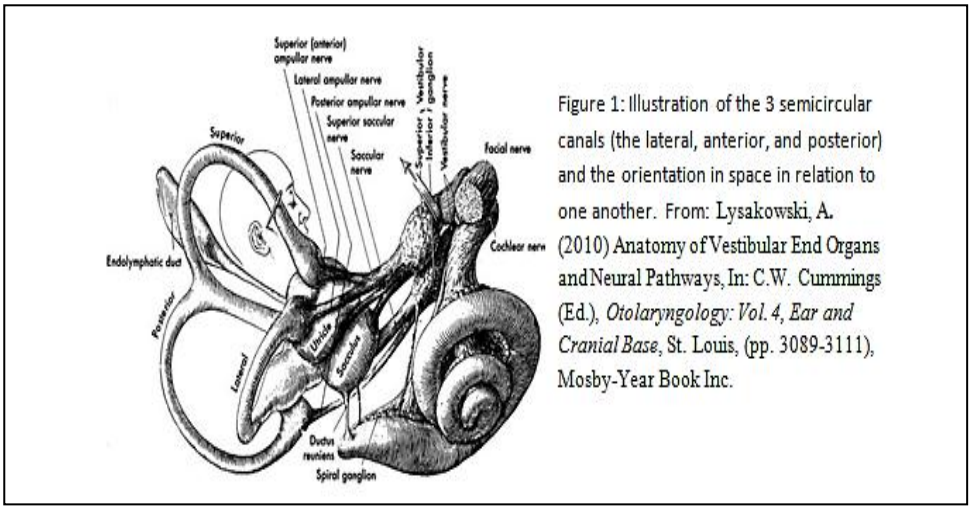

Figure 1: Illustration of the 3 semicircular canals (the lateral, anterior, and posterior) and the orientation in space in relation to one another. From: Lysakowski, A. (2010) Anatomy of Vestibular End Organs and Neural Pathways, In: C.W. Cummings (Ed.), *Otolaryngology: Vol. 4, Ear and Cranial Base*, St. Louis, (pp. 3089-3111), Mosby-Year Book Inc.

The three SCCs are all oriented differently in space. In relation to one another, they lie in a plane of approximately 90 degrees, referred by most as an orthogonal plane. Figure 1 shows the orientation of these 3 SCCs. The anterior and posterior SCCs are oriented 45 degrees from the midsagittal plane, while the horizontal canal or lateral canal lies in the horizontal plane and are tilted 30 degrees upward at its anterior end (Lysakowski, 2010; Bears et. al. 2001).

Each SCC has a widened area or a bulb like structure situated at the bottom of the canal, which is referred to as the ampulla. Within the ampulla are hair cells called cristae ampullaries. Each of these hair cells contains tiny hairs at the top of the cell, which are known as stereocilia. These stereocilia have an orderly organization based on their height, going from shortest to longest. The longest stereocilia is referred to as kinocilium. A gelatinous structure known as the cupula sits on top of the cristae ampullaries and the stereocilia are embedded in this structure. Although similar in their organization, the orientations of the kinocilium within the three SCCs are different. For the anterior and posterior SCCs, the kinocilium are located far away from the vestibule, whereas for the lateral SCC the kinocilium is located close to the vestibule (Lysakowski, 2010). Each of the three SCCs is paired with another SCC in the individual's opposite ear. Pairing is determined based on the canals which lie within the same horizontal or vertical plane. These results form three distinct pairs: the horizontal or lateral SCCs form the first pair, the right posterior and left anterior SCCs form the second pair, and the left posterior and right anterior SCCs forming the third pair. This coplanar pairing of the SCCs is depicted in Figure 2.

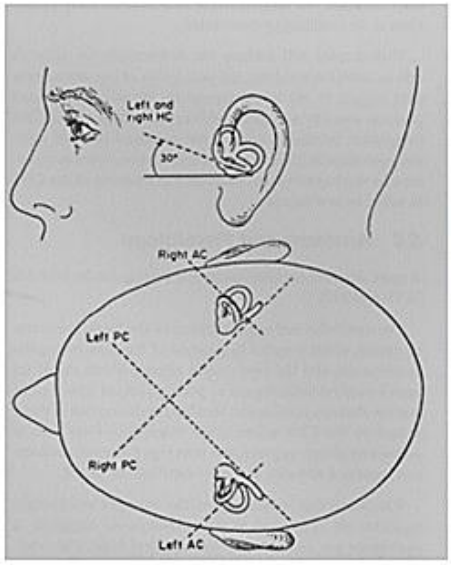

Figure 2: The paring between ears of the semicircular canals: From Lysakowski, A. (2010) Anatomy of Vestibular End Organs and Neural Pathways, In: C.W. Cummings (Ed.), Otolaryngology: Vol. 4, Ear and Cranial Base, St. Louis, (pp. 3089-3111), Mosby-Year Book Inc.

The pairing of the SCCs is critical to the coding of information related to angular acceleration of the head and or body. The role of the SCCs with respect to this coding will be discussed in detail in the physiology section of this literature review.

As previously mentioned, the two otolith or macular organs are the utricle and the saccule. The utricle is positioned in the horizontal plane and the saccule is positioned in the vertical plane, as shown in figure 1. The bundle of hair cells for each of these otolith organs is located in an area known as the macula. The hair cells in the otolith organs are similar to those found in the ampulla of the SCCs. A unique feature of the otolith organs is that they contain calcium carbonate crystals, known as otoconia. Otoconia sit above the gelatinous layer on top of the stereocilia of the hair cells. The weight of the otoconia

rests on the stereocilia of the hair cells and provides information regarding gravitational pull. The gravity of the otoconia is considerably larger (i.e., approximately 2.71- 2.94 times larger) than the surrounding endolymph fluid (Lysakowski, 2010).

Within each of these otolith organs is a thin strip known as the striola. This thin strip spans the diameter of the structures, depicted by the solid line shown in the center of the utricle and saccule seen in figure 3 below.

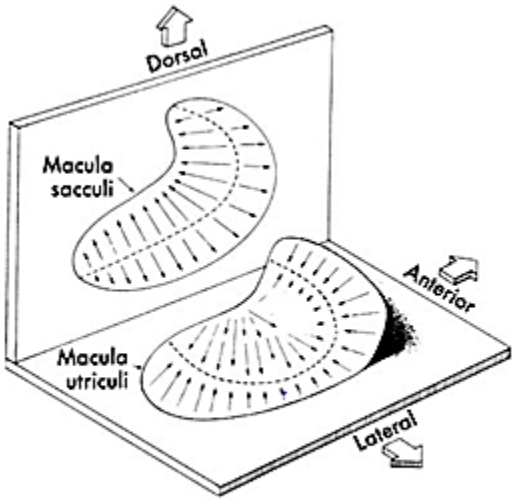

Figure 3: Both the utricle and the saccule. The utricle lies in the horizontal plane and the saccule lies in the vertical plane. The center bold strip within each organ is the striola and kinocillia orientation is reliant on this structure. From Lysakowski, A. (2010) Anatomy of Vestibular End Organs and Neural Pathways, In: C.W. Cummings (Ed.), Otolaryngology: Vol. 4, Ear and Cranial Base, St. Louis, (pp. 3089)

In the utricle, the kinocillia point toward the striola, whereas, the opposite pattern occurs in the saccule, with the kinocillia pointed away from the striola. The different orientation of the stereocilia in the striola is involved in coding of linear acceleration, which will be discussed in detail in the physiology section of this review.

Briefly mentioned above, the three SCCs and the two otolith organs have sensory hair cell receptors. Each of these structures has two types of hair cells which differ in their overall shape as well as their afferent and efferent innervation patterns. Type I hair cells are flask shaped cells and they have direct afferent endings or boutons. These afferent boutons are chalice like in shape and this shape is commonly described as calyx. The calyx encases the majority of the cell membrane as shown by the dark grey band surrounding the type 1 hair cells shown on the left side of figure 4.

The type I hair cells could have either a many to one or a one to one connection. (Wall & Vrabec, 2001) For example, a single type I hair cell could have 1 afferent ending, or be surrounded by 2-4 afferent endings. In the instances where a type 1 hair cell has 2-4 afferent endings this arrangement is referred to as a complex chalice ending. This complex chalice ending is typically found in the otolith organs when the hair cells are located near the striola (Lysakowski, 2010). The type 1 hair cells also have indirect efferent boutons, which attach to the afferent nerve pathway coming off the Type I hair cells.

Also illustrated on the right side of figure 4 are type II hair cells. Type II hair cells are cylindrical in shape. In contrast to type 1 hair cells, type II hair cells have direct

afferent and efferent boutons, meaning these connections directly touch the cell membrane

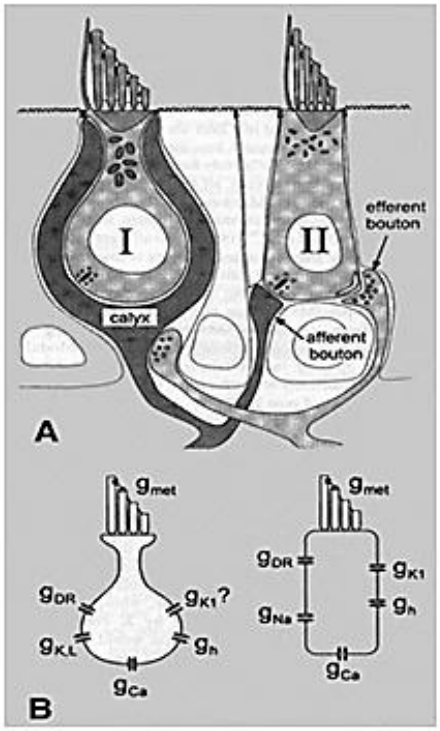

Figure 4: Depiction of type I hair cells located on the left side of the figure and type II hair cells located on the right side of the figure. Type I hair cells are calyx in shape with direct afferent boutons and type II hair cells are cylindrical in shape with efferent and afferent boutons.

The next section of the literature review will focus on the physiology of the peripheral vestibular system.

Physiology of the Peripheral Vestibular System

The primary role of the three SCCs is to code angular acceleration, or changes in angular head velocity. Angular acceleration is experience by most people when they shake their head from one shoulder to the other. In doing so, a set of excitatory and inhibitory responses occur primarily within the pair of SCCs that are coding information for that plane of space. For example, if an individual turns their head or body to the right, this action results in stimulation in the horizontal pair of SCCs (i.e., the right horizontal SCC and the left horizontal SCC). In the ipsilateral horizontal SCC (i.e., the right horizontal SCC) the stereocilia are bent toward the kinocilium. This action results in an increase in the neural firing rate above the spontaneous rate for this SCC and an excitatory signal is sent to the CNS. Simultaneously, the stereocilia in the contralateral horizontal SCC (i.e. the left horizontal SCC) are bent away from the kinocilium. This action results in a decrease in the neural firing signal of the spontaneous firing rate and an inhibitory signal is sent to the CNS. The brain then compares the excitatory and inhibitory input it is receiving from this pair of horizontal SCCs in order to determine the direction of angular acceleration that is occurring due to the head and/or body movement.

The remaining two vestibular organs, the saccule and the utricle, in contrast, are responsible for coding linear acceleration. Linear acceleration is the rate of change that an object moves in one direction over a period of time. The role of both structures is to detect movement changes of the body and then communicate these changes, via hair cells, to be processed more centrally. Although similar in their response capabilities, each structure is specialized in a planar direction of movement.

A good example of linear acceleration would be when a car comes to a sudden halt a force is exerted on that individual; as a result, the individual is thrust forward. The amount of force is dependent on the mass of the person in the car. In the macula this mass element comes from the otoconia that are suspended above the sensory hair cells in the utricle and the saccule. When linear acceleration occurs a chain of events begin. As depicted in figure 5 the gravity moves the otoconia; the macula's gelatinous cap is deflected, and in turn, either depolarizes or hyperpolarizes the sensory hair cells. When hair cells that are specialized in the same direction as the direction of the linear acceleration are displaced, maximum excitation occurs. It should be noted, that the vestibular system which only responds to acceleration and deceleration.

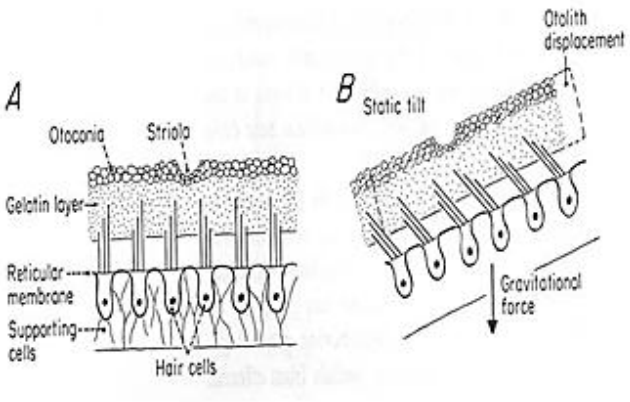

Figure5: Subset A shows the maculae organs at rest. Subset B shows the otolith displacement due to linear acceleration.

Additionally, stereocilia within the organs lie in varying directions using the striola as a reference. Within the utricle, when the stereocilia bend toward the striola an excitatory response is produced. However, when the stereocilia bend away from the striola an inhibitory response is produced. Within the saccule, the opposite is seen. When the sterocillia bend toward the striola inhibition occurs. Conversely, when the stereocilia bend away from the striola excitatory response is produced.

The anatomy and physiology of the peripheral vestibular system has now been discussed. Next in the literature review will be a description of the anatomy and physiology of the central vestibular system.

Anatomy and Physiology of the Central Vestibular System

As the afferent fibers leave the type I and type II hair cells of the five sensory organs located in the peripheral vestibular system, they come together to form the vestibular portion of the vestibule-cochlear nerve. These afferent fibers are neurons of Scarpa's ganglion. As shown in figure 6, the ganglion is divided into the superior branch of the vestibular nerve and the inferior branch of the vestibular nerve. The superior portion of the vestibular nerve receives input from the anterior and horizontal SCCs, the utricle, and the anterior portion of the saccule. In contrast, the inferior branch of the vestibular nerve receives input from the posterior SCC and the posterior portion of the saccule. These afferent fibers join with the cochlear portion of the VIIIth nerve. They then leave the petrous bone through the internal auditory meatus, and course to the junction of the medulla and the pons.

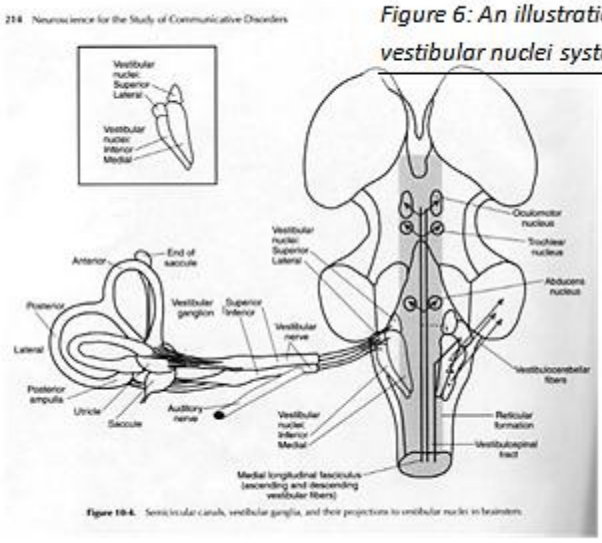

Figure 6: An illustration of the vestibular nuclei system

The vestibular nerve courses dorsally and medially and enters the vestibular nuclei within the brainstem. The vestibular nuclei are divided into four sections: the superior vestibular nuclei, the medial vestibular nuclei, the inferior vestibular nuclei, and the lateral vestibular nuclei (as seen in the inset of figure 6). Each division has a specialized function. The superior nuclei are involved in the vestibular ocular reflex (VOR) pathways. Specifically, the neurons in this nuclei synapse in relation to eye and/or head movements. The neurons in the nucleus project to the ocular-motor nucleus and are important in the control of the VOR. Below the superior nuclei, is the medial vestibular nucleus whose function is related to the cervico vestibulo-ocular reflex. Similar to the superior nuclei, the medial nuclei also projects efferent information to the ocular motor nucleus as well as to the cerebellum. Next to the medial vestibular nuclei are the inferior vestibular nuclei. The inferior nuclei receive a majority of its contributions from afferent

fibers coming from the two peripheral otolith organs. The primary efferent projections from this nucleus are to the cerebellum and to the reticular formation. Above the inferior vestibular nuclei are the lateral nuclei. The lateral nuclei are made up of two nuclei called the ventral lateral vestibular nuclei and the dorsal lateral vestibular nuclei. Afferent information from the lateral nuclei gives rise to the vestibular spinal tract, the vestibule-ocular pathways, and the vestibule-thalamic pathways (Lysakowski, 2010; Bears et. al, 2001).

Extraocular Eye Muscles

The vestibular nucleus also sends projection to the extraocular motor nerves which are involved in regulating eye movements. Eye movements are controlled by six ocular muscles, as illustrated in figure 7. The first four are rectus muscles and the remaining two are oblique muscles. The four rectus muscles and their functions are as follows: the lateral rectus pulls the eye out toward the lateral portion of the body, the medial rectus pulls the eye inward toward the midline, the superior rectus lifts the eye up and slightly off the midline, and the inferior rectus pulls the eye down and slightly off the midline. The two oblique muscles and their functions are as follows: the superior oblique pulls the eye up and approximately 50 degrees off center and the inferior oblique pulls the eye down and 50 degrees off center.

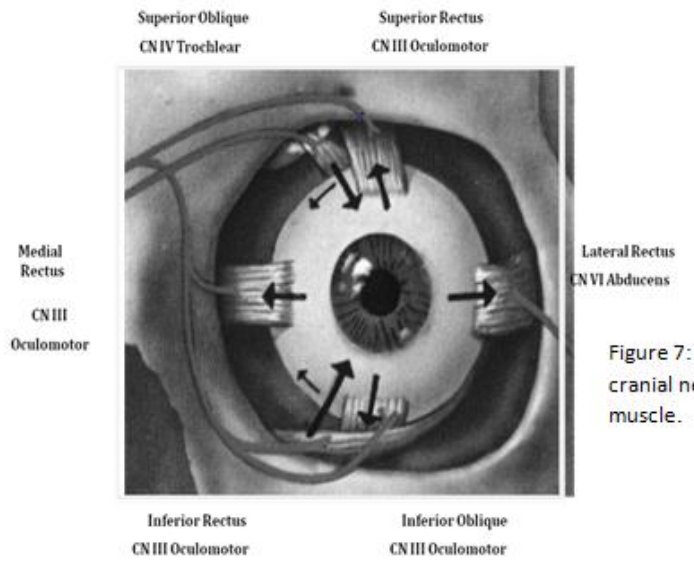

Figure 7: The 6 ocular muscles and the cranial nerve which innervate each muscle.

Each of the 6 ocular muscles is innervated by one of three cranial nerves which are: the oculomotor nerve, or CN III; the trochlear nerve or CN IV; and the abducens nerve or CN VI. The oculomotor nerve innervates the medial, superior, and inferior rectus muscles as well as the inferior oblique muscle. Whereas the trochlear nerve innervates the superior oblique muscle and the abducens nerve innervates the lateral rectus muscle.

Input from the six ocular eye muscles help to stabilize vision during multidimensional head movements. This stabilization is controlled by a reflex known as the vestibular ocular reflex. The next section of this literature review will discuss the vestibular ocular reflex.

Vestibular Ocular Reflex (VOR)

An important role of the central vestibular system is to stabilize movements of the eyes while the head is in motion. This control occurs due to the vestibular ocular reflex (VOR). The vestibular ocular reflex works by sensing motions of the head and the reflex

commands a compensatory movement of the eyes in an equal and opposite direction (Bears et al, 2001). For every one degree of head movement there is a corresponding one degree of eye movement. The eyes also move 180 degrees out of phase in relation to the head movement (Bears et al, 2001). This reflex allows visual stabilization during head movements.

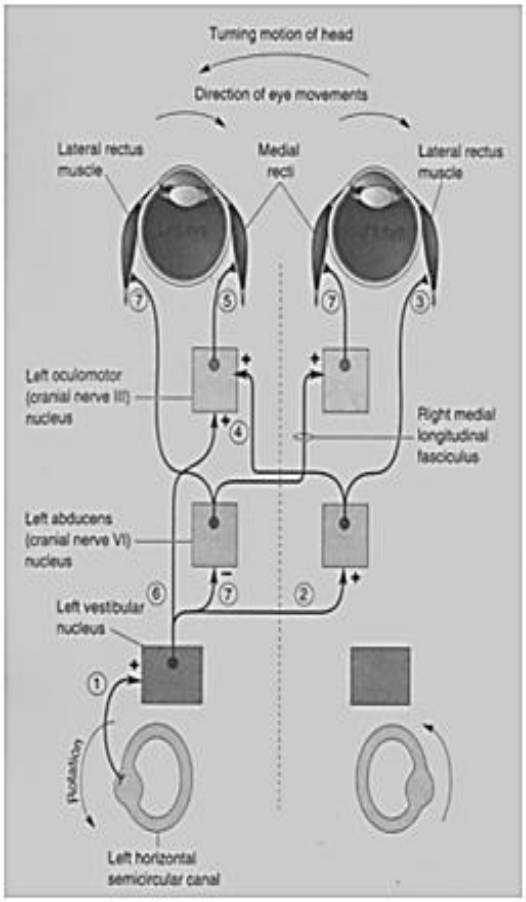

Figure 8: Model of the VOR pathway that is activated when the head is turned to the left causing activation of the horizontal SCC. From Chapter 11: The Auditory and Vestibular Systems in Bears, Connors and Paradiso (2007), Neuroscience Exploring the Brain. (Pgs, 381-386)

As shown in figure 8, if a person turns their head 90 degrees to the left the vestibular ocular reflex (VOR) would trigger both eyes to move in a compensatory movement 90 degrees to the right. The physiology of the reflex arc is also depicted in figure 8. When the head turns to the left, the left axons from the left horizontal canal innervate the left vestibule nucleus, as shown by in the number 1 in this figure. Next, information is sent to the excitatory axons in the right contralateral abducens nucleus (seen in step 2). This leads to an excitatory response being sent to the right lateral rectus muscle (seen in step 3 denoted by a plus sign). This excitatory input results in the right eye being pulled toward the right side. Another excitatory projection from the abducens nerve crosses the midline back to the left side and ascends via the medial longitudinal fasciculus to excite the left oculomotor nucleus (seen in step 4). This signal excites the medial rectus muscle of the left eye, resulting in the left eye drawn to the right side (seen in step 5). Additionally, the left medial rectus is also excited via the projection from the left vestibular nucleus directly to left oculomotor nucleus. To solidify the speed of this operation, the left medial rectus muscle also gets excited via a projection from the vestibular nucleus directly to the left oculomotor nucleus (seen in step 6). An inhibitory response is also activated in the lateral rectus and medial rectus. This inhibition occurs in muscles that may oppose the movement of the eyes moving to the right (seen in step 7). This normal VOR occurs with speed and efficiency with an intact vestibular ocular reflex (Bears et al., 2001).

In addition to the vestibular ocular reflex, there are 2 additional reflexes associated with the vestibular central system. These reflexes are: the vestibular spinal

reflex and the vestibularcollic reflex. The next section will briefly discuss these two reflexes.

Vestibulospinal Reflex and Vestibulocollic Reflex

The vestibulospinal reflex (VSR) controls motor mechanisms involved with maintaining posture and stability during motor limb movement tasks. A normally functioning VSR receives sensory input regarding the orientation of the head, neck, torso, and legs in space.

The VSR consists of three efferent pathways which are: the medial vestibulospinal tract (MVST), the lateral vestibulospinal tract (LVST), and the reticulospinal tract (RST). As shown in the picture below the MVST arise from the superior, medial, and lateral nuclei and there after travels into the medial longitudinal fasciculus. The LVST arise from only the lateral nucleus and then travels downward to the ipsilateral ventral funiculus (Murofushi & Kaga, 2009).

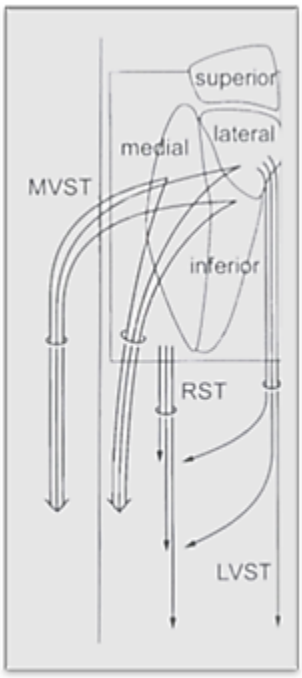

Figure 9: Depiction of the Vestibularspinal reflex pathways: With the medial vestibulospinal tract (MVST) traveling from the superior, lateral, and medial nuclei and the lateral vestibulospinal tract (LVST) originating from only the lateral nucleus. Also depicted in the figure is the reticulospinal tract (RST).

The vestibulocollic reflex is responsible for stabilization of the head through neck movements. Concerning VEMPs, there is a connection between the vestibular otolith organs and the motorneurons within the neck. This connection between the SCM motorneurons, saccule, and utricle was investigated by Kushiro, Zakir, Ogawa, Sato, and Uchino (1999). In their investigation post synaptic potentials were recorded at SCM motorneurons within 20 cats. Partial transection was made at the level of the obex, where the MVST is located. The MVST provides a direct connection from the saccule and the utricle to the SCM motorneurons. Through partial transection, visualization of the pathway between the otolith organs and the SCM motorneurons were made possible. When the saccule was stimulated results yielded inhibitory post synaptic potentials within the ipsilateral SCM motorneurons. In contrast, when the saccule was stimulated there

were little or no effects seen in the contralateral SCM motorneurons. Therefore, Kushiro and colleagues concluded that the pathway from the saccule to the SCM was primarily an ipsilateral dysynaptic inhibitory pathway. As seen in Figure 10 below, the pathway from the saccule connects only to the ipsilateral SCM. This is in opposition to the pathway from the utricle which connects to both the ipsilateral and contralateral motorneurons of the SCM (Kushiro et al., 1999).

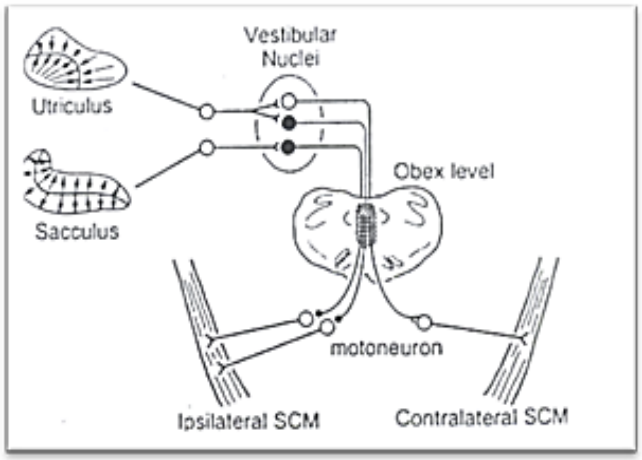

Figure 10: Illustrates the pathway between the saccule and the ipsilateral SCM as well as the pathway between the utricle and ipsilateral and contralateral SCM. The open circles are inhibitory neurons and the closed circles are excitatory.

Overview of the Vestibular Myogenic Response

The VEMP response is an evoked myogenic potential that provides information regarding the integrity of saccule through use of high intensity click or tonal stimuli. VEMP recordings in adults with a normal functioning vestibular system will yield a two component biphasic potential with a peak and a trough. The waveform consists first of a positive peak, relative to the baseline, labeled p13, which has a latency of approximately 13ms. The second component, a negative trough relative to the baseline, labeled n23 has a latency of approximately 23ms. Two later components, labeled n34 and p44 may also be seen during VEMP recordings. However, these later waves are not identified as part of the VEMP response because their origin is considered cochlear in nature (Welgampola & Colebatch, 2005). Based on this, for the remainder of the literature review the VEMP response will consist of peaks p13 and n23.

History of the Vestibular Evoked Myogenic Potential

The myogenic response was not always referred to as the vestibular evoked myogenic potential. Beginning in the mid 1960's, investigators began to explore an electrophysiological potential that was independent of the integrity of the auditory system. Researchers began recording this new averaged response at the inion and based on this recording location, the response received its name "the inion response". Below is a brief description of the history of the inion response and investigation into structures within the vestibular system that are responsible for the generation of this response.

First discovered by Cody, Jacobson, Walker, Bickford, and Minn in 1964, the inion response was recorded by placing an active electrode on the inion and a reference electrode on the ear or the nose. Investigators were interested in determining the clinical usefulness of averaged evoked responses within the audiovestibular system. Specifically, these researchers were interested in determining if this inion potential was mediated by the cochlea or by the vestibular system. Within the study performed by Cody et al. (1964), the inion recordings were conducted on subjects with normal functioning auditory and vestibular systems as well as on subjects with confirmed auditory and vestibular pathologies. The inion response was recorded to 85-120 dB click stimuli.

Waveform results from the normal control group revealed two primary diphasic waves labeled A wave and B wave as well as two secondary diphasic waves labeled C wave and D wave. The earliest wave, A wave, occurred between 6-11ms with the A wave peak latency ranging from 10 to 15msec. The next negative wave, B wave, had an onset that occurred between 23- 28ms with a peak latency that occurred between the ranges of 25-31ms. Cody et al. reported that waves A and B were present in all individuals who had no audiovestibular pathologies, whereas, the secondary waves, C wave and D wave, were only intermittently present across this group of subjects. Therefore, these researchers focused primarily on the first two negative waves, A wave and B wave. Figure 11 below shows an example of an inion response obtained from a normal hearing adult who had normal vestibular functioning (Cody et al., 1964).

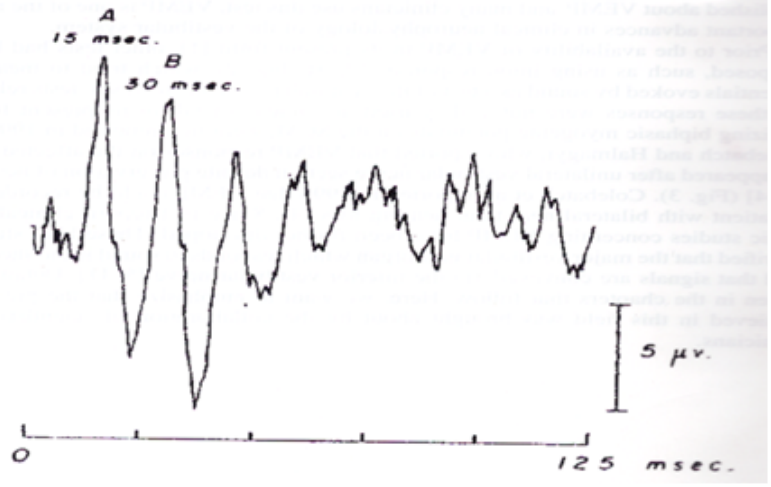

Figure11: Waveform of the inion response recorded on a healthy subject using a 70dBnHL click stimuli. With the peak of A wave and B wave occurring at 15ms, and 30ms respectively. From Murofushi, T. Kimitake, K. (2009). In Vestibular Evoked Myogenic Potentials: Its Basics and Clinical Applications. Tokyo: Springer

As previously mentioned, Cody et al. (1964) also recorded inion responses from individuals with audiovestibular pathology. In one case, the inion response was recorded to click stimuli in a 50 year old man with normal hearing bilaterally, normal vestibular functioning in the right ear, and poor vestibular functioning in the left ear. His waveform results showed a present inion response to right ear stimulation. However, there was no inion response to left ear stimulation. Additionally, when the patient was asked to relax his neck muscles the present response obtained with right ear stimulation disappeared. This case report showed that the inion response was mediated by the audiovestibular system and presence of the response appeared to be dependent upon contraction of the neck muscles. All three conditions for this individual are shown in Figure 12 below.

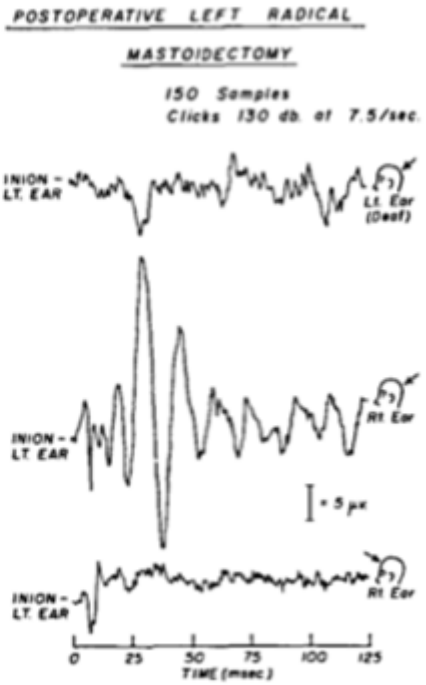

Figure 12: Top tracing-shows an absent response with left ear stimulation in a poor functioning left vestibular system. Middle tracing-shows a present response with right ear stimulation in a healthy functioning right vestibular system. Bottom tracing- shows the present right response disappears with relaxation of the neck muscles. Cody, Jacobson, Walker, Bickford, Minn (1964). *Averaged Evoked Myogenic Cortical Potentials to Sound in Man. Ann Otol Rhinol Laryngol, 73*:763-777.

Cody et al (1964) also reported on the results of another case of a 61 year old woman with a completely deafened right ear and a mild sensorineural hearing loss in her left ear. This individual had a right facial nerve paralysis which resulted from the removal of a right neurofibromatosis tumor on the right VIIIth cranial nerve. Her inion response results indicated an absent waveform when clicks were presented in the right ear; however, when clicks were presented in the left ear the inion waveform was present. The results, depicted below in figure 13, obtained on the 61 year old participant illustrates that the inion response depends on the integrity of cranial nerves VII and VIII.

Another case study investigated a 20 year old woman with a sudden hearing loss in the right ear and normal hearing within the left ear. For this participant, vestibular functioning was normal in both ears. Inion responses indicated present and robust

responses in both ears which are shown in the figure 14 below. These results illustrate that the averaging evoked inion response is independent of the auditory system and therefore must be mediated by the vestibular apparatus.

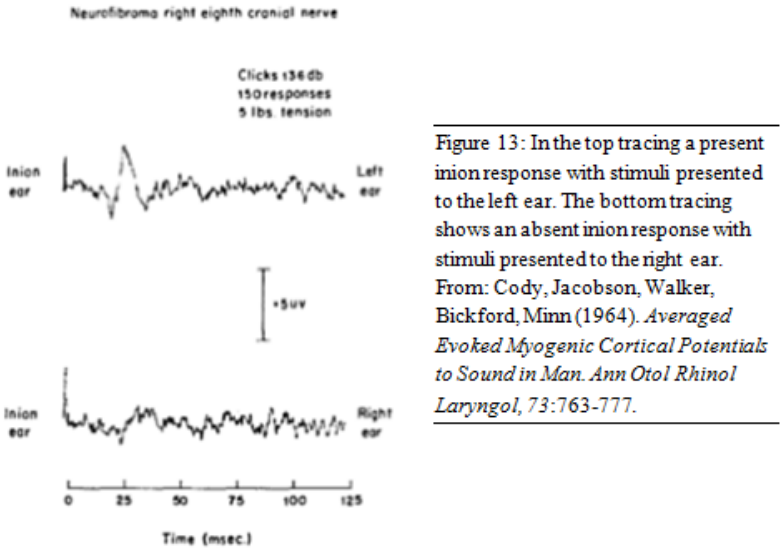

Figure 13: In the top tracing a present inion response with stimuli presented to the left ear. The bottom tracing shows an absent inion response with stimuli presented to the right ear. From: Cody, Jacobson, Walker, Bickford, Minn (1964). *Averaged Evoked Myogenic Cortical Potentials to Sound in Man. Ann Otol Rhinol Laryngol, 73*:763-777.

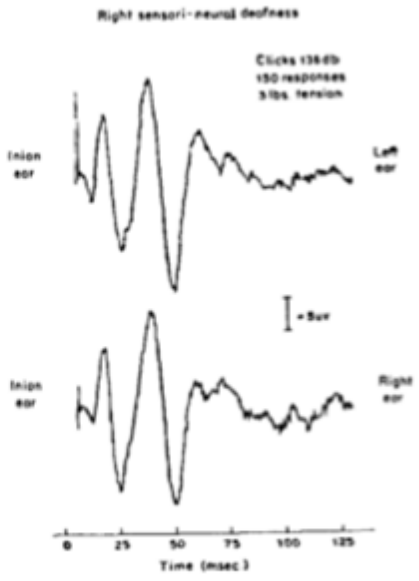

Figure 14: Present responses when click stimuli were presented to the right and the left ear. From: Cody, Jacobson, Walker, Bickford, Minn (1964). *Averaged Evoked Myogenic Cortical Potentials to Sound in Man. Ann Otol Rhinol Laryngol, 73*:763-777.

Based on the results from the above mentioned case studies of patients with normal hearing and balance, and patients with audiovestibular pathology; Cody et al. (1964) concluded that the inion response was mediated by the vestibular system. However, they determined further research was needed to determine which sensory structures within the peripheral vestibular system (i.e. the utricle, the saccule, or the semicircular canals) mediated the inion response.

Based on the previous findings observed in case one of Cody et al.'s (1964) study, the researchers were interested in further examining how muscle tension affects the presence/absence of the inion response. In order to achieve this goal, Bickford and colleagues had 30 normal subjects seated in a chair and calibrated force was placed on the subjects' heads in a forward and a backward direction. This force was delivered through the use of a plastic loop, pulley system, and weights. Inion responses were then recorded using 120 dB click stimuli. As depicted in figure 15, when Bickford and colleagues (1964) applied a forward pressure to the head of their subjects, an increase in the amplitude of the inion response was observed. In contrast, when a backward pressure on the head was applied, this caused a relaxation of the muscles in the neck. The increase in the amplitude of the inion response that was previously seen in 27 of their subjects disappeared with relaxation. Results from this study suggested that the inion response was myogenic in nature and was dependent on the neck muscles with termination near the inion at the scalp (Bickford, Jacobson & Cody, 1964). Based on these findings, the investigators concluded that the presence of the

averaging evoked inion response was dependent on the tension within the muscles of the neck.

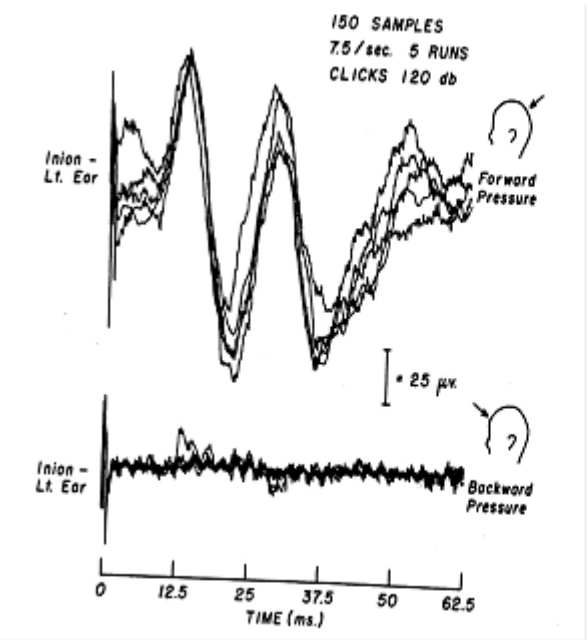

Figure15: With forward pressure the inion response causes a contraction of the neck muscles and the inion response is present/ robust in nature (top tracing). When backward pressure is applied and the neck muscles are relaxed, then the response is abolished. From Bickford, Jacobson, Cody, (1964). Nature of average evoked potentials to sound other stimuli in man. *Annal NY Academy of Science, 112*:204-223

Townsend and Cody (1971) were interested in determining which sensory structures within the peripheral vestibular system (semicircular canals or the otolith organs) were responsible for the inion response. In their study, Townsend and Cody recorded the inion response to a 1000 Hz tone burst at an intensity of 110 dB in a 59 year old man with normal hearing who had experienced streptomycin toxicity. Townsend and Cody reported that the inion response was present bilaterally for this individual. This finding was critical for researchers because a previous study performed by McGee and Olszewski (1962) had demonstrated that streptomycin toxicity destroys the epithelium of the cristae ampulaires within all three SCCs, while the sensory cells of the otolith organs remained intact. Therefore, obtaining a present inion response in a patient who has

damage to his SCCs provided clear evidence that the inion response was not generated within the semicircular canals. Rather, these findings suggested that the end organ receptor for the inion response are the otolith organs (Townsend & Cody, 1971; McGee & Olszewski, 1962).

Townsend and Cody (1971) were also interested in determining which of the two otolith organs (i.e., the saccule or the utricle) was the end organ receptor for the inion response. In order to accomplish this goal, they (1971) recorded the inion response on a patient with bilateral endolymphatic hydrops. In a previous study (Altman & Kornfield, 1965), it was reported that endolymphatic hydrops tend to cause an extreme dilation of the saccule which results in the saccule swelling and extending to surrounding peripheral structures. As a result of the extension of the saccule into neighboring structures, there is a swelling of the walls of the membranous labyrinth. Although significant deformity is noted within the saccule, minimal change is seen within the utricle (Altman & Kornfield, 1965). Therefore, it was reported by Altman and Kornfield that endolymphatic hydrops impairs the saccule more so than the utricle. Townsend and Cody (1971) reported that the inion response was absent bilaterally in the patient with bilateral endolymphatic hydrops which suggests the inion response is reliant upon the integrity of the saccule. (Altman & Kornfield, 1965; Townsend & Cody, 1971).

A resurgence of interest occurred in this myogenic potential when Colebatch, Halmagyi, and Skuse (1994), reported that a stable response from the vestibular system could be obtained when surface electrodes were placed over the SCM instead of the inion. These responses from the SCM were recorded in 10 subjects using 100 dB click stimuli. In their research, Colebatch and colleagues noted that when the active electrodes

were placed over the SCM, it allowed for greater accuracy in determining which muscles were generating the response. Additionally, electrodes placed over the SCM instead of the inion allowed investigators to avoid conflicts with midline recording sites to unilateral acoustic stimulation (Colebatch et al, 1994).

To further investigate this electrode placement, Robertson and Ireland (1995) conducted a pilot study on 7 normal participants and 20 participants with disordered vestibular systems to assess the clinical usefulness of the VEMP recording. They stated that when the electrode placement was changed from the inion to the SCM, changes in the EMG in response to the vestibular afferent nerves were more replicable than the inion response (Colebatch & Halmagyi, 1992; Colebatch et al, 1994, Robertson & Ireland, 1995; Meier-Ewert, Gleitsmann & Reiter, 1974).

Several researchers have agreed that it is optimal to record this myogenic response by placing electrodes on the SCM because of the pathway between the vestibular saccule and the ipsilateral SCM (Colebatch and Halmagyi, 1992; Robertson and Ireland, 1995). It is hypothesized that the p13n23 response travels along the following pathway: from the vestibular saccule, to the inferior vestibular nerve, to the vestibular nucleus, to the lateral Deiter's nucleus, and then to the lateral vestibulospinal tract to the SCM muscle (Ochi et al., 2001) This pathway is commonly referred to as the vestibule-spinal neural projection. Literature speculates that the vibration of the stapes footplate sends impulses to the saccule via the SCM (Colebatch and Halmagyi, 1992, Robertson & Ireland, 1995, Ochi et al., 2001).

In order to elicit a VEMP response many test parameters and subject factors must be considered. These factors will be discussed in the following sections.

Response Measurements of the VEMP Response

Several response indices are used when evaluating the VEMP response to determine normality versus abnormality of the response. These response indices include: absolute latency of waves p13 and n23, amplitude of waves p13 and n23, asymmetry ratio, and VEMP threshold. These four measurements will be discussed below.

Latency of Wave p13 and n23

The absolute latency of p13 and n23 of the VEMP response is taken into consideration for diagnostic purposes. The absolute latency is defined for p13 as the time of stimulus onset to the occurrence of the first positive peak, measured in milliseconds (ms). Conversely, the absolute latency for n23 is defined as the time of stimulus onset to the first negative response, again reported in milliseconds.

Normative data suggests that the mean latency values for healthy adults should be 11.8ms (± 0.86) and 20.8ms (±2.2) for p13 and n23 respectively. In a study by Welgampola and Colebatch in 2001, a VEMP recording was obtained to 100 dBnHL click stimuli in 12 healthy subjects. The average response latency for p13 was 11.5ms and the average response latency for n23 was 20.4ms. Similar results were acquired in the study performed by Colebatch et al. (1994). Click evoked VEMP results from this study indicated mean latency values were 13.3ms (±1.5) and 22.6ms (±2.4) for p13 and n23 respectively.

In another study performed by Basta, Todt, and Ernst (2005) normative latency data was collected using a 500 Hz tone burst instead of click stimuli. Sixty four normal subjects participated in the study and latency data revealed a normative mean p13 latency value of 20.3ms (±2) and a normative mean n23 latency value of 28.0ms (±2) for n23 (Basta et al., 2005).

In a retrospective study conducted by Murofushi, Shimizu, Takegoshi, and Cheng (2001) the clinical usefulness of latency was explored by acquiring latency values in 18 normal subjects as well as those with vestibular pathologies (Ménière's Disease, Vestibular Neuritis, and Vestibular Schwannoma) . Murofushi et al. (2001) recorded responses using click stimuli at an intensity of 95 dB and found the standard deviation (SD) for n23 was greater than the SD of p13. This larger SD indicates more variability in the latency values of n23, hence making p13 a more sensitive indicator of latency abnormality (Murofushi et al., 2001).

Amplitude of Wave p13 and n2

In addition to the absolute latency of waves p13 and n23 used in the assessment of the VEMP response, the peak-to-peak amplitude values of these waves are also taken into consideration when determining abnormality. The peak-to-peak amplitude value is measured from the positive peak of p13 to the negative peak of n23. This peak-to-peak amplitude value varies widely amongst healthy subjects; however, the VEMP literature suggests very broad normative range of values for peak-to-peak amplitude for the VEMP response. This normative range is between 16 to 179microvolts (μV) using a 100 dBnHL

click stimuli and a range of 15-337μV when recording VEMPs to a 500 Hz tone burst stimulus presented at an intensity of 90 dBnHL (Akin & Murnane, 2003).

Normative amplitude ranges were acquired in a study performed by Colebatch and colleagues in 1994. Investigators explored the effects of tonic EMG levels, laterality, and stimulus intensity in 10 healthy subjects, ages 29-63 years. VEMP responses were recorded with the active electrodes placed over the upper half the SCM and a reference electrode over the sternum. VEMPs were elicited using click stimulus at intensities ranging from 75-100dBnHL. At an optimal intensity of 95dBnHL, normal p13n23 amplitude values ranged from 18.3-137.1 μV (Colebatch et al, 1994). Similar results were obtained in a study performed by Welgampola and Colebatch in 2001a. Collectively, these researchers acquired VEMP responses on 70 volunteers using click stimuli presented at an intensity of 100 dBnHL. Peak-to-peak amplitude values collected from this study for all 70 participants ranged from 25-297μV (Welgampola and Colebatch, 2001a).

In the same year, Welgampola and Colebatch (2001b) investigated the characteristic of the VEMP response using tone burst stimuli. Recordings were collected on 12 volunteers using 100 dBnHL clicks using 4 different tonal stimuli (250, 500, 1000, and 2000 Hz). Average amplitude values for each frequency ranged from 30.3- 91.4mV (Welgampola and Colebatch, 2001b).

The amplitude of the VEMP is influenced by many different factors. These factors include: the tension within the SCM, the intensity of the click stimuli, stimulus frequency, and stimulus duration. These factors will be discussed later in the literature

review. To account for the inherent variability present in these amplitude values, many clinical practices calculate asymmetry ratio for amplitude. This ratio allows the patient to act as their own control as the p13-n23 amplitude value obtained on the patient's right side is compared to the p13-n23 amplitude values obtained on that same patient's left side. The amplitude asymmetry will be discussed in detail in the following section.

Amplitude Asymmetry Ratio/ Side-to-Side Asymmetry

Due to the variability in the range of amplitude values for p13-n23 that occurs within normal subjects, best practice indicates that clinicians compare the peak-to-peak amplitudes recorded from the left side to those recorded from the right side to determine pathology. These side-to-side amplitude differences can be calculated as a percent VEMP asymmetry ratio using the formula located below.

$$Percent\ VEMP\ Asymmetry = \left|\frac{(\text{Greater side p13}-\text{n23 amplitude}) - (\text{Lesser side p13}-\text{n23 amplitude})}{(\text{Greater side p13}-\text{n23 amplitude}) + (\text{Lesser side p13}-\text{n23 amplitude})}\right| \text{ x } 100$$

In a study performed by Welgampola and Colebatch (2001a) the asymmetry ratio was calculated in patients of various ages to determine if age affects the asymmetry ratio. Results from 70 participants using click stimuli at 100 dBnHL showed that subjects below the age of 60 years had an upper limit of normal as 35% (Welgampola and Colebatch 2001a). In the proposed study, it is expected that the subjects will range in age from 18-30 years and no age effects are expected. In the proposed study, the amplitude asymmetry ratio will be calculated on normal individuals free of vestibular pathology. The asymmetry ratio will be compared to the upper limits of 35% asymmetry obtained by Welgampola and Colebatch (2001a).

Threshold

Threshold for the VEMP response is considered the lowest intensity level where a visually replicable p13-n23 wave is recorded. The VEMP literature has reported fairly consistent VEMP thresholds for click-evoked responses. Colebatch et al. (1994) showed in 10 healthy adult participants the mean VEMP response threshold to 95 dBnHL click stimuli was 86 dBnHL. Various studies (Akin et al., 2003; Ochi et al., 2001; Welgampola & Colebatch, 2001) have obtained similar threshold values then those obtained by Colebatch et al. (1994) which are listed in table 1. Based on this literature review, in the proposed study VEMP threshold values below 70-75 dBnHL will be considered abnormal for both types of stimuli (i.e., clicks and 500 Hz tonebursts).

Table 1

VEMP Thresholds in dBnHL

	Range of Ages (N)	Stimulus Type and Intensity	Range VEMP Thresholds	Mean Threshold (SD)
Colebatch et al., (1994)	29-63yrs (10)	Click 95	75-85	86
Akin et al., (2003)	22-51yrs (29)	Click 100	80-100	91 (±5.2)
Ochi et al., (2001)	21-38yrs (18)	Click 95	80-95	87.8 (±4.54)
Welgampola & Colebatch (2001)	25-85yrs (70)	Click 100	75-100	89.6 (±6.9)

The table above displays VEMP threshold data obtained in the various studies within the VEMP literature. Denoted above is the range of ages in each study, the stimulus type and intensity used in each study, the range of VEMP thresholds obtained, and the mean VEMP threshold with the standard deviations noted in parenthesis.

Technical Parameters for the VEMP Part I

To obtain optimal VEMP waveform responses appropriate technical parameters should be employed during the recording. This technical parameters section will be divided into two sections. In the first section, the stimulus parameters of type, intensity, rate, and duration will be discussed.

Type of Stimuli

The VEMP response has been evoked through the use of two different types of stimuli, tone bursts and click stimuli. To date, there is some disagreement in the literature regarding which type of stimuli provides the largest amplitude responses and thus the lowest threshold values. These potential conflicts will be discussed below.

Akin, Murnane, and Proffitt (2003) investigated the advantage of recording the VEMP response to tonal stimuli at 120dB in 10 subjects at 6 different test frequencies. These stimulus frequencies include: 250, 500, 750, 1000, 1500,and 2000 Hz. Results from this investigation (Figure 16) showed that the largest p13-n23 amplitude values were obtained at 500, 750, and 1000 Hz. Thereafter, there was a marked decline in amplitude values at 1500 and 2000 Hz. Similar results were obtained in a study conducted by Welgampola and Colebatch in 2001b. Welgampola and Colebatch collected VEMP waveform data on 12 normal hearing participants. In this study, the optimal stimulus frequency and duration was investigated by using varying stimulus frequencies (250, 500, 1000, and 2000 Hz) and varying stimulus durations (1, 3, 5, 7, 10, and 20 ms), with all stimuli being presented at an intensity of 100 dBnHL. The amplitude of p13-n23 wave was largest at 500 Hz, yielding a peak-to-peak amplitude value of 91.4 μV. The

smallest amplitude was 30.3 µV which occurred at 2000Hz. Collectively, the results of these two studies suggest that the optimal frequency to obtain a VEMP response when using toneburst stimuli is 500 Hz frequency level.

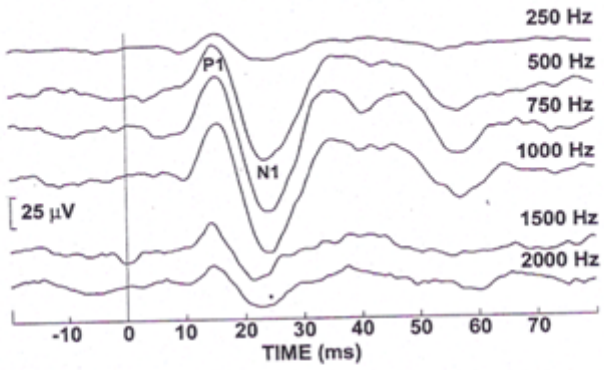

Figure 16: Each waveform is the mean of the three response obtained from on subject. The largest p13-n23 amplitude is noted at 500 and 750 Hz with the smallest amplitudes at 250, 1500, and 2000 Hz. From Akin, Mumane, and Proffitt (2003). The Effects of Click and Tone Burst Stimulus Parameters on the Vestibular Evoked Myogenic Potentail. Journal of the American Academy of Audiology, 12(9), 500-509.

Another advantage of using tonal stimuli discussed in the literature is it elicits VEMP responses at a slightly lower threshold for 500 Hz in comparison to click stimuli. Akin, Murnane, and Proffitt, (2003) investigated the effects of click and toneburst stimuli on the VEMP response. VEMP responses were obtained on 10 participants ranging in age from 22-23 years. As seen in figure 17, results concluded that the VEMP thresholds were

the lowest at 500 and 750 Hz and the highest at 250, 1000, 1500, 2000Hz (Akin, Murnane, & Proffitt, 2003).

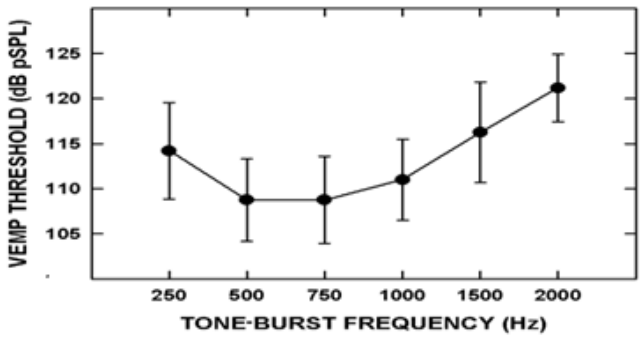

Figure 17: Means and standard deviations for VEMP thresholds obtained using 250, 500, 750, 1000, 1500, and 2000 Hz. Data suggest that the lowest threshold is obtained using 500 and 750 Hz stimuli. From Akin, Mumane, and Proffitt (2003). The Effects of Click and Tone Burst Stimulus Parameters on the Vestibular Evoked Myogenic Potentail. Journal of the American Academy of Audiology, 12(9), 500-509.

Akin et al. (2003) also noted through use of tonal stimuli that the latency of the VEMP response obtained from the 10 subjects using a 2 cycle rise and fall time decreases as the frequency of the tone burst used increases. However, when VEMP responses were obtained using a 4 cycle rise/fall time there was no significant latency difference using the 6 different tone burst frequencies (250, 500, 750, 1000, 1500, 2000Hz).

Conversely, some studies have shown that VEMP recordings show optimal results when obtained from click stimuli. It has been reported that some investigators prefer to use click versus 500 Hz tone burst stimuli when recording VEMPs because when using the latter, it is possible that both the utricular and the saccular hair cells are stimulated, as opposed to the saccular hair cells being stimulated independently when using click stimuli (Murofushi & Kaga, 2009).

Intensity

To elicit a VEMP recording, the stimuli have to be presented at a high acoustic intensity. This is evidenced in a study performed by Colebatch et al. in 1994 in which VEMP responses were recorded using click stimuli over a range of 85-100 dBnHL and were adjusted in 5 dB increments. Ten healthy participants with no history of audiovestibular disorders participated in this study. Colebatch and colleagues (1994) demonstrated that p13-n23 amplitude values increased as a function of click intensity with mean threshold occurring at 85 dBnHL. The p13-n23 amplitudes were 36% larger using a stimulus intensity of 100 dBnHL in comparison to the p13-n23 amplitude value obtained for the response obtained at 95dBnHL. This effect of stimulus intensity is illustrated in Figure 18 below (Colebatch et al, 1994). Similar findings were obtained a year later by Lim, Clouston, Sheeam, and Yiannikas (1995). Click evoked VEMP responses obtained on 10 healthy subjects ranging in age from 20-51 years showed that the p13-n23 amplitudes were ~30% larger using a click at an intensity of 100 dB in comparison to amplitude values obtained to clicks presented at 95dBnHL (Lim et al., 1995).

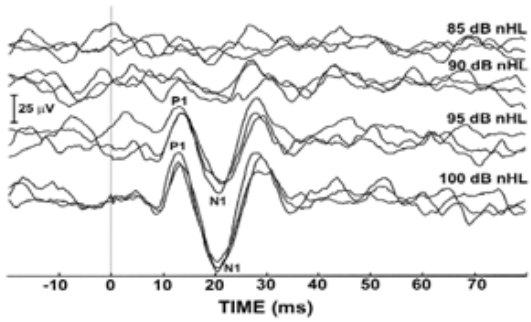

Figure 18: Waveform responses obtained from one participant using various intensity levels. P13-n23 amplitude is largest at 100 dBnHL. From Akin, Murnane, and Proffitt (2003). The Effects of Click and Tone Burst Stimulus Parameters on the Vestibular Evoked Myogenic Potentail. Journal of the American Academy of Audiology, 12(9), 500-509.

Rate

The optimal repetition rate used for recording the VEMP response has been investigated by Wu and Murofushi (1999). Wu and Murofushi (1999) recorded VEMP responses at varying stimulus rates (i.e., 1, 5, 20 Hz) on 12 healthy subjects using click stimuli at an intensity level of 95 dBnHL. Wu and Murofushi (1999) found that the highest VEMP amplitudes were obtained using a rate of 1 Hz and 5 Hz. Results showed that as the repetition rate increased, the VEMP amplitude decreased. It was noted that at the highest repetition rate of 20 Hz, the intersubject variability was the greatest and at the lowest repetition rate of 1 Hz the subject recordings were the most consistent. Although a repetition rate of 1 Hz yields the most consistent results, decreasing the repetition rate

requires a longer recording time to average the response. Subjects therefore have a tendency to become tired because they have to contract the SCM for a longer time (Wu & Murofushi, 1999). Therefore, Wu and Murofushi recommend that the optimal repetition rate for recording VEMPs is 5 Hz. In the proposed study, a 5 Hz repetition rate will be employed for both the click and tonal stimuli.

Duration

Huang, Su, and Cheng (2005) looked at the effect of click duration on vestibular evoked myogenic potentials in 18 healthy participants. In this study, varying click durations (0.1, 0.2, 0.5, and 1.0ms) were delivered at a stimulus rate of 5 Hz and a stimulus intensity of 105dBnHL. Results indicated that the highest/largest VEMP amplitudes were obtained using click durations of 0.5 and 1.0ms. Huang and colleagues also reported that as the click duration was shortened (i.e., 0.1, and 0.2ms), the amplitude of the VEMP decreased. Therefore, they determined that click durations of 0.1 and 0.2ms were ruled out as optimal choices for the duration of the click stimulus when recording the VEMP response. Rather, Huang et al., (2005) suggest that duration of 0.5ms for click stimuli be used because this duration showed smaller interaural latency differences and had less sound energy exposure.

In the proposed study, we will use click duration of 0.5ms. Although a duration of 0.5ms was determined as optimal for click stimuli, this is not the case when recording VEMPs to tonal stimuli. In a study performed by Welgampola and Colebatch in (2001b), VEMP recordings on 12 normal hearing participants were examined. On these

participants the optimal stimulus frequency and duration were investigated by using varying frequencies (250, 500, 1000, and 2000 Hz) and varying durations (1, 3, 5, 7, 10, and 20ms) at an intensity of 100 dBnHL. The study reported that the largest peak-to-peak amplitude values for the VEMP responses in their 12 participants occurred at durations of 7ms.

Technical Recording Parameters of the VEMP Part II

The recording parameters that will be discussed in this section include: Analog band pass filter setting, effects of electrode montage, effect of patient positioning, effect of tonic contraction, number of sweeps/averages, and unilateral versus bilateral recordings. Each of these parameters will be briefly discussed below.

Analog EEG Band Pass Filter Settings

To obtain an accurate VEMP response appropriate analog band pass filter settings are required for the recording. The response goal of the band pass filter setting is to record the spectral energy of the EMG response. The band pass filter settings that have been successfully used in recording the VEMP response have varied somewhat in the literature, with the high pass settings ranging from 8 Hz to 20 Hz and the low pass filter settings ranging from 1500 to 2000 Hz (Huang et al, 2005; Murofushi & Kaga, 2009; Welgampola and Colebatch, 2003). In the proposed study, an analog band pass filter setting of 50 – 2000 Hz will be employed to capture the spectral energy present in the VEMP.

Electrode Montage

Earlier investigators (Bickford et al., 1964; Cody et al., 1964; Cody & Bickford, 1964) believed that the optimal site of excitation for the VEMP response was an inion response. In an early study performed by Bickford et al., (1964) myogenic responses were recorded at the inion. However in a more recent study performed in 1992 by Colebatch and Halmagyi it was determined that using the SCM as a recording site yielded robust VEMP waveforms in all participants. This placement on the SCM was later adopted in the majority of studies to follow.

Sheykholeslami, Murofushi, and Kaga (2001) were interested in determining where the optimal location on the SCM for placing the recording electrode during VEMP testing. In this study, Sheykholeslami et al. (2001) placed the remaining electrodes as follows: a reference electrode placed over the upper sternum and a ground electrode is placed on the midline of the forehead (Fpz). VEMP recordings were acquired on fifteen subjects using tonal stimuli presented at an intensity of 95 dBnHL. The VEMP was recorded at four locations which include: the clavicle, the sternum, the middle part of the SCM, and the upper portion of the SCM, which are depicted in figure 20. The results clearly demonstrated that the largest peak-to-peak amplitudes for p13 and n23 occurred on the upper and middle parts of the SCM, as shown in the waveform figures displayed on the right side of figure 21 (Sheykholeslami et al., 2001).

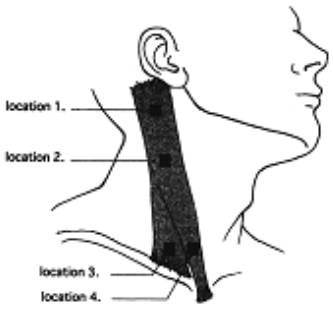

Figure 19: Depiction of electrode location. 1) Upper portion of the SCM, 2) Middle portion of the SCM, 3) Clavicle, 4) Sternum. From Sheykholeslami, Murofushi, and Kaga (2001). The effects of sternocleidomastoid electrode location on vestibular evoked myogenic potentials. *Auris Nasus Larynx, 12*:41-43

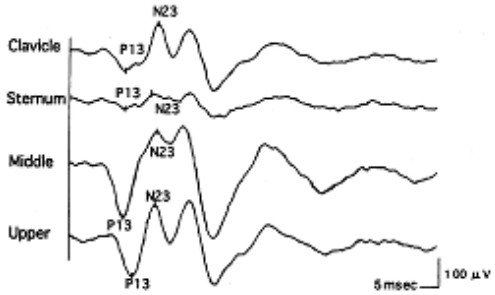

Figure 20: Waveform responses of a participant at varying electrode locations. Peak to peak amplitude is largest at the middle and upper portion and the SCM. From Sheykholeslami, Murofushi, and Kaga (2001). The effects of sternocleidomastoid electrode location on vestibular evoked myogenic potentials. *Auris Nasus Larynx, 12*:41-43

In the proposed study, the electrode montage will be the following: the active electrodes will be placed on the upper 1/3 of the left and right SCM; a reference electrode will be placed on the upper sternum; and the ground electrode will be placed in the middle of the forehead.

Patient Positioning

As previously mentioned, the amplitude of p13 and n23 are dependent on the contraction of the SCM. Proper patient positioning is critical to obtain activation of the sternocleidomastoid muscle (SCM). Varying positioning techniques have been used to

successfully record the VEMP response. Three of the most common ways to elicit sustained tonic contraction of the SCM will be discussed.

In a study conducted by Colebatch et al. (1994) VEMP recordings were successfully recorded with the patient lying in the supine position with the head slightly raised and turned as far as possible to the contralateral shoulder. This forceful pushing towards the contralateral shoulder allows for tensing of the ipisilateral SCM. Colebatch et al., (1994) also successfully recorded p13-n23 by having the patient place their forehead on a bar positioned in front of them to ensure maximum activation of the SCM. Another method to elicit the SCM contraction is to elevate the head with either a pillow or the patient laying in the supine position and holding his head up (Akin and Murnae, 2008). In a study performed by Ferber-Viart et al. (1997), a rubber ball was wedged under the patient's chin, while his head was pointed toward their chest. This test protocol allowed for monitoring of the contraction of the patients SCM. Wang and Young (2006) explored the difference between head elevation and head rotation to activate the SCM. Results obtained from their study showed that the head elevation method yielded a greater response rate and significantly larger amplitudes when compared to the head rotation method (Wang & Young, 2006). In the proposed study the patient will be reclined in a comfortable chair, with their head slightly raised and turned toward the contralateral shoulder.

EMG Recording

As noted in the amplitude section of this literature review, the amplitude of the VEMP response aids in the interpretation of an abnormal response. However, there is

great variability in the normative range of amplitude values. One factor that influences the magnitude of the amplitude is the strength of contraction of the SCM. Because the VEMP is a short latency electromyogram (EMG), and dependent on the tonic contraction of the SCM, it is important for patients to maintain an appropriate level of tonic EMG to successfully record a response. This portion of the literature review will review two important topics in this area: (1) how the tonic EMG level is monitored by clinicians and (2) the target EMG levels that are used to record successful VEMP waveforms.

One method of monitoring the rectified EMG level was described in a study performed by Colebatch et al. (1994). In this study, the EMG level was collected 20ms prior to stimulus onset and 80ms following. The rectified EMG level was low passed filtered and displayed on an oscilloscope screen which provides visual feedback to the participants. Through use of the Medelec Sensor EMG monitoring system, all 70 participants were able to successfully reach a target EMG level of 50-60μV. They were then asked to watch the oscilloscope screen and maintain the rectified EMG level just above the target level throughout the recording period. Typically these averages lasted approximately 3 minutes (Colebatch et al., 1994).

A study by Vanpsauwen, Wuyts, and Van de Heyning (2006) investigated if the reliability of the VEMP could be improved by using a blood pressure manometer. In this study 22 participants used a blood pressure manometer with inflatable cuff. For example, if VEMP recording were recorded on the patient's left side, the patient was instructed to flex his/her head 30 degrees forward and then rotate their head to the right. The blood pressure cuff was then inflated to 20mg and the patient was told to hold the cuff between their right hand and jaw until the pressure read by the manometer reached 40mg. VEMP

waveforms were recorded in two different test conditions. In condition 1, VEMP waveforms were recorded while the blood pressure manometer was held to a constant setting of 40 mg. In condition two, each subject was asked to push their jaw against the hand of the clinician to cause a certain level of tension of the SCM. Results of this study demonstrated that the mean amplitude of the VEMP was significantly less variable when using the manometer (mean = 34μV, SD=25) in comparison to the mean amplitude of the VEMP obtained (mean = 104 μV, SD=72) simply by having the patient apply force to the investigator's hand. These findings illustrated that the blood pressure manometer can successfully achieve and monitor adequate tonic EMG levels to successful record VEMP responses (Vanpsauwen et al., 2006).

In the proposed study, maintaining and acquiring an adequate target EMG level will be maintained and monitored through use of the DelSys EMG monitoring software. Subjects will be asked to achieve a tonic EMG level of 50μV and maintain this level for the left and right ear. They will be provided with a visual monitor to aid them in achieving and maintaining the tonic EMG level of 50 μV throughout the VEMP recordings.

In monitoring the tonic EMG level it is important to determine what target level should be reached and maintained by patients. Akin, Murnane, Panus, Caruthers, Wilkinson, and Proffitt (2004) researched the ability of participants to reach various target levels, as well as the effects that EMG target levels had on the latency and amplitude of click and tone burst VEMP recordings. In their study, clicks and 500 Hz tone bursts were presented at an intensity of 100 dBnHL in 11 volunteers. As shown in figure 21, the amplitude of p13-n23 increased as the target EMG level increased.

However, the latency of the VEMP was independent of target EMG level. These amplitude and latency findings were true regardless of stimulus type. Additionally, for both stimulus types, the variability of the amplitude was least at target levels of 30µV and 50µV, as shown below in figure 22. Therefore, to collect normative data in the proposed study, the EMG target used will be 50µV.

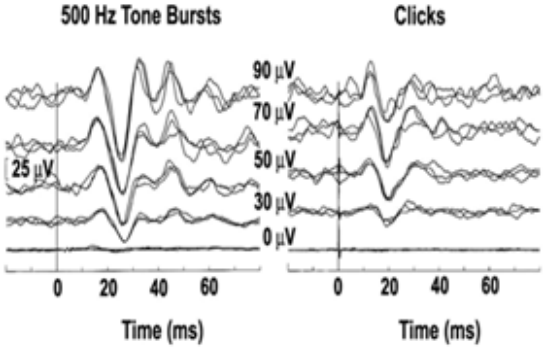

Figure 21: VEMP waveforms acquired using tone burst and click stimuli from a single participant. The target EMG levels are in the center. From: Akin, Murnane, Panus, Caruthers, Wilkinson, Proffitt, T.M., (2004). The influence of voluntary tonic EMG level in the vestibular evoked myogenic potential. *Journal of Rehabilitation Research & Development 41* (3B):473-480.

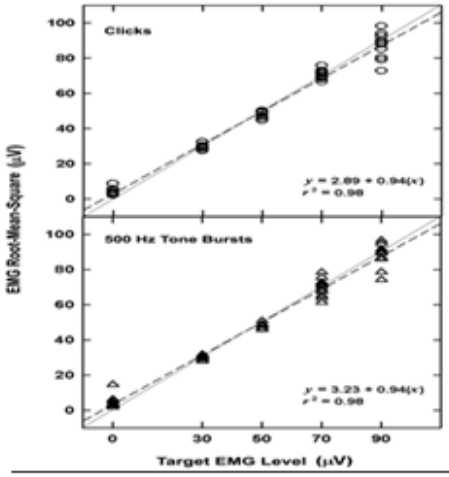

Figure 22: Plot of EMG amplitude as a function of target EMG level for both stimulus types. The solid line is when the EMG level equals the target EMG level and the dashed line is the linear functions of target EMG level. The least variable amplitudes occur when achieving a target level of 30 and 50 mV. From Akin, Murnane, Panus, Caruthers, Wilkinson, Proffitt, T.M., (2004). The influence of voluntary tonic EMG level in the vestibular evoked myogenic potential. *Journal of Rehabilitation Research & Development 41* (3B):473-480.

Number of Sweeps

In recording the VEMP response to high intensity click and/or 500 Hz toneburst stimuli, the peak-to-peak amplitude of p13-n23 is typically robust in nature. The mean amplitude values range from 16 to 179µV (Akin & Murnane, 2008). Because the VEMP is an EMG response, waveform identification is easier compared to other electrophysiological measures. For instance, in the auditory brainstem response (ABR), when there are low stimulus intensities, wave V amplitude is observed with a range from 0.10-0.30µV (Oates & Stapells, 1998). Because this amplitude is relatively small a larger number of sweeps are needed in order to identify the ABR response. In contrast, the amplitude of the VEMP response is larger (i.e., 16 to 179µV) in comparison to the ABR wave V amplitude. Therefore, a smaller number of sweeps is sufficient for recording the myogenic response because of its larger magnitude makes the response easier to identify. Several previous studies have successfully recorded the VEMP using approximately 120 – 150 sweeps per run. In the proposed study, we will conduct 128 sweeps per run. We will also replicate each run for a total of 256 sweeps per test condition. If it is determined that a run is not replicable, then a third or fourth run will be obtained. This will be done on an as needed basis.

Unilateral versus Bilateral and Laterality

Colebatch and investigators (1994) also explored amplitude and latency of the VEMP response when acquired with stimuli presented unilaterally versus bilaterally. Using click stimuli at an intensity of 100 dBnHL, when VEMP recordings were obtained

from only the contracted SCM with the stimuli presented to the ipsilateral ear p13-n23 amplitudes that were 98% larger than those obtained from the contracted SCMs with the stimuli presented bilaterally. For example, if the recordings were taken from the right contracted SCM, with the stimuli presented to only the right ear, then the p13-n23 amplitude would be 98% larger than the p13-n23 amplitude obtained from the recordings from the right contracted SCM if the stimuli were presented bilaterally. This observation is noted because it is difficult for a patient to maintain a position in which both the left and right SCM is contracted. These investigators also reported that if two recording channels (i.e., ipsilateral and contralateral) were used in the monaural test condition, often the VEMP response was absent or significantly reduced in amplitude in the contralateral recording channel (Colebatch et al., 1994).

Murofushi, Ochiai, Ozeki, and Iwasaki (2004) also investigated the laterality of the VEMP responses in 20 healthy adult participants. VEMP waveforms were recorded using click and 500 Hz short tone bursts, with both stimuli being presented at an intensity of 95 dBnHL. Results from this study concluded that VEMP potentials evoked using click stimuli are more likely to be present in the ipsilateral channel than in the contralateral channel. However, a somewhat different pattern of results emerged for VEMPs recorded to tonal stimuli. In this case, VEMP waveforms obtained using tonal stimuli showed ipsilateral dominance in 76% of the cases. In the remaining 24% of the cases, VEMP responses were not ipsilateral dominant. However, presence was seen in the contralateral tracing as well (Murofushi et al, 1994).

In another study performed by Bhagart in 2006, the goal was to observe the differences, if any, between using monaural and binaural stimuli when the VEMP

responses were recorded unilaterally. For the monaural condition, a 500 Hz tone burst was presented to the ear ipsilateral to the contracting SCM. In the binaural condition, the 500 Hz tone burst was presented ipsilaterally, while one of the four frequencies (250, 500, 750, 1000 Hz) was presented to the contralateral ear. Results from 18 subjects showed the amplitude of the p13n23 VEMP response in the binaural condition was 7-17% smaller than the amplitude of the p13n23 response when stimuli were only presented monaurally (Bhagart, 2006). In the proposed study, VEMP recordings will be presented monaurally in the left and right ears separately at varying stimulus intensities.

Subject Variables

In addition to using specific technical parameters used to optimize the VEMP recording, clinicians must also consider subject-related factors that may influence the presence of the VEMP potential. The VEMP may be affected by several subject factors, such as age and gender. These factors will be discussed in the next section of the literature review.

Maturation

Maturational-related changes are also an important subject factor when assessing VEMP amplitudes. The VEMP has been successfully recorded in infants ranging in age from 1 to 12 months. However, it should be noted that some amplitude and latency differences may occur. These age-related changes include variability in amplitude and shorter p13 and n23 latency values when compared to healthy adults. These changes were observed in a study performed by Chen, Wang, Wang, Hsieh, and Young (2007). Chen and colleagues recorded VEMPs in newborns 2-5 days old using 500 Hz short tone

bursts presented at an intensity of 95 dBnHL. The infants' VEMP waveforms were compared to the adults' responses. These investigators reported that the infants had significantly smaller p13-n23 amplitude values, increased latency values for p13 and n23, and lower interpeak amplitude values for p13-n23. Chen et al, (2007) attributed these maturational differences between adults and newborns to the incomplete maturity of the myelin sheaths in the infants' vestibular sacculocollic reflex pathways.

Age

Literature suggests that various response indices used to classify the VEMP response are affected by increases in an individual's age (Welgampola & Colebatch, 2001a; Akin and Murnane, 2008; Chang, Cheng, and Young, 2010). These changes include: a decrease in p13-n23 amplitude, an increase in the asymmetry ratio, and an increase in the mean threshold value. These changes are expected due to other age related changes affecting the vestibular system. These changes include: a decrease in vestibular hair cells by 6% per decade between the ages of 40 and 90 (Rosenhall, 1973), a decrease in vestibular nerve fibers (Bergstrom, 1973), a decrease in the number of cell bodies in Scarpa's ganglion (Richter, 1980), and a loss in neurons in the vestibular nuclear complex (Lopez et al., 1997).

In a study conducted by Welgampola and Colebatch in 2001a the affects of age on the VEMP response was explored. VEMP responses were obtained in 70 participants ranging in age from 25 to 85 years. Click-evoked recordings were collected using an intensity of 100 dBnHL. Results from this study showed age-related changes for the VEMP threshold, the asymmetry ratio, as well as the p13-n23 amplitude. First, click

thresholds for all 70 participants ranged from 75dBHL to 100 dBnHL. The mean VEMP threshold for subjects in their 3rd decade of life was 85dBnHL. In contrast, for older participants (i.e., those in their 8th and 9th decade of life), their mean VEMP threshold was 96 dB. A second response index for the VEMP that was influenced by age was the asymmetry ratio. The average asymmetry ratio for all 70 participants was 23.8%. For participants below the age of 60 years, their mean asymmetry ratio was 16.3%. This is in sharp contrast to the mean asymmetry ratio of 37.6% for participants over the age of 60 years. In addition to threshold and asymmetry ratio, the amplitude of the VEMP response is also affected by age. In this study, corrected amplitude values were used. Results showed that during the 3rd decade of life, the corrected amplitude value was 1.77. This decreased to 0.62 during the 8th and 9th decade of life. The decrease in amplitude was noted at the beginning of the 6th decade of life. Therefore, these investigators' (2001a) results demonstrated that in the 6th decade of life, there is an increase in VEMP thresholds, a decrease in the amplitude asymmetry ratio, and a decrease in amplitude of the VEMP response.

Brantberg, Granath, and Schart (2007) also investigated the effects of age on the VEMP response. Results from their study confirm data obtained from the previous study (Welgampola and Colebatch, 2001a), which noted a decrease in VEMP amplitude as age increases. Brantberg and colleagues reported that VEMP amplitudes decrease at a rate of 0.24μV per year.

Gender

Unlike age-related changes, the effects of gender on the VEMP are less clear. Most literature states that there is no significant amplitude or latency difference in the VEMP response as a function of gender (Brantberg et al 2007; Akin et al, 2003; Ochi & Ohashi, 2003; Basta et al. 2005). However, in a study performed by Brantberg and Fransson (2001) there was a significant age-sex interaction effect for females. VEMP responses were recorded in twenty three healthy participants using click stimuli presented at an intensity of 100 dBnHL. Brantberg and Fransson (2001) reported that as both genders aged, females had significantly shorter absolute latencies for p13 in comparison to males. Brantberg and Fransson hypothesized that this effect was because females tend to have shorter latencies in auditory brainstem responses than males due to their smaller head size (Brantberg & Fransson, 2001).

Clinical Relevance of the VEMP

The clinical application of the VEMP has been explored in the following audiovestibular pathologies: vestibular neuritis, superior semicircular canal dehiscence, Ménière's disease, and vestibular schwannoma's. In these four areas, VEMP abnormality generally consists of absent responses, decreased threshold, or amplitude asymmetry differences. The following section will discuss the specific results of the VEMP for the previously mentioned audiovestibular pathologies.

Vestibular Neuritis

Vestibular Neuritis, or neurolabyrinthitis, is a viral infection that affects the vestibular nerve. It is characterized by a sudden onset of severe vertigo, generally lasting

for several hours or days in the absence of auditory impairment (Akin & Murnane, 2008). Diagnosis is usually based on medical history and the results of caloric testing. The caloric response assesses the integrity of the horizontal SCC's and the superior branch of the vestibular nerve. If results from caloric testing for the suspected ear are abnormal (i.e. reduced or an absent response), such findings would suggest that there is a problem with the integrity of the superior vestibular nerve for these patients.

Although a majority of patients with vestibular neuritis have impairment of the superior vestibular nerve, there is a small subset of vestibular neuritis patients in which the disease only affects the inferior portion of the vestibular nerve. In cases in which the inferior portion of the vestibular nerve is impaired, the VEMP response would be abnormal because VEMP testing examines the inferior portion of the vestibular nerve. In this literature review, several cases of vestibular neuritis affecting the inferior portion of the vestibular nerve will be discussed.

Murofushi, Halmagyi, Yavor, & Colebatch (1996) obtained VEMP responses on 47 subjects with vestibular neuritis. The goal of their study was to evaluate the clinical usefulness of the VEMP response in assessing patients with vestibular neuritis. VEMP responses were obtained using click stimuli presented at 100 dBnHL. VEMPs were present in 31 of the 47 (66%) patients, suggesting that these individuals had vestibular neuritis due to the superior branch of the vestibular nerve. In contrast, the VEMP response was absent in the remaining 16 (34%) patients. Murofushi et al. (1996) suggested that the 34% of the population with absent waveforms had vestibular neuritis affecting the inferior vestibular branch. In a study performed in 2010 by Zang, Fan, Han, Yu & Wang 216 patients were tested with documented symptoms of vertigo. All patients

had radiologic, hearing, caloric, and VEMP testing performed. Eight out of the 216 participants were diagnosed with inferior vestibular neuritis. VEMP responses were obtained from these 8 patients using 500 Hz short tone bursts presented at 100 dBnHL. The results revealed all 8 patients had normal caloric testing and abnormal VEMP responses. The abnormal VEMP findings included either absent VEMPs or decreased amplitude of the p13-N23 components. These abnormal findings assisted the investigators in confirming a diagnosis of inferior vestibular neuritis and also provided additional clinical research evidence that a less common subset of vestibular neuritis does exist (Zang et al., 2010).

Superior Semicircular Canal Dehiscence

Superior Semicircular Canal Dehiscence (SSCD) is characterized by sound and/or pressure-induced vertigo caused by a dehiscence of the bone overlying the superior semicircular canal (Minor, Solomon, Zinreich, & Zee, 1998). A dehiscence is a thinning of the bone. Within the superior SCC, the thinning occurs on the roof of the canal (Minor et al., 1998) Additional symptoms and clinical findings for SSCD include: oscillopsia, conductive hyperacusis, vertigo, and nystagmus induced by loud sounds (Tullio Phenomenon), audiometric air bone gap in the presence of acoustic reflexes, and low threshold/ high amplitude VEMP responses (Akin & Murnane, 2008).

In patients with SSCD there are two anatomically normal windows. They are the oval and round window with the addition of an atypical window/ opening into the inner ear. This abnormal additional window allows access from the membranous canal into the middle cranial fossa and then acts as a place of increased compliance into the inner ear

(Strebel, Cremer, Carey, Weg, Minor, 2001). VEMP thresholds are lower than normal in this clinical population because the dehiscence creates an increase in the sensitivity of the vestibular receptors when there is a presence of pressure.

Brantberg, Bergenius, Mendel, Witt, Tribukair, and Ygge (2001) reported on the symptoms, findings, and treatment of the eight patients with SSCD. VEMP responses were obtained using click stimuli with intensities ranging from 60-100 dBnHL. VEMP waveform results from all 8 subjects showed a decrease in the VEMP threshold. This indicated a hypersensitivity to sound created by the additional mobile window. Below in table 23, is a typical VEMP waveform of an individual with SSCD. This individual had an SSCD in their right ear and a normal functioning vestibular system in their left ear. The VEMP responses for the right ear show that the p13 component is clearly present and replicable down through 65 dBnHL. In contrast, the VEMP response completely disappears at levels below 90 dBnHL in the left ear.

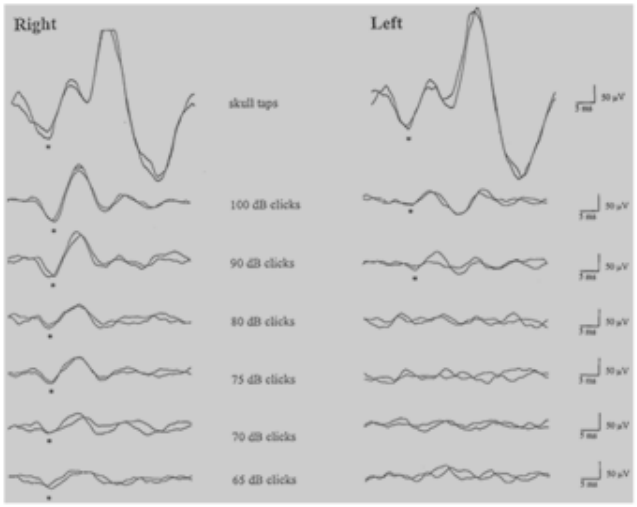

Figure 23: Typical VEMP response from an individual with SSCD in the right ear and a normally functioning vestibular system in the left ear. In the right ear the p13 peak can be easily identified at 65 dB (threshold) however, in the left ear the p13 peak becomes unidentifiable after 90 dB (threshold). Note threshold is much higher in the left ear. From Brantberg, Bergenius, Mendel, Witt, Tribukait, Ygge,(2001). Symptoms, Findings and Treatment in Patients with Dehiscence of the Superior Semicircular Canal. Acta Otolaryngol, 121:68-75

In 2003, Cox, Lee, Carey, and Minor documented a case of a 60 year old man with a moderate rising to mild low frequency hearing loss, normal mid frequency hearing proceeding to a slight high frequency hearing loss in both ears. The patient had normal tympanograms and elicited ipsilateral and contralateral acoustic reflexes at expected levels. VEMP testing was performed at intensities ranging from 60-103 dBnHL using click stimulus. VEMP responses obtained had abnormally low threshold of 80dBnHL. According to this case study, the threshold for normal vestibular functioning individuals is >85dBnHL. This study revealed that within the clinical population of patients with SSCD, it is expected that the VEMP threshold will fall below the normative range. This

is due to vestibular hypersensitivity to sound caused by an additional mobile window within the membrane (Cox et al., 2003)

Vestibular Schwannoma

Vestibular schwannomas are tumors that arise from Schwann cells and rest on the vestibular portion of the eighth nerve. To determine if the tumor is located on the superior or inferior portion of the vestibular nerve, caloric testing is administered. Results from caloric testing reflect the integrity of the horizontal semicircular canals and the functioning of its afferent nerve branch, the superior vestibular branch. If the result of caloric testing is abnormal, then it can be inferred that the tumor has affected the superior branch of the vestibular nerve. It has been demonstrated that Vestibular schwannomas most often occur on the vestibular branch of the eighth nerve prior to the cochlear portion of this nerve. With the advent of the VEMP response, assessment of the inferior branch of the vestibular nerve became possible. A robust VEMP response is dependent on a healthy inferior branch of the vestibular nerve as well as a healthy saccule. Therefore, VEMP testing can be used to assess the integrity of the inferior branch of the vestibular nerve. Futher, in this clinical population, VEMP testing could possibly assist in determining the location of the vestibular schwannoma.

The VEMP response was used to evaluate the integrity of the inferior branch of the vestibular nerve in vestibular schwannomas patients in a case study reported by Matsuzaki, Murofushi, and Mizuno in 1999. The goal of this study was to obtain a closer look at 33 patients with confirmed acoustic tumors in the presence of normal auditory brainstem responses. Caloric testing was also performed to assess the integrity of the

horizontal semicircular canal and its afferent nerve connection to the superior branch of the vestibular nerve. Out of the 33 patients with confirmed MRI acoustic tumors, 2 of them had a normal auditory brainstem response and caloric results. However, VEMP responses to 95 dBnHL click stimuli were absent on the affected side in both patients. This finding suggested that the tumors were affecting the inferior branch of the vestibular nerve for these individuals. Further, in cases with acoustic tumors, tumor location can be determined by using caloric testing, the auditory brainstem response, and the VEMP response.

Patko, Vidakl, Vibert, Tran Ba Huy, de Waele (2003) investigated VEMP responses obtained to 100 dBnHL clicks and 500 Hz short tone bursts in 170 patients with acoustic schwannomas. The purpose of this study was to investigate which type of stimuli (i.e., tone bursts or clicks) is optimal in determining the presence of a vestibular schwannomas and to assess which VEMP parameters are most affected by the presence of a vestibular schwannomas. The results indicated that 134 out of 170 (78.8%) patients had low or absent VEMP responses to clicks and/or short tone bursts.

Additionally, the latency of p13 was prolonged in 18 out of 170 (10.5%) of the patients but was normal in the remaining 152 (89.5%) participants. Patko and colleagues (2003) also reported that the VEMP response was absent in 118 out of 170 (69.4%) of the patients when recorded to click stimuli. A somewhat different pattern of findings emerged with the 500 Hz tone burst stimuli. Patko and colleagues re-assessed the 118 individuals who had absent VEMP responses to click stimuli. They found that all of them had normal or low amplitude VEMP responses to the 500 Hz tone burst stimuli. These findings suggest that low frequency tonal stimuli were more sensitive than click

stimuli in recording VEMP responses in patients with vestibular schwannomas. Patients with vestibular schwannomas will likely present with abnormally low amplitudes, absent VEMPs, or prolonged latency values for p13.

Ménière's Disease

Ménière's disease is a pathology that affects the inner ear and is due to a dysfunction within the endolymphatic sac. Ménière's is characterized by symptoms that affect the auditory as well as the vestibular system. Symptoms that affect the auditory system include: tinnitus, aural fullness, and a progressive permanent hearing loss. The primary symptom that affects the vestibular system is episodic vertigo. In 1987, two authors, Okuno and Sando, performed a histopathological study on 22 temporal bones of 16 patients with Ménière's disease. They reported that endolymphatic hydrops was more prevalent within the pars inferior (cochlear portion) of the patients' temporal bone. Interestingly, the next most prevalent portion of the temporal bone affected by endolymphatic hydrops was the saccule. Additionally, Okuno and Sando noted that in severe cases or Ménière's disease, endolymphatic fluid occured within the saccule. Specifically, they reported that for 17 out of the 22 temporal bones they examined, there was evidence that the membrane of the saccule swelled into the vestibule and attached to the stapes footplate. The results of this histopathological study suggested that although the cochlear portion of the temporal bone is most commonly affected by endolymphatic hydrops in Ménière's disease, it is important to assess the saccular functioning within these patients (Okuno & Sando, 1987).

In a study performed by de Waele, Tran Bu Huy, Diard, Freyss, and Vidal (1999), 59 patients with a confirmed diagnosis of unilateral Ménière's disease were evaluated using VEMPs recorded to high intensity (100 dBnHL) click stimuli. Results indicated that when the affected ear was stimulated, VEMP responses were absent in 32 out of the 59 (54%) patients. Findings also suggested there was no statistically significant peak-to-peak amplitude or absolute latency differences when compared to amplitude and latency values obtained from the normal control group (de Waele, Tran Bu Huy, Diard, Freyss, & Vidal, 1999).

In 2001, conflicting findings were found by Young, Wu and Wu. Young and colleagues investigated 10 patients with Ménière's disease ranging in age from 15 to 49 years. VEMP responses were present in all of the 10 subjects. Three out of the 10 participants had abnormally large VEMP amplitudes in the pathologic ear. It should be noted by Young et al. (2001) that their results were different from those previously reported by de Waele and colleagues in 1999. This variability between studies was attributed to patient age and the stage of Ménière's disease. Subjects of Young et al. (2001) were younger, and therefore in an earlier stage of Ménière's compared to participants evaluated in the study performed by de Waele et al. (1999). In the former study, the participants had experienced Ménière's disease for a longer period of time. Thus, it was hypothesized that the patients evaluated by de Waele et al. (1999) likely had greater saccular abnormality than those in the Young et al (2001) study due to the longer period of time that endolymphatic hydrops had affected the membrane.

In a study performed by Rauch, Zhou, Kujawa, Guinan & Herman in 2004, the VEMP potential was recorded in 34 patients with unilateral Ménière's disease. Results

showed the frequency tuning of the VEMP response at 500 Hz is altered in those with Ménière's disease. Additionally, the severity of the frequency tuning abnormality increased with the progression of the Ménière's disease. This frequency tuning was also noted in the unaffected ear of the patients. This suggested that the frequency tuning could be an indication of potential development of Ménière's disease on the unaffected side. An additional finding of this study reported an increase in the VEMP threshold for the Ménière's patients (Rauch et al., 2004).

In a subsequent study performed by Timmer in 2006, VEMP responses were obtained from 12 patients diagnosed with Ménière's disease and 12 patients with Ménière's disease who experienced drop attacks (severe cases of vertigo where patients drop to the ground due to the severity of the episode). VEMP responses were acquired using varying tonal frequencies (250, 500, and 1000 Hz).The results indicated that the VEMP response was absent in 41% of the patients with drop attack Ménière's disease, and absent in 13% of the patients with Ménière's disease and no symptoms of drop attacks. Further, there was an observed increase in the VEMP threshold for patients with Ménière's disease. The greatest increase in threshold was noted for patients with Ménière's disease and experienced drop attacks (Timmer et al, 2006).

Goals/Aims of the Study

The primary objectives of this study were: (1) collect normative data for the VEMP response to 90 and 95 dBnHL click stimuli recorded using the IHS Smart EP system; (2) determine the range of VEMP thresholds for these click stimuli in a group of adults with a negative history of vestibular pathology; and (3) since it has been anecdotally reported that the amplitude of the VEMP may decrease as intensity of the click stimulus increased, we were interested in determining if the amplitude of the click evoked VEMP response saturated at high sound pressure levels.

In the current study, click stimuli were used to record the VEMP. VEMP recordings were obtained at a total of six stimulus intensities (i.e., 95, 90, 85, 80, 75 and 70 dBnHL). When the click-evoked VEMP waveforms were collected at supra-threshold levels (i.e., 90 and 95 dBnHL) response measurements were taken for several response indices, including the absolute latency values for p13 and n23, peak to peak amplitude values for wave p13-n23, and interaural asymmetry ratios for amplitudes. These various response indices were assessed in order to determine normative data for use in the Speech and Hearing Clinic at Towson University. Several additional issues were also addressed in the current study. One of these issues was to investigate if any saturation effects were seen in the supra-threshold responses (i.e., did the amplitude of wave p13-n23 decrease as the intensity of the click stimulus was increased from 90 to 95 dBnHL). The second issue that was addressed was to determine the VEMP thresholds across this group of subject. This was accomplished by initially presenting the click stimulus at 70 dBnHL and if the response was absent at this low stimulus intensity then the intensity of the click was increased 5 dB step intervals in order to determine the VEMP threshold.

CHAPTER 3

MATERIALS AND METHODS

Subjects

Sixteen human volunteers (11 females and 5 males) participated in this study. Participants ranged in age from 22 to 28 years (with a mean age of 25 years). In order to be included in this study subjects met the following inclusion criteria: (1) normal middle ear function as defined by middle ear pressure ranging from +100 daPa to -100 daPa and static compliance values ranging from 0.36 to 1.66 ml (Wiley, 1989), (2) present middle ear muscle function in the ipsilateral test condition at 500, 1000, and 2000 Hz for the left and right ears, (3) a negative self-reported vestibular history assessed through use of our vestibular case history form seen in Appendix C. Participants were recruited via word of mouth and posted visual media (seen in Appendix D). The rationale for using acoustic immittance and tympanometry testing in our inclusion criteria was to rule out conductive pathology in any of our subjects. A conductive pathology would potentially decrease the sound pressure level arriving at the inner ear and would likely result in a reduced or less than optimal VEMP response.

Procedures

All testing was conducted in a double walled sound treated booth manufactured by Industrial Acoustics Company. Testing took place in one test session lasting approximately one hour per subject. The test session included audiologic as well as VEMP testing. Each participant was asked to complete our vestibular case history form

before the test session began. Otoscopy was performed first, followed by tympanometry. Next, ipsilateral acoustic reflexes were measured at 500, 1000, and 2000 Hz in the right and left ears. The vast majority (i.e., 14 out of 16 or 88%) of the participants had ipsilateral acoustic reflex thresholds at these three test frequencies bilaterally which were within the 90th percentile for acoustic reflex thresholds established by Gelfand, Schwander, and Silman (1990) for individuals with normal hearing. For the remaining two subjects, both had present ipsilateral acoustic reflexes at all three test frequencies in both ears, however; their acoustic reflex thresholds were slightly elevated at 500, 1000, and 2000 Hz. At these 3 test frequencies their acoustic reflex thresholds were at100 dB.

If subjects met the inclusion criteria of normal tympanograms and had present ipsilateral acoustic reflexes at all 3 test frequencies in both ears, then VEMP testing was conducted. If any subject did not meet the inclusion criteria, he/she did not participate in the present study. All 16 of our subjects met the inclusion criteria described above. All subjects were assigned a random number in order to keep their identities anonymous.

Stimulus

The click stimuli, which were 100 microseconds (μs) in duration, were generated by the Intelligent Hearing System (IHS) Smart Evoked Potential (EP) system. These stimuli were then presented to the participants' ears separately via ER3A insert earphone transducers.

The click stimuli were first presented at two high stimulus intensities (i.e., 90 and 95 dBnHL). Additionally, the click stimuli were presented at several lower stimulus intensities (i.e., 70- 85 dBnHL) in order to determine VEMP thresholds in each ear. In the

IHS system, when click stimuli are presented through ER3A insert earphones, a 0 dBnHL level for these stimuli is equivalent to 32 dB peak SPL. Therefore when the click stimuli were presented at stimulus intensities of 90 and 95 dBnHL, this was equivalent to 122 and 127 dB peak SPL, respectively. The specific protocol for determining VEMP threshold is discussed later in the methods section. The click stimuli were presented at a rate of 5 Hz as suggested by Huang et al (2005).

At the beginning of this study, the click stimuli were calibrated using a Brüel and Kjaer 2209 sound level meter. The actual dB peak SPL values delivered through the insert earphones were within one dB of these peak SPL values. The linearity of the click stimuli were also checked and were also found to be within one dB of the expected values for both insert earphones.

Recording Parameters

In the present study, standard gold cup electrodes were employed to record the VEMP response. The electrode montage implemented was as follows: (1) the active electrodes were placed on the upper 1/3 of both the left and right SCMs for each participant; (2) the ground electrode was placed on the forehead at the Fpz location for each of the participants; and (3) the reference electrode was placed on the participant's sternal notch. All electrode impedances were less than 7 kOhms. The length of the analysis window was 64ms post stimulus onset. The gain was set to a level of 50K and remained stable throughout all the VEMP recordings in the current study. Analog band pass EEG filters were set at 50-1500 Hz. Each trial consisted of 128 sweeps which were

replicated for each test condition. Thus, the total number of sweeps per test condition was 256 sweeps.

During this study each participant's EMG tonic level associated with their SCM contraction were also monitored using the DelSys 2 Channel Bagnoli EMG monitoring system. The EMG was only monitored for the SCM that was being contracted. The EMG data was collected by placing a sensor on both the right and left SCMs. The location of these sensors was below the level of the standard cup electrodes used for the VEMP recording. The montage for the EMG recordings is as follows: (1) the EMG sensor placed on the right SCM, which was designated as channel one on the DelSys EMG system (2) the EMG sensor placed on the left SCM, which was designated as channel two on the DelSys EMG system and (3) the reference electrode was placed on the back of the subject's hand. This sensor montage was used for each subject at all test intensities.

Prior to data collection, the DelSys software version 3.7 was downloaded onto a DELL netbook. An engineer from the DelSys support center was contacted. A basic test protocol was established in order for each subject's right and left SCMs EMG levels to be monitored and recorded separately. Two identical protocols were established with the only difference between the two protocols being the assigned ear. The DelSys recording test protocol used for the present study is as follows: EMG channel one was assigned to pick the data from the sensor on the right SCM. EMG channel two was assigned to pick up the data from the sensor on the left SCM. The gain for each EMG channel was set to 1000. The EMG levels were calculated as Root Mean Square (RMS) values. The length of the recording window on this software was 0.5 seconds.

Patient Positioning and Stimulus Delivery

At the beginning of the test session, each subject was seated in a recliner chair and reclined to the supine position. Each subject was asked to lift and turn their head toward the shoulder opposite the ear that was receiving the click stimuli. For example, if the participant was receiving stimuli in their right ear, they were asked to turn their head toward their left shoulder which elicited a contraction of their right SCM.

All VEMP recordings were conducted using monaural stimulation. When the click stimulus was presented to one ear, the VEMP was simultaneously recorded on two separate recording channels, which will be referred to as the ipsilateral and contralateral recording channels. For example, if the stimulus was delivered to the subject's right ear, then the VEMP recorded from the electrode located on the right SCM is considered/labeled the ipsilateral recording channel. In contrast, the VEMP recording from the electrode located on the left SCM is considered/labeled the contralateral recording channel. Both of these ipsilateral and contralateral recordings associated with monaural stimulation of the right ear are shown on the left side of Figure 24. However, if the stimulus was delivered to the subject's left ear, then the VEMP recording from the electrode located on the left SCM is considered the ipsilateral recording channel, whereas the VEMP recording from the electrode located on the right SCM is considered the contralateral recording channel (as seen on the right side of Figure 10). The sequence of which ear would be the first to receive the VEMP testing was randomized across all 16 test subjects.

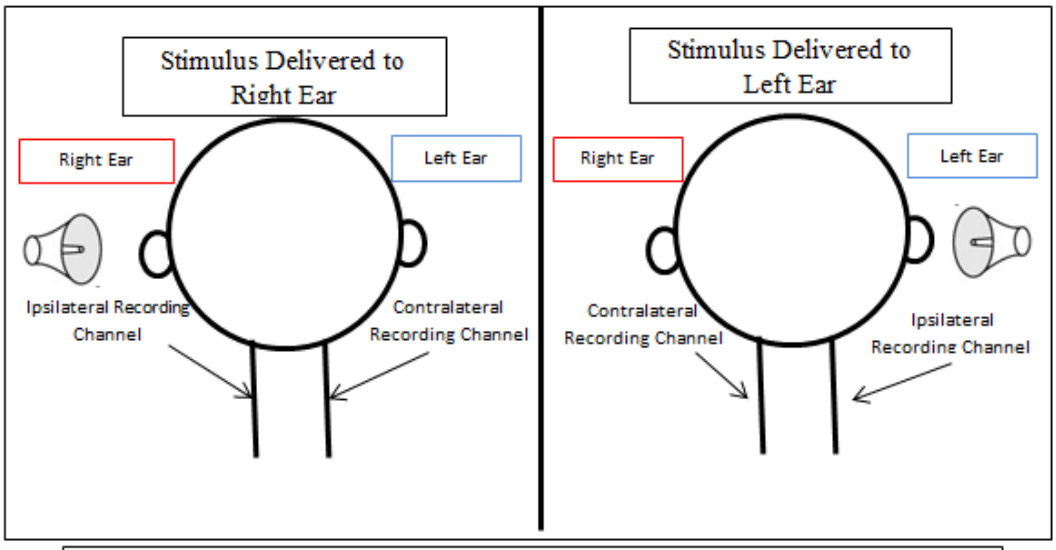

Figure 24: The figure above is an illustration of the two recording conditions employed in this study. On the left side of the figure is an example of the recording channels if stimulus was delivered to the right ear. On the right side of the figure is the recording channel set up if the stimulus was delivered to the left ear.

Pilot Data Related to Developing the EMG Test Protocol

At the beginning of this study, pilot data was collected on 5 individuals to establish test protocol for monitoring EMG tonic level associated at each subject's contracted SCM. Based on Dr. Akin and colleagues' studies, we expected to be able to record robust VEMPs to 90 or 95 dBnHL click stimuli while subjects maintained a constant EMG target level of approximately 50µV (Akin and Murnane 2003; Akin et al. 2004). However, during the current pilot study no observable VEMP waveforms were seen when the subjects maintained an EMG tonic level of 50µV. Therefore, in the present study it was decided that each subject would establish their own EMG target level. This EMG target level was established using the DelSys 2 Channel Bagnoli Handheld EMG monitoring system. Once the EMG target level was established, the current investigators ensured that each subject maintained their established EMG target level throughout all of

the VEMP recordings. A ± 10% deviation from this target was allowed. Additional details regarding how this EMG test protocol was applied is described in the next section.

Actual Test Protocol for Each Test Session

For clarity purposes, this section will be divided into two parts. The first part will address the EMG test protocol. The second part will address the VEMP test protocol. The VEMP response and the EMG recordings were conducted simultaneously for each subject. Two investigators were involved in conducting these VEMP and EMG recordings. The first investigator located on the subject's side of the booth monitored each participant's EMG levels using the DelSys system. The second investigator, on the other side of the booth where the EP equipment was located, conducted the VEMP recording.

EMG Test Protocol

The first step in this EMG test protocol was each subject's EMG target level was established while their VEMP was recorded to a 90 dBnHL click stimuli in their first test ear. In order to establish the participant's EMG target level, the individual was instructed to raise his head, and look toward the shoulder opposite the ear he was receiving the 90 dBnHL click stimulus. The examiner on the patient's side of the test booth would then observe if the ipsilateral SCM was visibly contracted. This examiner would then signal the second examiner to begin the VEMP recording. If that examiner obtained a clear p13 and n23 VEMP waveform components, then that starting EMG level became the subject's "established EMG target level". The examiner on the patient's side of the test booth then wrote down the time, in seconds, at which the VEMP response began and

ended for each trial. This timing information was noted on the DelSys 3.7 software. The timing information was later used to calculate the RMS value of the actual EMG tonic level that occurred during this specific time. This procedure was used for each trial in each subject's ear. We will describe how the EMG data was analyzed later in this section.

As previously mentioned, the subject was asked to maintain his established EMG target level (±10%) for all the subsequent VEMP recordings in each ear. To aid the subjects in achieving and maintaining their established EMG level (± 10%), the subjects were advised to watch a visual monitor on the DelSys screen. The examiner on the patient side would advise the patient to contract his SCM more or less depending on the feedback from the visual monitor. This visual aid was used throughout all the VEMP recordings for all 16 subjects.

VEMP Test Protocol

The first step in the VEMP test protocol was to record the VEMP response at 90 dBnHL in one ear. If a clear VEMP response was obtained, it was replicated and then the stimulus intensity was increased by 5 dBnHL (i.e., 95 dBnHL). Similarly, if a clear response was obtained at 95 dBnHL then the response was replicated. In the present study, VEMPs were recorded at 90 and 95 dBnHL to investigate if any saturation effects occurred on the p13-n23 amplitude. Saturation was defined as a smaller peak-to-peak amplitude value obtained at 95 dBnHL compared to the peak-to-peak amplitude value for p13-n23 obtained at 90 dBnHL. We could not investigate saturation effects at higher sound pressure levels due to the intensity limits for the click stimulus on the IHS system.

After the VEMP response was recorded at 90 and 95 dBnHL in the same ear, then the VEMP was recorded to a 70 dBnHL click. If the VEMP was judged to be absent at 70 dBnHL, then the VEMP was recorded at several higher stimulus intensities. In order to determine the threshold for the VEMP stimulus intensity the click was increased in 5 dB increments. Response absence was defined as no clearly defined p13 and n23 wave components. In contrast, if a VEMP response was judged to be present at 70 dBnHL, then the stimulus intensity was decreased by 5 dB steps until the response was judged to be absent. No participants in this current study had a VEMP waveform at 70 dBnHL. In order to determine VEMP threshold, both of the following criteria had to be met: (1) the lowest level at which clear p13 and n23 waveform components could be identified with a judgment of "no response" occurring at a stimulus intensity of 5 dB lower; and (2) these waveforms were replicable between trials one and two.

If the VEMP response was recording on a subject at 6 stimulus intensities (90, 95, 70, 75, 80, and 85 dBnHL) in both ears, then the total time for the VEMP ranged from 30 to 40 minutes, This time frame included frequent rest breaks between trials and stimulus intensity.

Response Measurements for the VEMP Response

The following response measurements were taken from the averaged VEMP waveform for each test condition: (1) the latency of waves p13 and n23; (2) the peak-to-peak-amplitude of p13-n23; and (3) the asymmetry ratio for amplitude which was calculated based on the formula below:

$$Percent\ VEMP\ Asymmetry = \left| \frac{(\text{Greater side p13}-\text{n23 amplitude}) - (\text{Lesser side p13}-\text{n23 amplitude})}{(\text{Greater side p13}-\text{n23 amplitude}) + (\text{Lesser side p13}-\text{n23 amplitude})} \right| \text{ x } 100$$

Statistical Analysis of the VEMP Response

Descriptive statistics (i.e. mean, standard deviation, median, and range values) as well as a series of one-way analysis of variance (ANOVAs) tests were calculated on the absolute latencies of waves p13 and n23, the peak-to-peak amplitudes of p13-n23, and the VEMP thresholds. Each wave component of the VEMP (i.e., waves p13 and n23) was evaluated separately. Similarly, the ANOVAs were conducted separately on the results at each stimulus intensity. A detailed description of the ANOVA results will be provided in the various sections of the results. In the first set of ANOVAs, we were interested in determining if there was a significant ear effect for these response indices (e.g., were the mean p13 latency values at 90 dBnHL in the right ear significantly different from the mean p13 latency values at 90 dBnHL in the left ear?). A second set of ANOVAs were calculated to determine if there were significant differences in these response measures as a function of stimulus intensity (90 dBnHL versus 95 dBnHL). The alpha level used to indicate statistical significance for all of the one-way ANOVAs was $p \leq 0.05$. A detailed description of the results from the ANOVAs will be provided in the following results section.

DelSys 2 Channel Bagnoil Handheld EMG Monitoring System Analysis

In order for EMG analysis to be performed, RMS values from each replication at 90 and 95 dBnHL for the left and right ears had to be obtained from the DelSys 3.7

Analyze software. RMS values associated with the EMG tonic level recordings were calculated for each trial. Prior to calculating these RMS values, several steps were taken to prepare the data. The following primary steps were suggested by the engineer at DelSys: (1) filter the raw data using a band stop filter which was set to a corner frequency of 55-65. Filtering the IIR was performed in order to filter out the 60 cycle hum introduced into the recording by environmental electrical noise. (2) Setting two separate cursors to encompass the specific time frame which corresponded to the time frame during which the VEMP recording took place. (3) The DelSys software then provided the RMS value for this specific time period for trial one at 90 dBnHL. These three steps were performed for each trial for both the 90 dBnHL response and the 95 dBnHL response in the left and right ears independently.

CHAPTER 4

RESULTS

This results section will be organized based on each response measurement taken from the individual subjects' VEMP waveforms. The first section will address the absolute latency values for wave p13 and wave n23 presented at stimulus intensities of 90 dBnHL and 95 dBnHL. The second section will report on the peak-to-peak amplitude values for p13-n23. Within this section, the amplitude asymmetry ratio will also be discussed. The third section of the results will report findings regarding VEMP thresholds for each of the subjects. The fourth section will examine the presence versus the absence of wave p13 and wave n23 in the ipsilateral versus the contralateral recording channels. The last section of the results will be a preliminary analysis of the results obtained with EMG monitoring system during the actual VEMP recordings. The EMG monitoring system used in this study was the DelSys 2 Channel Handhold Bagnoli EMG monitoring system. The organization of the discussion section will follow the same order as the results section.

Figure 25 below shows the VEMP waveform recorded in the left ear to click stimuli presented at 95 dBnHL from subject number 8047. The subject's data is an example of the waveforms obtained from most subjects in the present study. The bottom waveforms are the two replications at 95 dBnHL. The top waveform is the averaged response of the two replications at 95 dBnHL. From this averaged waveform all

measurement indices were taken. These measurement indices are the latency values of waves p13 and n23, the peak-to-peak amplitude, and the calculation of the amplitude asymmetry ratio.

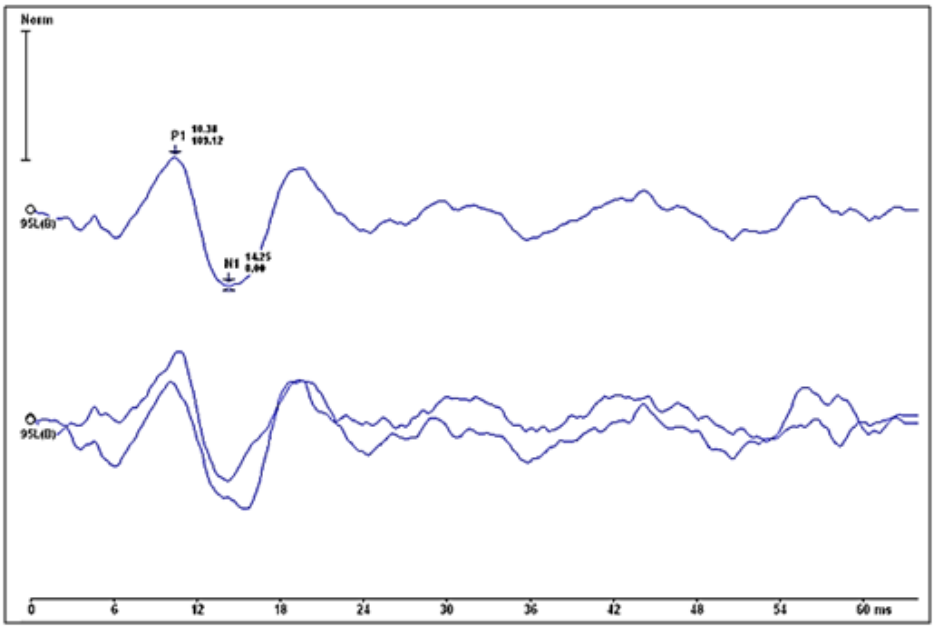

Figure 25 is a depiction of waveforms recorded from subject 8047 at 95 dBnHL to click stimuli. The top waveform is the averaged response from the two bottom replications. Time is measured in ms on the x axis and amplitude is measured in μV on the y axis. Positivity is represented as an upward deflection.

Absolute Latency of Waves p13 and n23

The left hand side of Table 2 displays each subject's (N=16) absolute latency values to click stimuli presented at 95 and 90 dBnHL. The absolute latency values are reported separately for the left and the right ear at each stimulus intensity. In the left ear, the absolute latency values for wave p13 at 95 dBnHL ranged from 9.63 to 13.13ms, with a mean latency value of 11.24ms. Similarly, in the right ear, the latency values at this

same intensity ranged from 9.50 to 13.88ms, with a mean latency values of 10.83ms. At the slightly lower stimulus intensity (i.e., 90 dBnHL), the p13 latency values in the left ear ranged from 8.88 to 15.25ms, with a mean latency value of 11.16ms; whereas the latency values in the right ear ranged from 9.75 to 12.00ms with a mean latency value of 10.89ms. There was no obvious difference in the p13 latency mean values between ears at either stimulus intensity. The variability in the p13 latency data, reflected in the SD values, is equal for the 95 dBnHL response. In contrast, at 90 dBnHL the variability is approximately 3 times greater for the left versus the right ear. Central tendency in the data, reflected in the median values, were essentially similar between ears at both stimulus intensities.

Also depicted on the right side of Table 2, are the individual subjects' latency values for wave n23 at 95 dBnHL and 90 dBnHL. In the left ear at 95 dBnHL, the absolute latency values for wave n23 ranged from 13.38 to 20.75ms, with a mean latency value of 15.99ms. Similarly, in the right ear the n23 latency values at the same intensity ranged from 12.75 to 18.88ms, with a mean latency of 15.33ms. In contrast, at 90 dBnHL, the n23 latency values ranged from 13.25 to 19.38ms for the left ear and from 12.5 to 18ms for the right ear with mean latency values of 15.96ms and 14.51ms, respectively.

Another pattern seen in Table 2 is a decrease in the detectability of the p13 and n23 responses for the higher (95 dBnHL) versus the lower (90 dBnHL) stimulus intensities. Specifically at 95 dBnHL, 15 out of 16 (i.e., 94%) subjects had replicable waves p13 and n23 bilaterally. The one exception to this pattern was subject number 8021 who only had replicable p13 and n23 responses in his/her right ear. In contrast, at

90 dBnHL, 13 out of 16 (i.e., 81%) participants had obtained replicable waves p13 and n23 bilaterally. Of the three remaining subjects (i.e., subject numbers 8021, 2887, and 9979), two individuals had absent responses for p13 and n23 in one ear, and one individual had an absent p13 and n23 response bilaterally. Therefore, these subjects' latency data was removed prior to conducting statistical analysis.

Table 2

ABSOLUITE LATENCY in ms

Subject Number	Wave p13				Wave n23			
	95 dBnHL		90 dBnHL		95 dBnHL		90 dBnHL	
	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear
7166	9.63	9.63	9.38	9.75	13.38	13.88	13.25	13
9652	11.13	9.5	11.63	10.63	17.25	14.88	18.13	14.25
8021	*No Response*	*10.88*	*No Response*	*No Response*	*No Response*	*17*	*No Response*	*No Response*
9956	13.13	12.13	15.25	10.38	15.88	15	17.5	13.63
6036	10.25	10.25	9.75	10.13	14.13	13.25	13.25	15.5
8018	12.88	13.88	11	11.5	16.88	18.5	18.13	15.38
2887	10.63	12.13	12.13	*No Response*	15	16.63	19.38	*No Response*
8047	10.38	10.13	10.25	10.88	14.25	14.5	14.5	14.25
4519	11.13	10.38	9.13	11	16.88	14.5	14.13	14.75
1657	9.63	10.13	9.75	9.75	16	16.5	14.5	15.38
8627	10.25	10.88	10.5	11.75	13.88	13.63	13.88	13.63
5206	11.25	10.88	13.75	11.38	16.5	18.88	19.38	18
9688	10.75	11.88	10.13	10.88	16.5	14.88	16.63	14.5
9979	12.38	9.88	9.63	*No Response*	20.75	16.63	14.13	*No Response*
7614	12.38	10.13	14.75	10.13	16.88	12.75	16.75	12.5
5188	12.75	10.63	8.88	11.38	15.63	15.5	17.5	16.38
Mean	11.24	10.83	11.16	10.89	15.99	15.33	15.96	14.51
SD	1.19	1.19	2.08	0.69	1.81	1.8	2.11	1.51
Median	11.13	10.38	10.25	10.88	16	14.88	16.63	14.25
Range	9.63-13.13	9.50-13.88	8.88-15.25	9.75-12	13.38-20.75	12.75-18.88	13.25-19.38	12.5-18
N	15	16	15	13	15	16	15	13

The table above depicts each individual subject latency values for waves p13 and n23 for 90 dBnHL and 95 dBnHL in the left and the right ears. Subjects from whom no measurable waveform data was available are listed above as "No Response". At the bottom of the table are descriptive statistics for individual subject's latency values for waves p13 and n23 in the left and right ears at 95 dBnHL and 90 dBnHL.

The first one-way ANOVA investigated whether there was a significant difference in latency values of wave p13 for the right versus the left ear at 95dBnHL presentation level. Results of this ANOVA revealed no statistically significant ear effect

for the mean p13 latency values for the right versus the left ear. The mean latency value of 11.24ms for the left ear and 10.83ms for the right ear at 95 dBnHL were not statistically significant [F (1, 29) 0.88; p= .36]. Similarly, the results of the second one-way ANOVA revealed no statistically significant ear effect for p13 latency values for the 90 dBnHL responses [F (1, 25) 0.20; p= 0.66]. The results of the remaining 2 ANOVAs also revealed there were no statistically significant ear effects for wave n23 at 95 dBnHL [F (1, 29) .20; p= 0.33] and at 90 dBnHL [F (1, 25) 4.06; p= 0.06].

Given the fact no statistical significant effects were found for the latency values for either wave p13 and wave n23 at 95 and 90 dBnHL, the latency values of waves p13 and n23 were collapsed across ears by averaging the values for the right and left ears for each wave component separately. This averaging process was performed independently at both stimulus intensities for each ear. This averaged collapsed data is represented in *Table 3* below.

Table 3

Descriptive Statistics
Absolute Latency

	Latency of Wave p13		Latency of Wave n23	
	95 dBnHL	90 dBnHL	95 dBnHL	90 dBnHL
Mean	11.03	11.02	15.66	15.24
Standard Deviation (SD)	1.00	1.07	1.51	1.61
Median	11.07	11.07	15.69	14.94
Range	9.63-13.38	9.57-12.82	13.63-18.69	13.13-18.69
Number of Subjects (N)	15	13	15	13

The above table displays the means, standard deviations, medians, ranges, and N values for the latency values of wave p13 and wave n23. Because no statistically significant difference was found the descriptive statistics above are calculated on the averaged/collapsed data between ears.

As evidenced in *Table 3*, there were no obvious differences between the mean absolute latency values of p13 at 95 dBnHL versus 90 dBnHL (i.e., 11.03ms versus 11.02ms, respectively). Similarly, no obvious differences were noted between the mean absolute latency values for n23 at 95 dBnHL versus 90 dBnHL (15.66ms versus 15.24ms). The variability in the data, reflected in the SD values, was similar for waves p13 and waves n23 at both stimulus intensities. To determine if there was a statistically significant intensity effect for the mean latency values for wave p13, a one-way ANOVA was computed. Results revealed that there was no significant difference in mean latency values for wave p13 at the 95 and 90 dBnHL response [F (1, 25) 0.00; p= 0.96]. A similar one- way ANOVA was calculated for wave n23. Results showed no statistical significant intensity effects for the mean latency values for wave n23 between 95 and 90 dBnHL response [F (1, 25) .06; p= 0.82].

Amplitude

For clarity purposes, this section of the results will be divided into 2 subsections. The first section will discuss the peak-to-peak amplitude value of p13-n23 and the second section will discuss the amplitude asymmetry ratios calculated for the 95 and 90 dBnHL responses.

Peak-to-Peak Amplitude

Illustrated in Table 4 are each subject's peak-to-peak amplitude values in microvolts (µV) for wave p13-n23 in the left and right ears at 95 dBnHL and 90 dBnHL. Descriptive statistics were calculated on these amplitude values and are noted at the bottom of *Table 4*. The peak-to-peak amplitudes ranged from 11.34 to 146.1µV, with a

mean peak-to-peak amplitude value of 41.85 µV for the 95 dBnHL response. Similarly, in the right ear, the peak-to-peak amplitude values at this same intensity ranged from 7.53 to 109.08 µV with a mean peak-to-peak amplitude value of 42.82 µV. At a slightly lower intensity level (i.e., 90 dBnHL), the subjects' peak-to-peak amplitudes ranged from 8.89 to 99.69 µV, with mean peak-to-peak amplitude of 27.96 µV in the left ear and ranged from 4.54 to 81.96 µV, with mean amplitude of 31.42 µV in the right ear. There was no obvious difference in the mean peak-to-peak amplitude values between ears at either stimulus intensity. The variability in the data, reflected in the SD values, was equal between ears for the 90 dBnHL responses, and slightly greater for the left ear responses at 95 dBnHL. The central tendencies, reflected in the median values, were essentially the same between ears at 95 dBnHL and 90 dBnHL.

Also evidenced by Table 4, the mean peak-to-peak amplitude data for p13-n23 shows no saturation effects. A review of the individual subject peak-to-peak data revealed little, if any, direct evidence of a saturation effect in which the amplitude response increased with the increase in stimulus intensity (i.e., VEMP response obtained at 90 dBnHL and 95 dBnHL). For example, for 15 possible responses in the left ear, only one subject (subject number 5206) had slightly smaller p13-n23 amplitude at 95 dBnHL versus 90 dBnHL (13.6 µV versus 11.34 µV, respectively). Similarly, for the 13 possible responses in the right ear, again, only one subject (subject # 9652) had smaller p13-n23 amplitude at 95 dBnHL versus 90 dBnHL (13.22 versus 19.79).

In agreement with the statistical analysis calculated on the absolute latency data, two separate one- way ANOVAs were also performed on the peak-to-peak amplitude data. These two independent one-way ANOVAs were used to investigate if there were

significant ear effects for the p13-n23 amplitudes at each stimulus intensity level. Again, the results at stimulus intensities were evaluated separately. Results of the first one-way ANOVA revealed no statistically significant ear effect for p13-n23 amplitude value at 95 dBnHL [F (1, 29) 0.01; p= 0.95]. The results of the second one-way ANOVA also revealed no statistically significant ear effect for the p13-n23 amplitude at 90 dBnHL, [F (1, 25) .13; p= 0.73].

Table 4

Peak-to-Peak Amplitude Values for Wave p13-n23 in(μV)

	Peak-to-Peak Amplitude 95 dBnHL		Peak-to-Peak Amplitude 90 dBnHL	
	Left Ear	Right Ear	Left Ear	Right Ear
7166	146.1	98.54	99.69	61.94
9652	25.25	13.22	12.83	19.79
8021	*No Response*	76.24	*No Response*	*No Response*
9956	29.64	70.96	26.7	40.88
6036	20.86	31.99	17.79	19.45
8018	16.47	11.95	13.25	4.54
2887	26.63	11.91	15.99	*No Response*
8047	109.12	109.08	45.06	81.96
4519	14.07	34.62	8.89	27.72
1657	40.47	57.25	29.78	26.31
8627	97.87	41.13	44.32	11.23
5206	11.34	25.33	13.6	12.84
9688	43.83	89.85	26.33	70.12
9979	12.28	7.53	7.42	*No Response*
7614	19.73	23.45	15.98	19.7
5188	14.06	15.51	9.32	12.02
Mean	41.85	42.82	27.96	31.42
SD	41.50	34.18	24.70	24.80
Median	25.25	31.99	17.79	19.79
Range	11.34-146.1	7.53-109.08	8.89-99.69	4.54-81.96
N	15	16	15	15

The above table displays each individual subject's amplitude values for 90 dBnHL and 95 dBnHL for the left and the right ears. Subjects from whom no measurable ratio data was available are listed above as "No Response". Subjects' mean standard deviation, median, range and N values for the amplitude values at 90 and 95 dBnHL for the left and right ears are noted at the bottom of the table.

Given that no statistical significance differences in the p13-n23 amplitude values occurred between the ears for either the 95 or 90 dBnHL responses, the peak-to-peak amplitude values were collapsed across ears by averaging the amplitude values for the

left and right ears. This averaged data is displayed in *Table 5 with* descriptive statistics of this data noted at the bottom of the table.

Table 5

Descriptive Statistics

Peak-to-Peak Amplitude

	95 dBnHL	90 dBnHL
Mean	42.33	29.69
Standard Deviation (SD)	35.41	21.91
Median	24.35	18.62
Range	9.91-122.32	8.90-80.82
Number of Subjects (N)	15	13

The table above illustrates the mean, standard deviation, median, minimum to maximum and N values for the latency values of wave p13 and wave n23 from the collapsed amplitude data. Because no statistically significant difference was found between the individual's right and the left ears, the amplitude values for each subject were averaged and the above mean data is calculated from the collapsed data.

In *Table 5* it is evident that mean peak-to-peak amplitude values for the 95 dBnHL response is somewhat larger than the mean amplitude value for 90 dBnHL response (i.e., 42.33 µV versus 29.69µV respectively). However, the variability in the data, reflected in SD values, is considerably greater for the 95 versus 90 dBnHL responses (i.e., 35.41 µV versus 21.91 µV). A one-way ANOVA was calculated to determine if there was a statistically significant intensity effect. Results of the ANOVA showed no statistically significant difference between the mean peak-to-peak amplitude values for the 90 versus 95 dBnHL responses [F (1, 25) 1.20; p= 0.17]. The lack of statistically significant difference in these amplitude values as a function of stimulus intensity is supported by the median values which suggest that the central tendency in the amplitude data is similar at 90 and 95 dBnHL.

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Amplitude Asymmetry Ratio

The next table, Table 6, depicts individual subject's amplitude asymmetry ratio values. The amplitude asymmetry ratios for each subject were calculated using the formula below:

$$Percent\ VEMP\ Asymmetry = \left|\frac{(\text{Greater side p13}-\text{n23 amplitude}) - (\text{Lesser side p13}-\text{n23 amplitude})}{(\text{Greater side p13}-\text{n23 amplitude}) + (\text{Lesser side p13}-\text{n23 amplitude})}\right| \times 100$$

This asymmetry ratio was calculated separately for the response at 95 and 90 dBnHL. For the 95 dBnHL responses the asymmetry ratio could be calculated for 15 subjects. For the 90 dBnHL responses, the asymmetry ratio could be calculated for only 13 subjects.

As seen in Table 6, at 95 dBnHL, the asymmetry ratios ranged from 0% to 42%, with a mean value of 25%. In contrast, at 90 dBnHL, the asymmetry ratios ranged from 3% to 60%, with a mean value of 21%. There is no obvious difference in the mean asymmetry ratios as a function of stimulus intensity. The variability in the data, reflected in the SD values, showed slightly less variability for the 95 dBnHL responses in comparison to the 90 dBnHL response values (i.e., 14% versus 19% respectively). A one-way ANOVA was calculated to compare the asymmetry ratios obtained at 95 dBnHL versus the asymmetry ratios obtained at 90 dBnHL, and determine if there was a statistically significant intensity effect. Results of the ANOVA showed no statistically significant intensity effect [F (1, 25) 0.78; p= 0.78]. The central tendency of the data, reflected in the median, is similar across stimulus intensity level. Additionally, we found that at 95 dBnHL, 80 % (12/15) of our subjects had asymmetry ratios less $\leq$40%. In contrast, at 90 dBnHL, 67% (9/13) of our subjects had amplitude asymmetry ratios $\leq$40 %. As mentioned in the literature review, normative cutoff for asymmetry ratios for individuals with no vestibular pathology is $\leq$40 % (Akin & Murnane, 2008).

Table 6

Amplitude Asymmetry Ratio in Percent

Subject Number	95 dBnHL	90 dBnHL
7166	19%	23%
9652	31%	21%
8021	*Could Not Calculate*	*Could Not Calculate*
9956	41%	21%
6036	21%	4%
8018	16%	49%
2887	38%	*Could Not Calculate*
8047	0%	29%
4519	42%	51%
1657	17%	6%
8627	41%	60%
5206	38%	3%
9688	34%	45%
9979	24%	*Could Not Calculate*
7614	9%	10%
5188	5%	13%
Mean	25%	26%
SD	14%	19%
Median	24%	21%
Range	0%-42%	3%-60%
N	15	13

The above table displays each subject's asymmetry amplitude values for 90 dBnHL and 95 dBnHL. Subjects from whom no calculated ratio data was obtained are listed above as "Could Not Calculate". Subjects mean, standard deviation, median, range and N values for the amplitude asymmetry values are noted at the bottom of the table.

VEMP THRESHOLDS

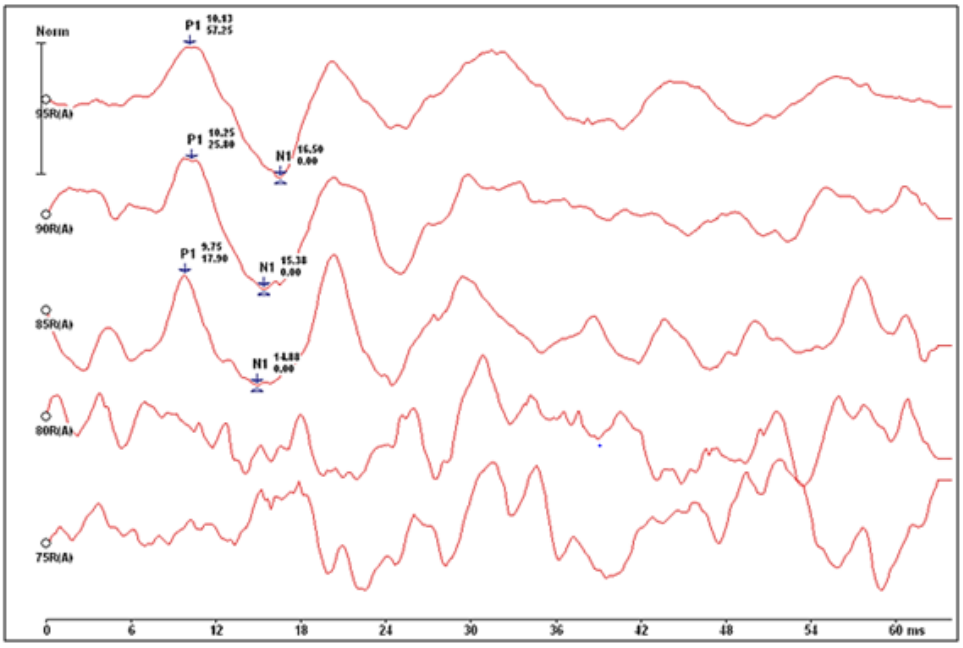


Figure 26 is a depiction of waveforms recorded from subject 1657 at 95, 90, 85, 80 and 75 dBnHL to click stimuli. All waveforms are the averages of the two replications at that given intensity. Threshold was determined to be 85 dBnHL in the right ear for subject 1657 above. Time is measured in ms on the x axis and amplitude is measured in μV on the y axis. Positivity is represented as an upward deflection.

Figure 26, above, is an example of VEMP waveforms obtained from subject number 1657 at various click intensities. The varying intensities for each waveform are noted in a descending order with 95 dBnHL noted as the top waveform and 75 dBnHL as the bottom waveform. As expected, it is noted that as stimulus intensity decreased, the morphology of the VEMP waveform began less distinct. Threshold was defined as the lowest level at which clear replicable p13 and n23 wave components could be noted with

no response recorded at the stimulus intensity 5 dB lower. In subject 1657 VEMP threshold was 85 dBnHL.

Table 7 depicts each subject's VEMP threshold in the left and right ears separately. In the left ear, VEMP thresholds ranged from 75 to 95 dBnHL, with a mean VEMP threshold of 86.88 dBnHL. Similarly, in the right ear, VEMP thresholds ranged from 75-95 dBnHL, with a mean VEMP threshold of 86.33 dBnHL. Mean VEMP thresholds were essentially identical between ears, with the left ear mean thresholds only .55 dBnHL greater than mean thresholds in the right ear. The variability of the VEMP threshold data, reflected in the SD values, is essentially equal for the left and right ears. Moreover, the central tendency in the data, reflected in the median values, was 85 dBnHL bilaterally. To determine if there was a statistically significant difference in VEMP threshold between ears, a one-way ANOVA test was performed. This statistical analysis was performed on 15 subjects' data. Subject number 8021's data was not included in this analysis as he/she had no replicable VEMP responses at any stimulus intensity. As expected, there was no statistically significant difference in VEMP threshold between the left and the right ears [F (1, 28) 0.00; p=1.00].

Table 7

VEMP Thresholds

Subject Number	Left Ear	Right Ear
7166	75	85
9652	90	90
8021	95	*No Response*
9956	85	85
6036	85	85
8018	90	90
2887	85	95
8047	85	80
4519	85	80
1657	85	85
8627	85	85
5206	90	90
9688	80	75
9979	90	95
7614	90	85
5188	95	90
Mean	86.88	86.33
SD	5.12	5.50
Median	85	85
Range	75-95	75-95
N	16	15

The above table depicts each subject's threshold data in the left and right ears. Subjects from whom no measurable waveform data was available are listed above as "No Response". Descriptive statistics are noted at the bottom of the table.

Presence versus Absence of Wave p13 and Wave n23 in the Contralateral versus the Ipsilateral Recording

In the present study, VEMP responses were simultaneously recorded using the ipsilateral and the contralateral recording channels at 90 and 95 dBnHL. *Table 8* is a visual illustration of whether each subject obtained a repeatable VEMP response in the ipsilateral and/or contralateral recording condition at each stimulus intensity. The criteria we used to judge a response in either the ipsilateral or contralateral recording channel present was defined as a repeatable p13 and n23 wave component from which amplitude and latency response measurements could be taken. At the bottom of *Table 8* is a summary of the percentage of subjects with "present responses". These percentages have been reported separately for each ear at 90 and 95 dBnHL.

The primary finding in this study regarding this issue is that ipsilateral recordings have a considerably higher percentage of VEMP responses judged to be present in comparison to the contralateral recordings at both stimulus intensities. This was especially apparent for VEMP responses in the ipsilateral and contralateral channels for 90 dBnHL. For example, the percent rate judged to be present for the ipsilateral recording at 95 dBnHL was 94% (averaged across ears) in comparison to a percent rate of 78.5 % (averaged across ears) in the contralateral recording at this same intensity. Similarly, the percent rate judged to be present for the ipsilateral recording at 90 dBnHL was 87.5% (averaged across ears) versus a rate of 53% (averaged across ears) for the contralateral recording.

Table 8

Presence vs. Absence in the Contralateral and Ipsilateral Recordings

Subject Number	95 dBnHL				90 dBnHL			
	Ipsilateral		Contralateral		Ipsilateral		Contralateral	
	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear	Left Ear	Right Ear
7166	P	P	P	P	P	P	P	P
9652	P	P	P	P	P	P	P	P
8021	A	A	A	A	A	A	A	A
9956	P	P	P	P	P	P	A	P
6036	P	P	P	P	P	P	P	P
8018	P	P	A	P	P	P	A	A
2887	P	P	P	P	P	A	A	A
8047	P	P	P	P	P	P	A	P
4519	P	P	A	P	P	P	A	A
1657	P	P	P	P	P	P	P	P
8627	P	P	P	P	P	P	P	P
5206	P	P	A	P	P	P	A	A
9688	P	P	P	P	P	P	P	P
9979	P	P	P	A	P	A	P	A
7614	P	P	A	P	P	P	A	P
5188	P	P	P	A	P	P	P	A
Number of Present Responses	*94% 15/16*	*94% 15/16*	*69% 11/16*	*88% 14/16*	*94% 15/16*	*81% 13/16*	*50% 8/16*	*56% 9/16*

The above table displays the presence and absence of the response for each subject at 90 dBnHL and 95 dBnHL for the right and the left ear in the contralateral and ipsilateral recording. A criterion for presence of the response is defined as a repeatable p13 and n23 component. Denoted in percent at the bottom of the table is how many subjects obtained a present response.

**P= Presence *A=Absence*

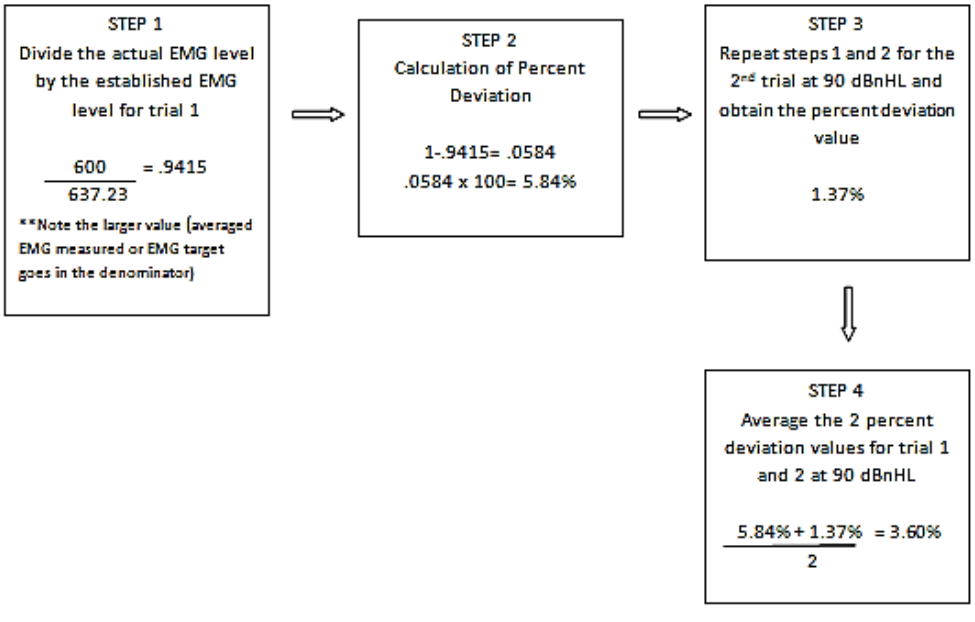
EMG Monitoring System

Depicted in Table 9 below are each subject's established EMG target level and their actual EMG level measured during their VEMP recording. These actual EMG levels were measured separately for each ear trial at both 90 and 95 dBnHL. For example, for subject number 7166, his/her established EMG target was 600. The actual tonic EMG levels measured were 637.23 for the first trial at 90 dBnHL, and 608.31 for the second replication at this same intensity.

Figure 27

Left Ear

		EMG Target	EMG Measured	Deviation (%)	Average Deviation (%)
7166	90	600	637.23	5.84%	
	90	600	608.31	1.37%	3.60%



The Figure above is an example of how percent deviation was calculated for each subject at each intensity level (90 and 95 dBnHL).

Table 9

Percent Deviation from EMG Target

			Left Ear			**Right Ear**		
		EMG Target	Actual EMG Measured	Percent Deviation (%)	Average Deviation (%)	Actual EMG Measured	Percent Deviation (%)	Average Deviation (%)
7166	90	600	637.23	5.84%		565.54	5.74%	
	90	600	608.31	1.37%	3.60%	621.11	3.40%	4.57%
	95	600	590.29	1.62%		572.39	4.60%	
	95	600	589.95	1.68%	1.65%	581.48	3.09%	3.73%
9652	90	750	735.76	1.90%		802.093	6.49%	
	90	750	743.97	0.80%	1.35%	765.59	2.04%	4.27%
	95	750	717.95	4.27%		753.068	0.41%	
	95	750	784.61	4.41%	4.34%	776.908	3.46%	3.72%
8021	90	1250	1183.03	5.36%		1150.78	8.00%	
	90	1250	1176.94	6.00%	5.68%	1201.26	3.90%	5.95%
	95	1250	1380.00	9.42%		1283.89	3.00%	
	95	1250	1190.88	4.73%	7.07%	1332.81	6.21%	3.66%
9956	90	1000	976.75	2.33%		1107.75	9.73%	
	90	1000	953.75	4.63%	3.48%	1010.21	1.01%	5.37%
	95	1000	952.39	4.76%		1021.28	2.08%	
	95	1000	937.65	6.24%	5.50%	981.59	1.84%	3.55%
6036	90	900	865.16	3.87%		942.479	4.51%	
	90	900	835.50	7.17%	5.52%	927.989	3.02%	3.76%
	95	900	898.48	0.17%		829.383	7.85%	
	95	900	873.66	2.93%	1.55%	828.678	7.92%	3.61%
8018	90	400	418.69	4.46%		380.458	4.89%	
	90	400	432.69	7.56%	6.01%	391.349	2.16%	3.52%
	95	400	378.51	5.37%		389.86	2.54%	
	95	400	390.67	2.33%	3.85%	418.018	4.31%	3.41%
2887	90	550	555.24	0.94%		538.022	2.18%	
	90	550	552.68	0.48%	0.71%	523.599	4.80%	3.49%
	95	550	570.97	3.67%		519.176	5.60%	
	95	550	550.40	0.07%	1.87%	527.127	4.16%	3.41%
8047	90	1000	983.01	1.70%		1022.91	2.24%	
	90	1000	972.07	2.79%	2.25%	999.988	0.00%	1.12%
	95	1000	993.14	0.69%		1010.91	1.08%	
	95	1000	996.16	0.38%	0.53%	1011.76	1.16%	3.46%
4519	90	300	318.86	5.92%		312.132	3.89%	
	90	300	312.24	3.92%	4.92%	305.172	1.69%	2.79%
	95	300	305.17	1.69%		311.124	3.58%	
	95	300	312.52	4.01%	2.85%	312.222	3.91%	3.66%
1657	90	750	767.69	2.30%		724.233	3.44%	
	90	750	732.00	2.40%	2.35%	768.1	2.36%	2.90%
	95	750	724.89	3.35%		730.158	2.65%	
	95	750	737.18	1.71%	2.53%	728.234	2.90%	3.71%
8627	90	700	695.13	0.70%		678.069	3.13%	
	90	700	746.04	6.17%	3.43%	685.079	2.13%	2.63%
	95	700	714.34	2.01%		712.735	1.79%	
	95	700	692.43	1.08%	1.54%	734.699	4.72%	3.89%

5206	90	1100	1025.44	6.78%		1089.2	0.98%	
	90	1100	1061.06	3.54%	5.16%	1054.5	4.14%	2.56%
	95	1100	1037.81	5.65%		1036.53	5.77%	
	95	1100	1019.43	7.32%	6.49%	1055.64	4.03%	4.11%
9688	90	600	603.48	0.58%		593.275	1.12%	
	90	600	584.25	2.62%	1.60%	630.328	4.81%	2.97%
	95	600	613.83	2.25%		622.164	3.56%	
	95	600	595.41	0.77%	1.51%	616.531	2.68%	4.17%
9979	90	600	623.52	3.77%		560.718	6.55%	
	90	600	621.93	3.53%	3.65%	559.159	6.81%	6.68%
	95	600	626.24	4.19%		563.893	6.02%	
	95	600	612.77	2.08%	3.14%	566.525	5.58%	3.87%
7614	90	600	652.58	8.06%		580.309	3.28%	
	90	600	575.76	4.04%	6.05%	580.309	3.28%	3.28%
	95	600	567.82	5.36%		592.442	1.26%	
	95	600	580.02	3.33%	4.35%	590.488	1.59%	3.43%
5188	90	600	649.58	7.63%		640.378	6.31%	
	90	600	567.62	5.40%	6.51%	641.93	6.53%	6.42%
	95	600	612.30	2.01%		622.471	3.61%	
	95	600	591.60	1.40%	1.70%	607.67	1.26%	2.44%

The above table displays each subjects EMG target level, actual EMG measured, percent deviation, and the averaged deviation for both trials for the 90 and 95 dBnHL responses in the left and right ear.

Next, the examiners calculated the percent deviation that each individual's actual EMG level differed from their target EMG level. The percent deviation was calculated for each trial by dividing the EMG target level by the actual EMG level in that trial (displayed as step 1 in Figure 27). Then, that value was subtracted from 1 and then converted into a percent by multiplying by 100 (as seen in step 2 in Figure 27). Next, the percent deviation was calculated for the second trial at the same intensity (illustrated in step 3 of Figure 27). Next, the percent deviations for both trials were averaged and this value is referred to as the averaged percent deviation (as noted in step 4 in Figure 27). These same procedures were replicated for each subject for both the 90 and 95 dBnHL responses.

For all subjects (n=16), the averaged percent deviation in the left ear ranged from 1.12% to 6.68% and ranged from 1.94% to7.89 at 90 dBnHL and 95 dBnHL respectively. In the right ear, the averaged percent deviation for all subjects ranged from 0.71% to 6.51% and ranged from 0.53% to 7.07% at 90 dBnHL and 95 respectively. Therefore, all of the subjects were able to maintain a tonic EMG level within ± 10% of their established EMG tonic level.

For all subjects (n=16), the averaged percent deviation in the left ear ranged from 1.12% to 6.68% and ranged from 1.94% to7.89 at 90 dBnHL and 95 dBnHL respectively. In the right ear, the averaged percent deviation for all subjects ranged from 0.71% to 6.51% and ranged from 0.53% to 7.07% at 90 dBnHL and 95 respectively. Therefore, all of the subjects were able to maintain a tonic EMG level within ± 10% of their established EMG tonic level.

CHAPTER 5

DISCUSSION

The primary goal of this study was to collect normative VEMP waveform data for the Speech, Language, and Hearing Center at Towson University. Currently, in clinical settings the most widely used diagnostic tool to assess the peripheral and central vestibular systems are the Electronystagmography (ENG)/ Videonystagmography (VNG) tests. If clinicians rely solely on the caloric portion of the ENG/ VNG tests to assess the peripheral vestibular system, they are limiting their results to the status of horizontal semicircular canals. The peripheral vestibular system has several additional sensory structures which are not being clinically assessed via this ENG/VNG test protocol. Adding the VEMP test to a clinician's vestibular test battery would allow for the assessment of the integrity of the saccule and the posterior branch of the vestibular nerve.

The VEMP is a relatively new diagnostic tool and has been used in the clinical population since the mid-1990s. Some clinicians have incorporated this test into their vestibular test battery without a sound understanding of the influence that various stimuli and recording parameters may have on the absolute latency values of waves p13 and n23, the peak-to-peak amplitude values of wave p13-n23, and VEMP threshold. This situation may lead to an inaccurate diagnosis of the vestibular pathology and appropriate referrals may not occur. In the current study, we have collected normative data for click evoked VEMP responses at presentation levels of 90 and 95 dBnHL in adults with a negative self-reported history of vestibular pathology. The investigators of the present study have also determined VEMP thresholds for these individuals and have investigated the presence versus absence of the response in the ipsilateral and contralateral recording

channels. Lastly, we conducted a preliminary investigation of the EMG tonic levels that occur during SCM contraction using the DelSys EMG monitoring system.

The organization of this discussion will follow the organization of the previous results section. There will be an additional section discussing limitations of the current study which include establishing and monitoring EMG tonic levels during VEMP recording in a clinical setting and directions for future research.

Absolute Latency of Waves p13 and n23

The results of the current study revealed that our mean p13 latency values, collapsed across ears, were 11.03ms (±1.0) for the 95 dBnHL responses and 11.02ms (±1.07) for the 90 dBnHL responses. These current p13 latency findings are in harmony with Colebatch et al. (1994) who reported a mean p13 latency value of 13.3ms (±1.5) for click stimuli presented at 95 dBnHL. Similarly, Murofushi et al. (2001) reported a mean p13 latency value of 11.8ms (±0.86) to click stimuli presented at 95 dBnHL. Collectively, the results of these two studies are in firm agreement with those mean p13 latency values obtained from the present study.

In the current study, the mean n23 latency values, collapsed across ears, were 15.66ms (±1.51) for the 95 dBnHL response and 15.24ms (±1.61) for the 90 dBnHL response. Our current mean n23 latency value is slightly shorter than the mean n23 latency values of 22.6ms (±2.4) reported by Colebatch et al. (1994) and of 20.8 (±2.2) reported by Murofushi et al to 95 dBnHL click stimuli. However, if we take the variability of the data into account, reflected in the SD values, our mean n23 latency data at 95 dBnHL is in relatively good correlation with the mean latency values reported by

Murofushi et al. (2001) for that same stimulus intensity. Both of these studies used a similar number of subjects (i.e., 16 individuals in the current study verses 18 in the Murofushi et al. study (2001)). Therefore, the variability in the data is spread across an approximately equal number of subjects.

Another interesting pattern noted in the current study was that the response detection rate for the VEMP was lower at the higher stimulus intensities. Specifically, the response detection rate decreased from 94% for the 95 dBnHL responses to 81% for the 90 dBnHL responses. In the current study, both waves p13 and n23 had to be judged as present at a particular stimulus intensity in order for the VEMP response to be judged as a present response. This decrease in the response detection rates, as a function of stimulus intensity, was similar to the findings reported by Akin et al. (2003). Specifically, these investigators reported that their responses' detection rate for the VEMP decreased from 92% to 53% as the intensity of the click stimulus was lowered from 95dBnHL to 90 dBnHL.

Amplitude

For clarity purposes, this section on response amplitudes will be divided into 2 sub-sections. The first sub-section will discuss the peak-to-peak amplitude values of waves p13-n23 and the second sub-section will discuss the amplitude asymmetry ratios.

Peak-to-Peak Amplitude

In the present study, the mean peak-to-peak amplitude values, collapsed across ears, were 42.33μV when click stimuli were presented at a level of 95 dBnHL and 29.69μV when click stimuli were presented at 90 dBnHL. A broad range of amplitude

values were obtained for both the 95 dBnHL and 90 dBnHL responses in this present study. Specifically, these amplitudes ranged from 9.91 to 122.32µV for the 95 dBnHL response and from 8.90 to 80.82µV for the 90 dBnHL response. Our current peak-to-peak amplitudes for wave p13-n23 are similar to the broad ranges reported by investigators of the VEMP response. Authors obtained amplitude values ranging from 18.8-137.1 µV (Colebatch et al. 1994) and approximately 20-131 µV (Akin and Murnane 2003) to 95 dBnHL click stimuli.

The mean peak-to-peak amplitude value for waves p13-n23 recorded to the 95 dBnHL click stimuli in the current study is similar to the mean peak-to-peak amplitude value of approximately 40µV reported by Akin and Murnane (2003) to click stimuli presented at the same intensity level. In contrast, the mean amplitude value for waves p13-n23 for the 95 dBnHL click stimuli reported by Colebatch et al (1994) is twice the magnitude of that obtained from the current study (i.e., 85.3µV versus 42.33µV, respectively). The difference in these mean peak-to-peak amplitude values, across studies for the 95 dBnHL response, is likely, or at least in part, due to the large range of amplitude values seen across studies (i.e., 9.91-122.32µV in the current study, 18.8-137.1µV in Colebatch et al. (1994) study, and 20-131µV in the Akin and Murnane (2003) study)

In the current study, we also investigated whether there was any decrease in the response amplitudes of waves p13-n23 (i.e., a saturation effect) as the intensity of the click stimulus was increased from 90 to 95 dBnHL. There was no evidence of any saturation effects in the individual data for the vast majority of subjects. Specifically, 15 out of 16 individuals had larger peak-to-peak amplitude values at the higher versus lower

stimulus intensities (as seen in Table 4). Similarly, our mean amplitude values in the current study revealed a growth in amplitude as the stimulus intensity was increased (i.e., the mean peak-to-peak amplitude value was 29.29μV for the 90 dBnHL response in comparison to 42.33μV for the 95 dBnHL response. It is noted that our reader needs to realize that we could not thoroughly investigate all possible saturation effects because 95 dBnHL was the highest stimulus intensity to which click stimuli could be presented using the IHS Evoked Potential System. This might be an area for future researchers. However, they would need to be concerned regarding safety issues of delivering extremely high sound levels (i.e., ≥ 100 dBnHL) into the inner ear of an adult.

Amplitude Asymmetry Ratio

As discussed in the previous sub-section, our peak-to-peak amplitude values for waves p13-n23 were quite broad. To account for this broad range of amplitude values, many clinical practices calculate an amplitude asymmetry ratio value. In calculating this asymmetry ratio, the individual acts as his own control by comparing the amplitude values for wave p13-n23 obtained from the left SCM versus the amplitude values obtained from the right SCM (Akin and Murnane, 2008; Rauch 2006). In the present study, minimal differences were seen in the mean amplitude asymmetry ratio values as a function of stimulus intensity (i.e., 25% for the 95 dBnHL responses and 26% for the 90 dBnHL responses). The range of these asymmetry ratios in the current study were from 0-40% for the 95 dBnHL response and from 3-60% for the 90 dBnHL response.

Our current mean amplitude asymmetry ratio values are similar to those reported in the earlier literature which calculated these asymmetry ratios on VEMPs recorded to

high intensity click stimulus (95-100 dBnHL). Specifically, Li et al. (1999) reported a mean amplitude asymmetry ratio value of 22.8%. Brantberg and Frannson (2001) and Welgampola and Colebatch (2001) both reported mean amplitude asymmetry ratios values of 22%. Similarly, the range of the amplitude asymmetry ratios in our study were well aligned with those reported in these earlier studies (i.e., 0-37% for Li et al. (1999) study; 8-30% for Brantberg and Frannson (2001) study; and 0-35% Welgampola and Colebatch (2001) study).

In their review of the VEMP literature, Akin and Murnane (2008) suggested using a conservative normative cutoff value of $\leq$40 % to determine that the VEMP asymmetry ratio for amplitude was within normal limits. In the present study, if we apply this normative cut off value of $\leq$40 % to our subject's asymmetry ratio values at each stimulus intensity, 12 out of 15 (80%) of our subjects would have values considered within normal limits for their 95 dBnHL responses and 9/13 (70%) of our subjects would have normal asymmetry ratio values for the 90 dBnHL response. For the remaining subjects whose asymmetry ratio values did not meet this normal criterion, several of the subjects had values very close to this normative cutoff (as seen in table 6).

VEMP Threshold

In the present study, the mean VEMP threshold occurred at 86.88 dBnHL in the left ear and at 86.33 dBnHL in the right ear. These mean threshold values obtained in the present study are essentially identical to the mean VEMP threshold values to click stimuli reported by Colebatch et al. (1994) and Ochi et al. (2001) which were 86 dBnHL and 87.8 dBnHL, respectively.

Two additional VEMP studies performed by Welgampola and Colebatch (2001) and Akin et al. (2003) reported slightly higher mean VEMP threshold values (i.e., 89.6 dBnHL and 91 dBnHL, respectively) in comparison to these reported in the current study. The table below summarizes the mean VEMP threshold and the range of threshold values reported in these earlier studies as well as in the current study. Even though there are slight differences reflected across these studies, these differences are not of clinical relevance.

Table 10

VEMP Thresholds in dBnHL

	Range of Ages (N)	Stimulus Type and Intensity	Range VEMP Thresholds	Mean Threshold (SD)
Colebatch et al., (1994)	29-63yrs (10)	Click @ 95	75-85	86
Akin et al., (2003)	22-51yrs (29)	Click @ 100	80-100	91 (±5.2)
Ochi et al., (2001)	21-38yrs (18)	Click @ intensities 95	80-95	87.8 (±4.54)
Welgampola & Colebatch (2001)	25-85yrs (70)	Click @ 100	75-100	89.6 (±6.9)
Present Study	22-28 (16)	Click @ 95	75-95	86.88 (±5.1) Left Ear
				86.33 (±5.5) Right Ear

The table above compares VEMP threshold data obtained in the various studies within the VEMP literature to VEMP threshold data recording from the present study. Denoted above is the range of ages in each study, the stimulus type and intensity used in each study, the range of VEMP thresholds obtained, and the mean VEMP threshold with the standard deviations noted in parenthesis.

Presence versus Absence of Wave p13 and Wave n23 in the Contralateral versus the Ipsilateral Recording

In the current study, the VEMP response at 95 dBnHL was judged to be present 94% of the time in the ipsilateral recording channel and 78.5 % of the time in the contralateral channel. However at the lower stimulus intensity (i.e., 90 dBnHL) there was a bigger difference in the response presence rates between ipsilateral and contralateral (87.5% versus 53%, respectively). These findings at both stimulus intensities in the present study do not correlate with results from Murofushi et al. (2004). These investigators reported that 100% of their subjects (n=21) had present VEMP response for the ipsilateral recording channels to 95 dBnHL click stimuli. However, only 1 out of 21 individuals, or 0.04%, had present VEMP responses in the ipsilateral recording channel at this same stimulus intensity.

These findings of a huge difference in response presence for the ipsilateral versus the contralateral recording channels reported by Murofushi et al (2004) are supported by neurophysiologic findings in cats reported by Kushiro et al. (1999). Specifically, Kushiro et al. (1999) described that the reflex pathway which connects the saccule to the motorneurons of the SCM is primarily an ipsilateral dominant pathway with little, if any, influence from the contralateral motorneurons of the SCM. The results from the present study also show a higher rate of responses judged to be present in the ipsilateral versus the contralateral recording channels. However, we obtained a much higher response present rate in the contralateral channels then that reported by Murofushi and colleagues (2004).

There are two possible explanations that could explain this higher presence of the VEMP responses seen in the contralateral channel in the current study. One explanation is related to the positioning of the reference electrode for the VEMP recording. Li et al. (1999) recorded their VEMP responses using the location of the reference electrode placed on the wrists of the subjects. These investigators reported that if the reference electrode is placed on the sternum of the subjects, as was the case in the present study, then VEMP waveforms would be seen more often on the contralateral recording channel. When the reference electrode is placed on the sternum, it is located relatively close to both the ipsilateral and contralateral SCMs. If this ipsilateral SCM is contracted, as it would be during VEMP testing, this muscle activity may be seen in the contralateral SCM site. This possibly increases the likelihood of seeing VEMP activity in the contralateral recording.

Another explanation for higher incidence of VEMP responses judged to be present in the contralateral channel was investigated by Colebatch and Rothwell (2004). These investigators observed a short excitation of firing of the motorneurons of the contralateral SCM when recording the VEMP response to click stimuli.

EMG Monitoring System

Several investigators in the literature have reported that the amplitude of the VEMP response is dependent on the tonic EMG level that occurs in the ipsilateral SCM during muscle contraction (Bickford et al., 1964; Colebatch et al., 2001; Colebatch et al., 1994; Lim et al., 1995). Therefore, it appears that if the level of the EMG tonic level is

considerably stronger on one side of the neck in comparison to the opposite side of the neck, then the amplitude asymmetry ratio would be negatively affected.

Akin et al. (2003) as well as Colebatch et al. (1994) have reported that they have specifically recorded robust VEMP response while the subjects' maintained tonic EMG levels of approximately 50μV. The EMG tonic levels in these studies were measured using a visual feedback monitor on the DelSys EMG system in the Akin et al. (2003) study and using the Medelec Sensor EMG monitoring system in the Colebatch et al. (1994) study. As previously mentioned, in the current study we initially attempted to record VEMP responses while having our subjects maintain a tonic EMG level of approximately 50μV as suggested by these earlier investigators. However, in our pilot data we found that it was impossible to obtain identifiable VEMP waveforms using an EMG RMS value of 50μV. Additionally, it was visually observed by the examiners that when subjects did achieve a EMG tonic level of 50μV, they were barely contracting their SCM. Therefore, a decision was made that each subject would establish his own EMG tonic level at the beginning of the test session while their VEMP response was being recorded to a 90 dBnHL click stimulus in one ear. The subject was then asked to maintain this established target level ($\pm$ 10) for all the subsequent VEMP recordings. The subject watched a visual feedback monitor on the DelSys to achieve this goal.

In the present study, our target EMG levels ranged from 300μV to 1250μV, which was 7 to 25 times greater than the EMG target levels reported by Akin and colleagues. In the present study we also measured the actual EMG levels that occurred for each trial (trial 1 and trial 2) for both the 90 and 95 dBnHL response. These measurements were taken separately while the stimuli were delivered to the subjects'

right and left ears. The RMS mean calculations from the actual trials revealed that all subjects were able to maintain a stable tonic contraction level (i.e., $\leq$ 10% of their established target level) for the VEMP recordings across all test conditions. Therefore, based on this RMS data, we can assume that the amplitude asymmetry ratios that were calculated were not negatively influenced by a difference in the strength/magnitude of the EMG tonic level established on each side of the neck that took place during the VEMP recordings.

Optimal Choice of Stimuli for Recording the VEMP

The data collected in this present study represents approximately 50% of the entire normative VEMP data collected for use in the Towson University Speech, Language, and Hearing Center. In the entire study, VEMP responses were recorded to both click and to 500 Hz tone burst stimuli. The click evoked VEMPs were recorded from 16 adults who all had a negative self-reported history of vestibular pathology. Similarly, the VEMPs to 500 Hz tone burst stimuli were recorded from a separate group of 18 adults who also had a negative self-reported history of vestibular pathology. VEMP responses were recorded at two supra-threshold levels (i.e., 90 and 95 dBnHL) and at several lower stimulus intensities (i.e., 70-85 dBnHL) for both the click and 500 Hz tone burst stimuli.

The data obtained from the VEMP responses to both types of stimuli suggested that the optimal type of stimulus to utilize in recording VEMP responses was the 500 Hz tone burst. Two pieces of evidence support this finding. First, the VEMP responses for the 95 dBnHL 500 Hz tone burst in the ipsilateral recording channel were judged to be

present at a slightly higher rate (i.e., 100 dBnHL) in comparison to the 94% detectability rate for the click stimuli at this same presentation level. Second, the overall morphology of the VEMP was clearly better for the responses to the 500 Hz tone burst stimuli in comparison to the responses to the click stimuli. This finding was true at both supra threshold levels.

Limitations of the Study, Clinical Relevance of these Findings, and Directions for Future Research

One of the primary limitations of this study was associated with the DelSys EMG monitoring system. During each phase of the present study, the DelSys EMG monitoring system presented a new set of challenges.

The first problem encountered was during the experimental design process. It was determined during this time that in order to monitor the tonic EMG levels within each subject, additional equipment would need to be obtained. This equipment, the DelSys EMG monitoring system, required that an extra examiner sit on the patient's side of the test booth. This examiner was responsible for visually monitoring the patient's contracted SCMs, monitoring the visual feedback screen, and recording the time frames in which the simultaneous VEMP recording was being performed.

The next set of challenges occurred during data collection and data analysis. Before each test session, the DelSys system would have to be set up. This set up entailed connecting numerous cables between the portable laptop and the two parts of the EMG DelSys (i.e, USB 6009 and the Bagnoli handheld device). Also, it was difficult for the EMG sensors that were placed below the standard cup electrodes to adhere to the

subject's skin. Therefore, tape was placed over the entire EMG sensor pad. Moreover, if the subject had a relatively thin SCM, during SCM contraction the EMG sensor pad would detach from the skin. This would then require the examiner on the patient side of the test booth to reattach the sensor and apply more tape.

During data analysis to obtain the actual RMS values, a series of time consuming steps had to be performed. The data had to be scaled, adjusted, filtered, and the means had to be removed for each trial (i.e., trial 1 and trial 2) for both the 95 and 90 dBnHL responses for each participant's data. Obtaining the actual RMS value from the DelSys software took approximately 30 minutes for each subject in the present study.

Realistically, in a clinical setting, where time allotted to each patient is limited, it would not be feasible to execute the time consuming steps previously discussed. It is also noted that at the present time, we do not have evidence as to whether there would be any difference in the VEMP normative data if examiners used as strict a time consuming EMG monitoring protocol, as performed in the current study. Additionally, it is unknown if more commonly accepted methods that are being used clinically yield similar results to those obtained using a strict EMG monitoring protocol. Examples of clinical acceptable methods are subjects squeezing a tennis ball between their chin and right and left shoulders separately or clinicians' visually observing contraction of their patient's SCMs. Further research is needed to investigate the optimal method in eliciting this contraction and how this monitoring strategy may or may not affect the VEMP response indices.

It would also be insightful to further investigate saturation effects. Due to the limits of the IHS Smart EP system, the stimulus intensity could not be increased any

further than 95 dBnHL. Literature has stated that stimulus intensity is proportional to VEMP amplitude (Colebatch et al, 1994). Colebatch and colleagues (1994) reported that VEMP amplitude at 100 dBnHL was 36% larger than VEMP amplitude obtained at 95 dBnHL. Therefore, it is uncertain if this substantial increase in VEMP amplitude is seen for every 5 dB increase in intensity or if the response does finally plateau. Further research would be beneficial to investigate such saturation effects.

Although all attempts were made to control for confounding subject factors it is important to note that results and statistical analysis were obtained on a relatively small sample size which limits how much we can generalize to an entire population. Additionally, it is important to note that a disproportionate amount of females to male subjects were tested in this study.

Finally, it should be noted that the click-evoked VEMP normative data recorded in the present study can only be applied to a young adult clinical population with healthy vestibular systems. Several investigators have demonstrated that the physiology of the vestibular system is adversely affected by increasing age (Rosenhall, 1973; Bergstrom, 1973; Richter, 1980). Zapala and Brey (2004) have also reported that increasing age has a negative effect on various response indices of the VEMP. Specifically, these investigators stated that the absolute latency values of waves p13 and n23 increase as a function of age and the peak-to-peak amplitude values for waves p13-n23 decrease as a function of age. Zapala and Brey also noted that muscle fatigue related to contraction of the SCM was more common in their elderly subjects in comparison to their younger subjects who participated in their study. Based on these age-related changes in both the physiology of the vestibular system as well as in the VEMP response, the current

investigators suggest that normative data for the VEMP be collected for the older clinical population. Future studies can investigate whether normative data is needed on a decade by decade basis for clinical populations above approximately 50-60 years of age, or if this normative can be generally applied to the clinical population greater than 60 years of age.

Summary

In the present study VEMP responses to click stimuli were recorded in 16 subjects with self-reported normal vestibular functioning. The focus of the current study was to obtain normative data for the VEMP to use in the Speech, Language, and Hearing Center at Towson University. Table 11below provides a summary of the various stimulus and recording parameters used to collect this normative data. All of the VEMP testing was conducted using the IHS Smart EP system.

Table 11

Suggested VEMP Stimulus and Recording Parameters

Stimulus Parameters			
	Stimulus Type	Click	
	Rate	5 Hz	
	Duration	100 µs	
	Intensity	Suprathreshold 95 and 90 dBnHL (122 and 127 dB peak SPL)	Threshold Search 70-85 dBnHL (102-117 dB peak SPL)
	Transducer	ER3A Insert earphones	
	Stimulation	Monaural	
Recording Parameters			
	Recording Channels	Ipsilateral Channel	Contralateral Channel
	Electrode Montage	Left Ear • Noninverting electrode on upper 1/3 of left SCM • Inverting electrode on sternal notch • Ground electrode on forehead (Fpz)	Right Ear • Noninverting electrode on upper 1/3 of right SCM • Inverting electrode on sternal notch • Ground electrode on forehead (Fpz)
	Amplifier Gain	50K x	
	Band pass filter settings	50-1500 Hz	
	Number of Sweeps	128	
	Post stimulus Analysis Window	64 msec	
Table 11 above displays the various VEMP stimulus and recording parameters used in the present study.			

Table 12 below provides descriptive statistic values (i.e., mean, standard deviation, and ranges values) for all response indices of the click-evoked VEMP responses. These response indices include: the absolute latencies for waves p13 and waves n23, the peak-to-peak amplitudes for p13-n23, interaural amplitude asymmetry ratios, and threshold values for VEMPs recorded to 90 and 95 dBnHL (122 and 127 dB peak SPL, respectively) click stimuli. This summary chart is intended to be used as normative VEMP data in our new vestibular clinic.

Summary of Values from Various VEMP Response Indices to Click Stimuli

Table 12

	Absolute Latency (ms)				Peak-to-Peak Amplitude (μV)		Amplitude Asymmetry Ratio (%)		VEMP Threshold (dBnHL)	
	p13		n23							
	95 dBnHL	90 dBnHL	95 dBnHL	90 dBnHL	95 dBnHL	90dBnHL	95 dBnHL	90 dBnHL	Left Ear	RightEar
Mean	11.03	11.02	15.66	15.24	42.33	29.69	25%	26%	86.88	86.33
Standard Deviation	1.0	1.07	1.51	1.61	35.41	21.91	14%	19%	5.12	5.50
Range	9.63-13.88	8.88-15.25	12.75-20.75	12.50-19.38	7.53-146.1	4.54-99.69	0-42%	3-60%	75-95	75-95

Table 12 is a summary of the mean, standard deviation values, and ranges obtained from all the measurement response indices (i.e., absolute latencies, peak-to-peak amplitudes, amplitude asymmetry ratios, and threshold values) to click stimuli.

- Based on our data for amplitude asymmetry ratios, we are suggesting using a criteria of ≤ 40% to indicate normal interaural amplitude differences.
- In the current study, no normal control subject had a VEMP response to click stimuli present at 70 dBnHL. As a result we expect that individuals who have vestibular pathologies affecting their VEMP threshold, such as SCD, would likely have VEMP thresholds ≤70 dBnHL (102 dB peak SPL).

The VEMP test has many potential benefits as a diagnostic tool in the assessment of a patient's vestibular system. The VEMP provides information regarding the integrity of the saccule and the posterior branch of the vestibular nerve. Several issues, however, need to be further researched in order for this clinical tool to reach the popularity of the ENG/VNG. Further educational programs are needed to describe the optimal stimulus and recording parameters for this response. Secondly, it is important that clinicians have a thorough understanding of the various factors that may affect the successful recording of this response. Lastly, it is also recommended that clinicians

obtain their own normative data for the various response indices of the VEMP and then compare their normative data to that reported in the existing VEMP literature.

APPENDICES

APPENDIX A

IRB Approval Letter

TOWSON UNIVERSITY

APPROVAL NUMBER: 12-A025

To: Lauren McGrath
8E Misty Wood Circle
Timonium MD 21093

From: Institutional Review Board for the Protection of Human Subjects Steven Mogge, Member

Date: Friday, December 16, 2011

RE: Application for Approval of Research Involving the Use of Human Participants

APPROVED DEC 16 2011 TOWSON IRB

Office of University Research Services

Towson University
8000 York Road
Towson, MD 21252-0001

t. 410 704-2236
f. 410 704-4494

Thank you for submitting an Application for Approval of Research Involving the Use of Human Participants to the Institutional Review Board for the Protection of Human Participants (IRB) at Towson University. The IRB hereby approves your proposal titled:

Normative Study on Vestibular Evoked Myogenic Potentials (VEMPS)

If you should encounter any new risks, reactions, or injuries while conducting your research, please notify the IRB. Should your research extend beyond one year in duration, or should there be substantive changes in your research protocol, you will need to submit another application for approval at that time.
We wish you every success in your research project. If you have any questions, please call me at (410) 704-2236.

CC: Dr. Margaret Korczak
File

APPENDIX B

Informed Consent Form

PRINCIPAL INVESTIGATOR: **Lauren McGrath and Sasha Phillips**PHONE: **Lauren: (856) 904-0331**

Sasha: (301) 523-0946

Purpose of the Study:

This study is designed to gather normative data on the Vestibular Evoked Myogenic Potential (VEMP) assessment for the Towson University Speech-Language and Hearing Center (SLHC at TU). The VEMP is a diagnostic tool used to assess of one of the sensory organs in the inner ear, the saccule, which contributes to balance and equilibrium. Currently, the SLHC at TU offers one diagnostic tool to assess balance and equilibrium, the ENG. This test provides audiologists with information regarding the integrity of the semicircular canals (SCCs). These SCCs provides neural information to the brain regarding rotations of the head/body from one side of the body to the other side of the body. As a center, we would like to be able to offer an additional diagnostic assessment for our clients with dizziness or equilibrium complaints. Thus, there is a need for a normative study on VEMPs.

Procedures:

Participants will undergo a screening procedure to determine eligibility to be included in a group of normal participants. Prior to the test session, interested participants will complete a case history questionnaire to ensure that they have a negative history of dizziness and equilibrium or have had no ear related surgery. This screening procedure will take place at the beginning of the participant's test session and will include the establishment of normal middle ear function. After eligibility criteria have been met, VEMP testing will commence.

Set up for VEMP testing consists of electrodes being secured to both sides of the participant's neck, their forehead, and the upper surface of their chest (i.e., sternum). The patient is then instructed, during each recording, to turn their head to the side to contract the sternocleidomastoid (SCM) muscle, located on the side of their neck. Acoustic stimuli will be presented to the participant's ears, using insert earphones. The response will be measured from the contracted SCM muscle and VEMP recording will be taken from each side of the neck. Approximately six stimulus intensities will be used when testing each side of the neck. Each of these responses will be replicated. It is expected that the entire test session should take approximately one hour.

Risks/Discomfort:

There are no known risks associated with participation in the study. Should the higher stimulus intensities become uncomfortable at any time during VEMP recordings, the testing will be terminated immediately.

Benefits:

Collecting normative data for the VEMP assessment will allow clinicians at the SLHC at TU to accurately evaluate patients with complaints of balance disorders or dizziness. In order to administer best clinical practice in diagnosing this patient population, VEMP results must be compared to the findings from individuals with normal balance function.

Alternatives to Participation:

Participation in this study is voluntary. You are free to withdraw or discontinue participation at any time.

Cost Compensation:

Participation in this study will involve no costs or payments to you.

Confidentiality:

All information collected during the study period will be kept strictly confidential. The only information that will be collected from each participant will be name, age, and gender. This information will be kept in a password protected document, which only the principal investigators and faculty sponsor will have access to. Each participant will be identified through identification numbers. No publications or reports from this project will include identifying information on any participant. If you agree to join this study, please sign your name below.

_____ I have read and understood the information on this form.

_____ I have had the information on this form explained to me.

_____ _____

Subject's Signature Date

_____ _____

Witness to Consent Procedures Date

_____ _____

Principal Investigator Date

If you have any questions regarding this study please contact Dr. Margaret Korczak, the faculty sponsor of this study and an Associate Professor in the Department of Audiology at Towson University, by phone at (410) 704-5903 or by e-mail at pkorczak@towson.edu or the Institutional Review Board Chairperson, Dr. Debi Gartland, Office of University Research Services, 8000 York Road, Towson University, Towson, Maryland 21252; phone: (410) 704-2236; e-mail address: ours@towson.edu.

APPENDIX C

Vestibular Case History Form

Subject #: _____

Vestibular Case History Questionnaire

1. Have you ever had surgery on either of your ears?

   Circle: **YES** **NO**

   If yes, please state the name/describe the surgery, and state the ear(s) on which the surgery was performed.

   _____
   _____

2. Have you ever had symptoms/episodes of dizziness?

   Circle: **YES** **NO**

   If yes, please describe the sensation of dizziness (e.g., lightheaded, spinning, off-balanced)

   _____
   _____

   How often does the dizziness occur?

   _____

3. Have you ever been medically evaluated for dizziness or undergone treatment for symptoms of dizziness?

   Circle: **YES** **NO**

   If yes, please describe.

   _____
   _____

APPENDIX D

PARTICIPANT FLYER

Participants needed for Dizziness Research!

Why? 	To gather normative data on the Vestibular Evoked Myogenic Potential (VEMP), a diagnostic tool used in regular clinical practice to evaluate the dizzy patient. Normative data will give the Towson University Speech, Language & Hearing Center the ability to conduct VEMP testing on campus, in order to provide a more comprehensive test battery for the evaluation of dizzy patients.
What is the VEMP?	The VEMP is a diagnostic test used to evaluate the function of the saccule, one of the balance/equilibrium organs in the inner ear.
Who?	We are looking for participants aged 18-30 years with normal ear function.
Where?	All testing will be conducted at the *Towson University Speech, Language & Hearing Center* located in Van Bokkelen Hall.
When?	Appointments for testing will be offered throughout the year during evenings, weekends, and during holiday breaks. Total test time will take approximately 1 hour.
Interested in learning more?	**If you are between 18 and 30 years of age and willing to volunteer for our study, please contact:** Lauren McGrath (Audiology Doctoral Student) at **lmcgra2@students.towson.edu** or **(856) 904-0331** Sasha Phillips (Audiology Doctoral Student) at **sphil5@students.towson.edu** or **(301) 523-0946** Dr. Peggy Korczak (Associate Professor) at **pkorczak@towson.edu** or **(410) 704-5903**

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