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NORMATIVE STUDY ON 500 Hz TONE BURST VESTIBULAR EVOKED MYOGENIC POTENTIAL (VEMP) ASSESSMENT

by
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THESIS APPROVAL PAGE

This is to certify that the thesis prepared by **Lauren Gene McGrath** entitled **Normative study on 500 Hz tone burst Vestibular Evoked Myogenic Potential (VEMP) assessment** has been approved by the thesis committee as satisfactorily completing the thesis requirements for the degree **Doctor of Audiology (Au.D.)**.

Peggy Korczak — Chair, Thesis Committee — 5-15-2012 — Date

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ABSTRACT

NORMATIVE STUDY ON 500 Hz TONE BURST VESTIBULAR EVOKED MYOGENIC POTENTIAL (VEMP) ASSESSMENT

Lauren Gene McGrath

The goal of the current study was to establish normative data on the various response indices of the VEMP assessment, when the response was elicited to 500 Hz tone burst stimuli. Eighteen adults with normally functioning vestibular systems participated in the current study. Responses were recorded on the sternocleidomastoid (SCM) muscles on each side of the necks of these subjects. Ipsilateral and contralateral recordings occurred for both sides of the neck, with the ipsilateral channel recording from the contracted SCM muscle on the same side of the monaural stimulation of 500 Hz tone burst stimuli, and the contralateral channel recording from the non-contracted SCM muscle on the opposite side to stimulation. The tonic EMG levels of the SCM muscle contraction were recorded during VEMP recordings for the ipsilaterally contracted SCM muscles.

Two supra-threshold (i.e., 95 and 90 dB nHL) stimulus intensities were utilized to explore the characteristics of the robust VEMP response. Several response indices were measured from these supra-threshold VEMP responses. These included: (1) the absolute latency value of waves p13 and n23; (2) the peak-to-peak amplitude values of p13-n23;

(3) interaural asymmetry ratio values for amplitude (4) laterality of the VEMP response; and (5) tonic EMG levels recorded during the supra-threshold VEMP recordings. The VEMP response was evaluated at lower stimulus intensities (i.e., 70-85 dB nHL) to determine each subject's VEMP threshold.

The findings from the current study were, for the most part, found to be consistent with the VEMP literature. Latency values were stable across subjects for each of the VEMP components. Peak-to-peak amplitude values of p13-n23 were highly variable across our subjects. Interaural asymmetry ratio values were largely less than 30%, but in some cases were elevated and/or differed across stimulus intensities in the same subject. VEMP thresholds were consistent with the documented literature. The VEMP response was found to be largely dominant in the ipsilateral recording channel. Tonic EMG levels were found to be extremely variable across subjects with the natural contraction of SCM muscles. However, these tonic EMG levels were found to be very consistent across VEMP recordings in a single subject.

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ABBREVIATION KEY

Abbreviation	Longhand Name
ENG	Electronystagmography
VNG	Videonystagmography
SCC	Semi-circular Canal
VEMP	Vestibular Evoked Myogenic Potential
CNS	Central Nervous System
VOR	Vestibulo-ocular Reflex
SVN	Superior Vestibular Nucleus
LVN	Lateral Vestibular Nucleus
MVN	Medial Vestibular Nucleus
IVN	Inferior Vestibular Nucleus
SCM	Sternocleidomastoid
PAM	Post-auricular Muscle
BPPV	Benign Paroxysmal Positional Vertigo
EMG	Electromyographic
SCD	Superior Semi-circular Canal Dehiscence
SD	Standard Deviation
ABR	Auditory Brainstem Response
NR	• Table 2 – not reported • All other tables/figures – no response
ms	Milliseconds
µV	Microvolts
RMS	Root Mean Square
ER	Etymotics Research
CT	Computerized Tomography
MRI	Magnetic Resonance Imaging
FPA	Frequency Peak Amplitude
IHS	Intelligent Hearing Systems
ANOVA	Analysis of Variance

CHAPTER 1

INTRODUCTION

Dizziness is a common complaint of patients who seek medical attention. In fact, it is the third most common medical complaint in the American adult population, and the cost of medical care for dizzy patients exceeds $1 billion per year (Gans, 1999; NIDCD, 2008). Audiologists and hearing scientists rely on a number of diagnostic tests to assess patients with varying symptoms of dizziness. In current clinical practice, the electronystagmography/videonystagmography (ENG/VNG) test battery is the most prevalent assessment to evaluate patients with symptoms of dizziness (Wuyts, Furman, Vanspauwen, & Van de Heyning, 2007). A considerable limitation of the caloric portion of the ENG/VNG test battery is that it only evaluates the integrity of the horizontal semicircular canals (SCCs). The ENG/VNG test battery is also limited in the frequency range of head and body movements that can be assessed.

The vestibular evoked myogenic potential (VEMP) is also an important diagnostic tool for use in patients with vestibular disorders because it assesses the integrity of one of the Otolith organs, the saccule, which would not otherwise be evaluated in the ENG/VNG evaluation. Since the horizontal SCC is innervated by the superior branch of the vestibulo-cochlear nerve, whereas the saccule is innervated by the inferior branch of the vestibular nerve (Lysakowski, 2005), the VEMP also allows for the assessment of the inferior portion of the vestibular nerve. The current study will focus on the VEMP; therefore, the ensuing literature review will be limited to that topic.

In order for audiologists and/or hearing scientists to understand the VEMP, these individuals need to have a good understanding of the anatomy and physiology of the

peripheral and central vestibular systems. It is also important that these clinicians appreciate how the VEMP procedure was developed. In order to correctly administer this test and properly interpret the results, audiologists and hearing scientists need to exhibit knowledge regarding the optimal stimulus and recording parameters, as well as subject factors, that may influence the VEMP test results. Lastly, it is also important to understand which patients are appropriate referrals for this type of diagnostic testing. The organization of the literature review that follows will focus on these various topics.

CHAPTER 2

LITERATURE REVIEW

Anatomy and Physiology of the Vestibular System

There are three sensory systems in the human body that contribute to one's sense of equilibrium and position in space. The visual and somatosensory systems receive input from external sources, namely visual and tactile input from one's surroundings. In contrast, the vestibular system receives input internally, and relays information regarding the body's direction and velocity of movement. One's internal reference of equilibrium receives input from the peripheral vestibular system and central vestibular system. The overview of the anatomy and physiology of the vestibular system, which follows, will discuss the anatomy of the peripheral and central vestibular systems, and will include a discussion of the physiology of these two systems. Lastly, there will be a discussion of how the central nervous system integrates the information from these three sensory systems.

The Anatomy of the Peripheral Vestibular System

The peripheral vestibular system is anatomically located in the petrous portion of the temporal bone in each inner ear, which is in close proximity to the cochlea, the organ responsible for our sense of hearing. Similar to the cochlea, the sensory organs of the peripheral vestibular system are encapsulated by a membranous labyrinth, which is contained within the bony labyrinth (vestibule). Each of these labyrinths is filled with fluids of different chemical compositions. The membranous labyrinth is filled with potassium-rich endolymph fluid, and the space between the membranous labyrinth and the vestibule is filled with sodium-rich perilymph fluid. The sensory organs in the

peripheral vestibular system are collectively responsible for detecting the linear and angular motions of the head and body in space. These organs are comprised of three SCCs and two Otolith organs in each ear. These sensory organs send information to the central vestibular system, which integrates the information from the peripheral vestibular system with the information from the visual and somatosensory systems in the central nervous system (CNS).

Each of the sensory organs in the peripheral vestibular system responds to input from different movements of the head and/or body. There are six total SCCs in the human vestibular system, three in each ear. The SCCs are oriented in roughly ninety-degree angles in relation to one another and occupy three parallel planes of space. The SCCs are named for the angular orientation or plane that they occupy: horizontal, anterior, and posterior. Each of the three SCCs in one ear is paired with the SCC in the other ear that occupies the same angular orientation or plane. The anterior end of the horizontal or lateral SCC in each of the ear is oriented approximately thirty degrees above the horizontal axis (see Figure 1A). The horizontal SCCs from each ear occupy the same plane, and act as one pair. The anterior and posterior SCCs are oriented vertically at roughly forty-five degrees from the midsagittal plane, and thus occupy two different angular orientations in each ear. The anterior SCC in one ear acts as a coplanar pair with the contralateral posterior SCC, as they both occupy the same angular orientation or plane. Figure 1B illustrates the orientation of the anterior and posterior SCCs in the head.

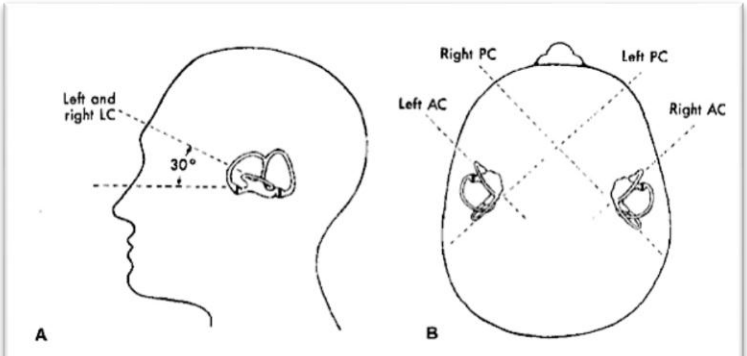


Figure 1. The orientation of the SCCs in the **head. A:** The anterior ends of the horizontal SCCs is oriented roughly 30 degrees above the horizontal **axis. B**: The anterior and posterior SCCs are oriented at roughly 45 degree angles from the **midsaggital** plane. The anterior SCC from one side is oriented at the same angle as the posterior SCC **from** the opposite side. Obtained from Lysakowski (2005).

At the base of each of the semicircular canals is a bulb-like area, called the ampulla. This area contains a bundle of hair cells referred to as the cristae ampullaris. The ampulla also contains a structure known as the cupula, which is a gelatinous plug that sits on top of the bundles of hair cells. At the top of each hair cell sits a group of stereocilia of varying lengths; the tallest of them is referred to as the kinocilium. An illustration of these structures is provided in Figure 2A. The orientation of the kinocilium is important in the physiology of the peripheral vestibular system because when the stereocilia are bent toward the kinocilium, the hair cell provides an excitatory response. In contrast, when the stereocilia are bent away from the kinocilium, an inhibitory response occurs.

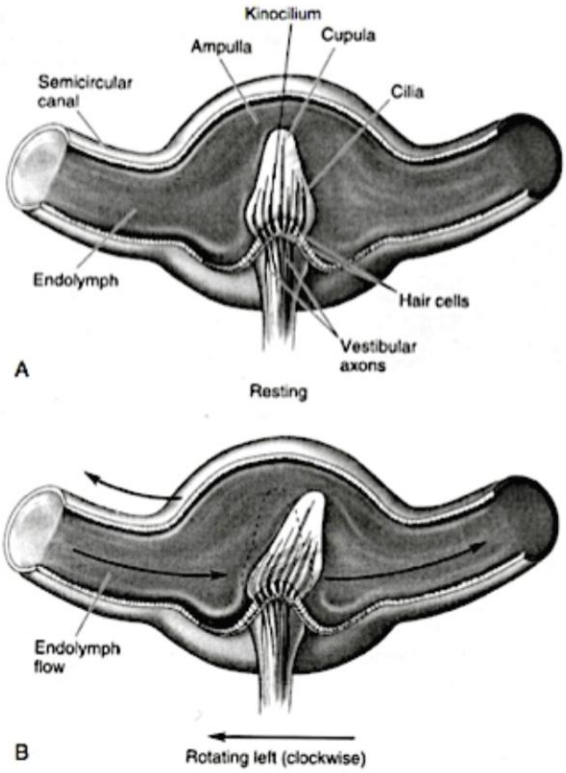


Figure 2. Cross-sectional view of the ampullar structures of each SCC. **A**: The hair cells are located within the gelatinous cupula in each ampulla. The stereocilia are embedded in the cupula. The tallest of the stereocilia is termed the kinocilium. **B**: When the SCC is rotated leftward, the endolymph fluid flows in the opposite direction, applying force to the cupula. The stereocilia are bent towards the kinocilium, providing an excitatory response. Adapted from Bear et al. (2007).

In the horizontal SCC, the kinocilium for each hair cell is located on the side of the cell that is closest to the utricle in that ear. Whereas in the posterior and anterior SCCs, the kinocilium is located on the side of the hair cell that is farthest away from the utricle (Lysakowski, 2005). The SCCs are designed to detect the direction and velocity of angular acceleration or head rotations. When one SCC provides an excitatory response, it is paired with a SCC on the opposite side, which provides an inhibitory response in that same plane of space. The comparison between the responses from the two SCCs is then encoded by the central vestibular system.

The utricle and saccule are collectively referred to as the Otolith, or macular organs. The Otolith organs provide information regarding gravitational pull. The utricle is oriented roughly in the horizontal plane, and detects linear acceleration in the horizontal plane. The saccule is oriented roughly in the vertical plane, and detects linear acceleration in the vertical plane. The mound of hair cells, or sensory epithelium, located in each of these sensory organs is located in structures referred to as the maculae. Within the macula is a gelatinous plug similar to the cupula in the SCCs, which sits on top of the hair cells.

Each of the macular organs also contains a unique collection of calcium carbonate crystals, which are referred to as otoconia. The otoconia vary in size, with most of the crystals being approximately 5 to 7 μm (Lysakowski, 2005). The otoconia have a higher density in comparison to the surrounding endolymph fluid (Bear, Connors, & Paradiso, 2007). The gravity of the otoconia has been found to be approximately 2.7 times that of the endolymph fluid (Wall & Vrabec, 2001), making their movement sensitive to gravitational pull and other linear acceleration forces. Figure 3 illustrates the anatomical structure of the maculae.

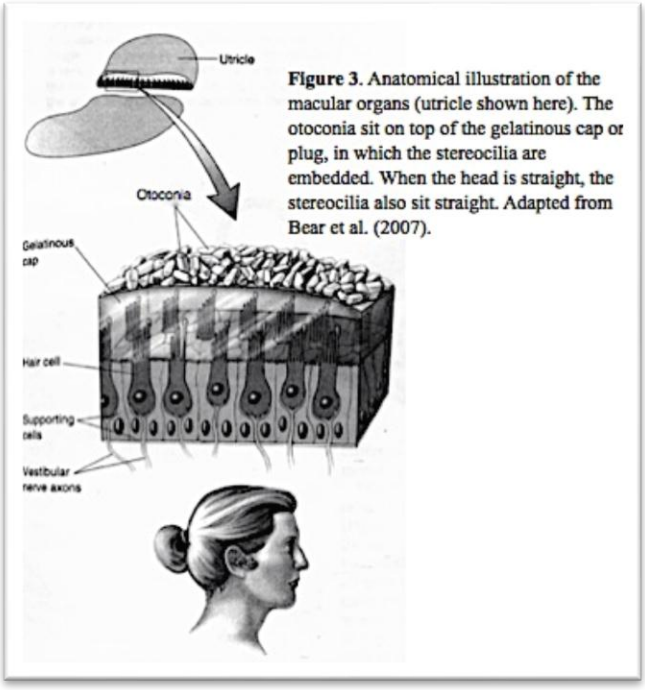


Figure 3. Anatomical illustration of the macular organs (utricle shown here). The otoconia sit on top of the gelatinous cap or plug, in which the stereocilia are embedded. When the head is straight, the stereocilia also sit straight. Adapted from Bear et al. (2007).

In the center of each of the macular organs, a thin, curved line called the striola exists. This curved line determines the orientation of the kinocilium. In the utricle, the kinocilium are oriented towards the striola, whereas in the saccule the kinocilium are oriented away from the striola (Wall & Vrabec, 2001). The Otolith organs are curvature in nature so that they may provide input regarding gravitational pull in any direction to the central nervous system. As in the SCCs, the mirror-like orientation of the hair cells in the macular organs causes a hair cell to provide an excitatory response on one side and an inhibitory response at the hair cell in the same location on the opposite side (Bear et al., 2007; Lysakowski, 2005). The orientation of the macular organs and the direction of their kinocilium towards or away from the striola are illustrated in Figure 4.

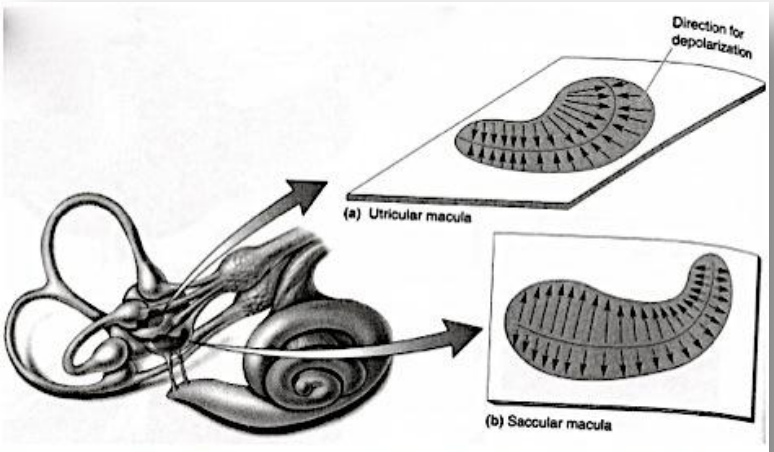


Figure 4. Orientation of the macular organs. The arrows indicate the direction in which the stereocilia must bend to provide an excitatory response. **A**: The utricle is oriented roughly in the horizontal plane. The stereocilia are bent toward the striola to provide an excitatory response. **B**:The saccule is oriented roughly in the vertical plane. The stereocilia are bent away from the striola to provide an excitatory response. Obtained from Bear et al. (2007).

There are two types of hair cells found in the three SCCs as well as the two macular organs. These are type I and type II hair cells. These hair cells are analogous to the two types of hair cells that are located in the cochlea. One primary difference that exists for these two types of hair cells is the afferent and efferent connections they receive. The type I hair cells receive a direct afferent connection. This connection is referred to as a calyx. It is a chalice-like connection that envelops the majority of the hair cell. In contrast, the efferent connection to the Type I hair cells is indirect in nature, in that the efferent nerve does not synapse directly with the hair cell. Specifically, the efferent nerve synapses on the afferent nerve below the Type I hair cell. Type II hair cells are cylindrical in shape, and they receive a direct afferent and efferent synaptic connection.

The vestibular portion of the vestibulocochlear nerve (or VIIIth cranial nerve) is divided into the superior and inferior branches. The superior branch receives input from the utricle, anterior portion of the saccule, and the horizontal and anterior SCCs, while the inferior division receives input from the posterior portion of the saccule as well as the posterior SCCs. These branches of the vestibulocochlear nerve join the cochlear portion of the VIIIth nerve in the internal auditory meatus and project ipsilaterally to the vestibular nuclei.

Anatomy of the Central Vestibular System

The vestibular nuclei, located in the lateral-dorsal area of the medulla (Bhatnagar, 2002), are classified into four subdivisions: the superior, lateral, medial, and inferior vestibular nuclei. The superior vestibular nucleus can be divided into two regions: the

central portion, which contains medium-sized neurons, and the peripheral portion, which contain smaller-sized neurons (Lysakowski, 2005). The superior vestibular nucleus receives input from eye as well as head movements, and is involved in the vestibulo-ocular reflex (VOR). The lateral vestibular nucleus can also be divided into two regions: the dorsal lateral vestibular nuclei, which contain large neurons, and the ventral lateral vestibular nucleus, which contains medium-sized neurons. The dorsal lateral vestibular nucleus gives rise to the lateral vestibulo-spinal tract. The ventral lateral vestibular nucleus, on the other hand, is responsible for, in part, forming the vestibulo-ocular pathways, the medial vestibulo-spinal tract, and the vestibulo-thalamic pathways. The lateral vestibular nuclei are collectively involved in the vestibulo-spinal and vestibulo-collic reflexes (Lysakowski, 2005).

The medial vestibular nuclei contain small- and medium-sized neurons, and can be divided into three subdivisions: the rostral magnocellular and parvocellular regions, and the caudal region (Lysakowski, 2005). The rostral medial vestibular nucleus is also related to eye movements. The caudal portion of the medial vestibular nucleus projects to the cerebellum. The inferior vestibular nucleus primarily receives afferent input from the macular organs. The primary projections of the inferior vestibular nucleus are to the cerebellum and reticular formation, and some of the cells project to the vestibulo-spinal tract (Lysakowski, 2005). Figure 5 shows the divisions of the vestibular nuclei.

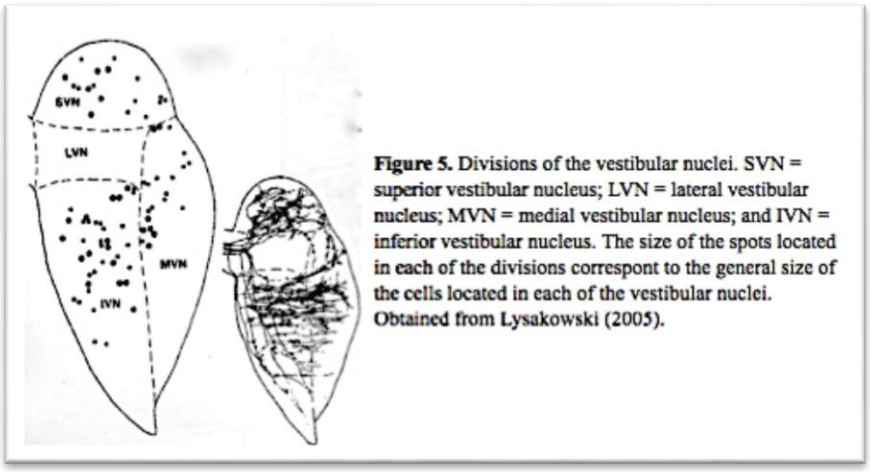


Figure 5. Divisions of the vestibular nuclei. SVN = superior vestibular nucleus; LVN = lateral vestibular nucleus; MVN = medial vestibular nucleus; and IVN = inferior vestibular nucleus. The size of the spots located in each of the divisions correspont to the general size of the cells located in each of the vestibular nuclei. Obtained from Lysakowski (2005).

Upon entering the vestibular nuclei, a majority of the afferent vestibular nerve fibers branch off into the rostral and caudal branches of the vestibular nerve. The rostral branch projects to the superior and medial vestibular nuclei and also sends a secondary

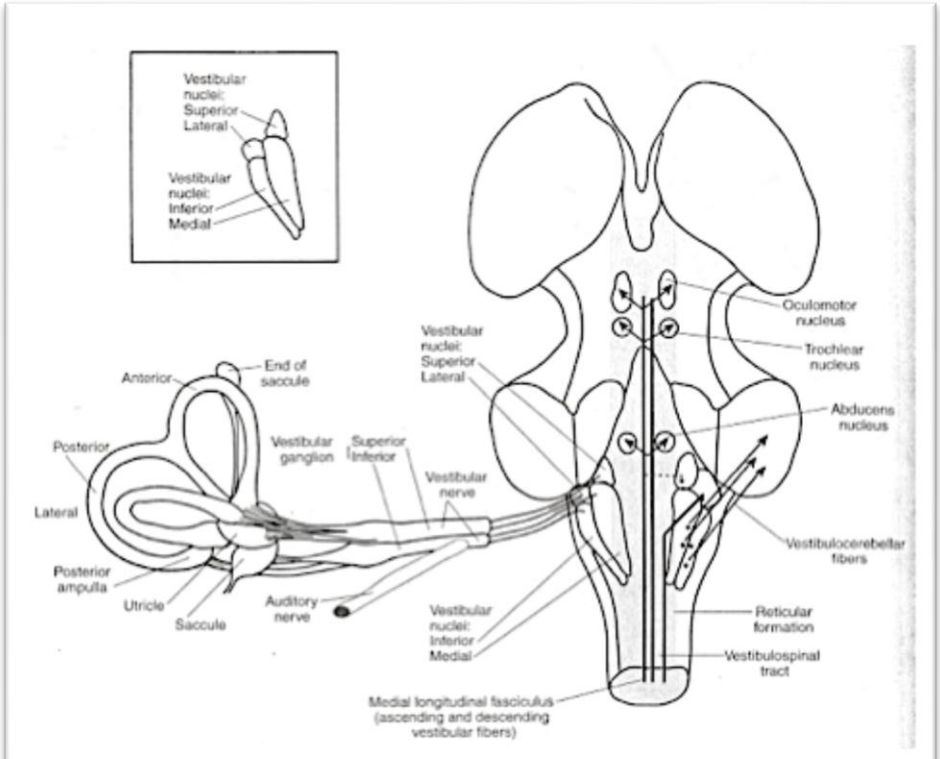


Figure 6. The peripheral vestibular organs and their projections to the vestibular nuclei, located in the brainstem. Obtained from Bhatnagar (2002).

portion to the cerebellum. The rostral branch of the vestibular nerve primarily sends information regarding Otolith function (Lysakowski, 2005). The caudal branch, in contrast, projects to the inferior and medial vestibular nuclei as well as the venterolateral portion of the lateral vestibular nucleus. The caudal branch primarily receives information regarding SCC function (Lysakowski, 2005). Figure 6 provides an abstract view of the innervations provided by the divisions of the vestibular nerve. The four vestibular nuclei and cerebellum, when discussed as a unit, integrate the information sent from the peripheral vestibular centers with visual and tactile input. The appropriate information is sent to the muscles of the eyes and motor pathways, providing the proper motor response to maintain balance.

As mentioned above, the vestibular nuclei collectively receive afferent information through nerve connections from the visual system, cerebellum and spinal cord. Collectively, the input entering the vestibular nuclei aids in supplementing the vestibular input through the visual surroundings and modifying head movements in compensation for maintaining balance. The input to the vestibular nuclei also allows for the act of maintaining a visual object in line of view while moving the head. The supplemental input from these other centers can be utilized when a lesion occurs in the peripheral vestibular system. The vestibular nuclei provide several efferent connections that facilitate communication with the cranial nerves (oculomotor, trochlear, and abducens) that innervate the extraocular muscles and the spinal cord. The anatomical connections provide for important functional roles that activate eye movements and maintain posture.

Efferent connections from the superior, medial, and ventral lateral vestibular nuclei form the pathways involved in controlling eye movements (Lysakowski, 2005). The six extraocular muscles collectively account for all possible eye movements. The lateral and medial rectus muscles move the eyes outward and inward, respectively. The lateral rectus muscle is innervated by the motor axons from the abducens (VI) cranial nerve and the motor axons from the oculomotor (III) cranial nerve innervate the medial rectus muscles. The superior rectus muscles are responsible for moving the eyes upward and slightly inward and outward, resulting in a torsional or curvilinear motion. The inferior rectus muscles are responsible for moving the eyes downward and slightly inward and outward, resulting in a similar torsional movement. Both the superior and inferior rectus muscles generate these torsional or curvilinear motions of the eyes and both are innervated by the motor axons of the oculomotor (III) cranial nerve.

The superior oblique muscles generate rotations of the eyes so that the tops of the eyes are oriented toward the nose (medially). The inferior oblique muscles generate rotations of the eyes so that the tops of the eyes are oriented away from the nose (laterally). The superior oblique muscles are innervated by the motor axons of the trochlear (IV) cranial nerve and the inferior oblique muscles are innervated by the motor axons of the oculomotor (III) cranial nerve. The six extraocular eye muscles are important in the proper function of the VOR, which stabilizes eye movements as well as vision during movements of the head (Lysakowski, 2005). A further description of the physiology of the VOR can be found in a later portion of the literature review.

The second major efferent pathway originating in the vestibular nuclei is referred to as the vestibulo-spinal tract. The vestibulo-spinal tract is further divided into two

tracts: the medial vestibulo-spinal tract and the lateral vestibulo-spinal tract. The efferent connections from the medial and ventral lateral vestibular nuclei collectively form the medial vestibulo-spinal tract, which ultimately terminates in the cervical spinal cord (Lysakowski, 2005). The functions of the medial vestibulo-spinal tract stabilize the head by producing muscle contractions in the neck to adjust for reflexive head movements. The primary role of the lateral vestibulo-spinal tract is to maintain posture. This tract originates in the dorsal lateral vestibular nucleus, which is known to be sensitive to tilting motions of the head. The lateral vestibulo-spinal tract terminates in the lumbar region of the spinal cord (Lysakowski, 2005).

The Physiology of the Vestibular System

Physiologically, the peripheral vestibular system encodes information regarding angular acceleration in the SCCs, and linear acceleration in the utricle and saccule. Angular acceleration refers to rotations of the head from side to side (i.e., looking from the left to the right), moving the head from the chin touching the chest and backwards, and the head movements associated with each ear tilting toward the shoulder on its respective side (Wall & Vrabec, 2001). Any of these head and/or body motions can activate any of the six SCCs, however each type of movement will maximally activate one pair of SCCs and partially activate the remaining pairs. For example, moving the head to look from one side of the body to the other will maximally activate the horizontal canals, providing an excitatory response from the side at which the head is turned and an inhibitory response from the opposite canal.

This occurs because the anatomical positioning of the kinocilium on the right side is a mirror image of the anatomical positioning of the kinocilium on the left side. The endolymph fluid in each of the SCCs flows through to the ampulla towards the opposite direction of the given head movement, applying force on the cupula, distorting this gelatinous plug in the direction of the endolymph flow. The distortion of the cupula moves the stereocilia either toward or away from the kinocilium, activating the crista on that side to provide either an excitatory or inhibitory response. See Figure 2B for a visual representation of this physiologic phenomenon. If the head were turned to the right side, the endolymph fluid would flow towards the left in each of the paired SCCs. This would cause the cristae in the right horizontal SCC to provide an excitatory response of a given magnitude, and cristae in the left horizontal SCC would provide an inhibitory response of the same magnitude. When a hair cell is activated with an excitatory response, the stereocilia are displaced toward the kinocilium.

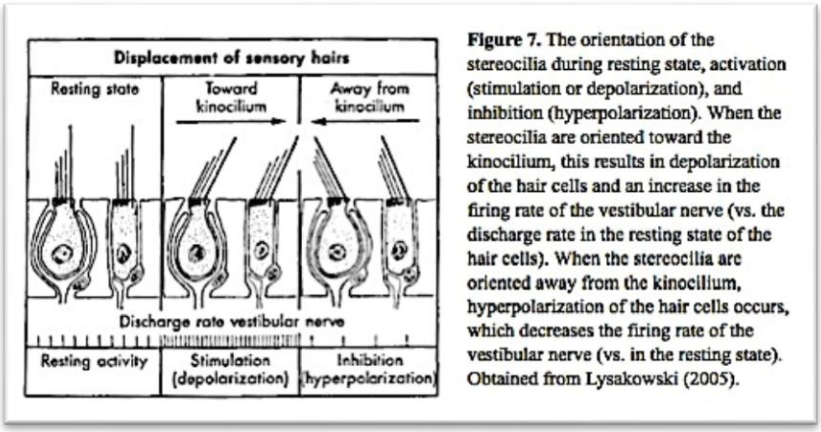


Figure 7. The orientation of the stereocilia during resting state, activation (stimulation or depolarization), and inhibition (hyperpolarization). When the stereocilia are oriented toward the kinocilium, this results in depolarization of the hair cells and an increase in the firing rate of the vestibular nerve (vs. the discharge rate in the resting state of the hair cells). When the stereocilia are oriented away from the kinocilium, hyperpolarization of the hair cells occurs, which decreases the firing rate of the vestibular nerve (vs. in the resting state). Obtained from Lysakowski (2005).

This depolarizes the hair cell, opening the potassium channels in the membrane of the hair cell, and an action potential increases the rate of nerve firing for that side. In contrast, the hair cells in the opposite paired canal are hyperpolarized simultaneously.

The stereocilia are displaced away from the kinocilium, resulting in a decrease in discharge rate in the corresponding neurons. See Figure 7 for an illustration of the depolarization and hyperpolarization of the vestibular hair cells. Comparisons are made between the responses of each of the paired SCCs and the encoded messages are sent through the afferent pathways to the central vestibular system.

In the macular organs, the same depolarization and hyperpolarization processes take place in their respective hair cells. Both of the macular organs detect gravitational pull and linear acceleration, but they do so in different planes. The utricle, oriented roughly in the horizontal plane, detects linear acceleration in terms of forward and backward motion (Bear et al., 2007). Examples of this type of motion include walking forward or backward, or driving in a car. The saccule detects linear acceleration in terms of up and down movements, as it is oriented in roughly the vertical plane (Bear et al., 2007). Examples of this type of motion include ascending or descending in an elevator, head bobbing while walking, or jumping up and down.

The information regarding linear acceleration is coded depending on which section of the macula that the hair cells are excited and where they are inhibited. As previously stated, the kinocilium are oriented in the opposite way in the utricle and the saccule depending on the relation of the kinocilium to the striola. In the utricle, stereocilia bent towards the kinocilium, as well as the striola, excites the corresponding hair cell (Wall & Vrabec, 2001). In the saccule, the opposite is true. The stereocilia bent toward the kinocilium but away from the striola provide an excitatory response to the afferent nerves contacting that hair cell (Wall & Vrabec, 2001).

Because the striola is curvature in nature, the stereocilia located in the Otolith organs are oriented in a complex manner, and correspond to any direction of gravitational head movements (Wall & Vrabec, 2001). Any given movement will maximally activate (or inhibit) stereocilia in specific regions of the macula and partially activate (or inhibit) stereocilia in other regions, providing specific encoded responses to the central vestibular system (Lysakowski, 2005). Refer to Figure 8 for an illustration of the kinocilium orientation in each of the Otoliths in comparison to those in the cristae ampullaris of the SCCs. Figure 9 provides a visual display of several natural head and body movements and identifies the type of acceleration that is activated.

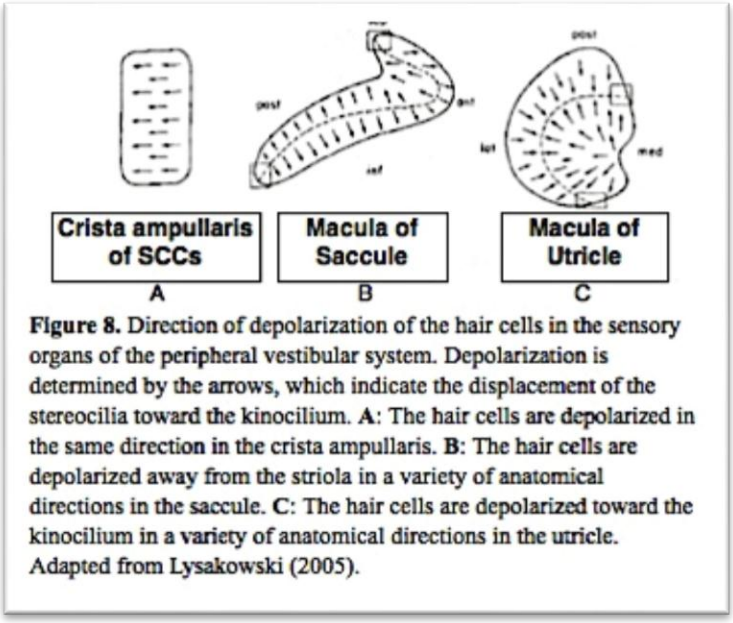


Figure 8. Direction of depolarization of the hair cells in the sensory organs of the peripheral vestibular system. Depolarization is determined by the arrows, which indicate the displacement of the stereocilia toward the kinocilium. **A**: The hair cells are depolarized in the same direction in the crista ampullaris. **B**: The hair cells are depolarized away from the striola in a variety of anatomical directions in the saccule. **C**: The hair cells are depolarized toward the kinocilium in a variety of anatomical directions in the utricle. Adapted from Lysakowski (2005).

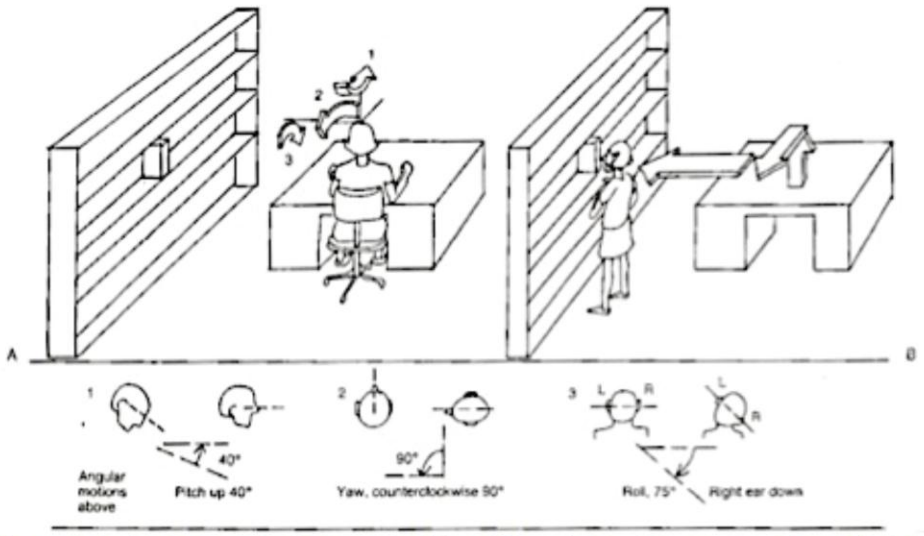


Figure 9. An illustration of various angular (A) and linear (B) head movements. The numbered illustrations (bottom pane) display the angular motions shown in A. **A:** ***Angular acceleration.*** 1. The illustrated person, looking down at a book on the desk, lifts her head up (40 degrees) to look straight ahead. 2. She then turns her head 90 degrees to the left to look at the book on the bookshelf. 3. In order to read the title on the spine of the book, she tilts her head with her right ear down (75 degrees). **B**: *Linear Acceleration.* In order to take a closer look at the book on the bookshelf, she moves back in her chair, stands up, and moves to her left, walking to the bookshelf (indicated by the arrows). Obtained from Wall & Vrabec (2001).

The afferent connections from the peripheral vestibular system are integrated in the vestibular nuclei with connections from the cerebellum, spinal cord, and visual systems. The vestibular nuclei then provide output to the muscles of the eye and through the cerebellum and spinal cord, ultimately reaching muscles in the legs, neck and spine (postural muscles). The pathway leading to the eye muscles, called the vestibular oculo-motor pathway, is involved in the VOR, an important reflex dependent on the proper functioning of the SCCs of the vestibular system and its corresponding pathways. The connection between the vestibular system and the oculomotor muscles allows for the stabilization of the eyes during active head movements.

The VOR, when functioning properly, describes the one-to-one ratio between the movement of the head and the inverse reaction of the eyes in terms of phase angle. As a

general example, turning one's head to the right 45 degrees activates the VOR via input from the horizontal SCCs, forcing both eyes immediately towards the left in equal magnitude (i.e., 45 degrees). This allows for fixation on the visual target of interest and for the ability of the eyes to focus. While the information from the horizontal canals provides the eyes the ability to focus in the horizontal plane, the anterior and posterior canals provide them the ability to focus in the vertical plane. The cohesive functioning of the SCCs and their connective pathways is integral to achieve the VOR.

To review, a left head-turn would elicit a series of afferent and efferent connections to generate the VOR, ultimately facilitating the movement of both eyes to the right. The following description of the VOR only involves the input of the horizontal SCCs. The afferent connections originating in the left horizontal SCC activates the pathway to the left vestibular nuclei (Bear et al., 2007). This activation in the left vestibular nuclei sends an excitatory response to the right (contralateral) abducens nucleus, where the abducens cranial nerve (VI) originates. This excitatory connection, in turn, provides activation in two pathways. The first activates the motor axons from the abducens nerve, which in turn activates the right (ipsilateral) lateral rectus muscle, moving the right eye in the right direction. The second excitatory projection from the abducens crosses the midline and projects to the contralateral (left) oculomotor nucleus, where the oculomotor cranial nerve (III) originates, via the medial longitudinal fasiculus (Bear et al., 2007).

The activation of the left oculomotor (III) nerve, in turn, activates the motor axons from the oculomotor nerve, which in turn activates the left medial rectus muscle, moving the left eye toward the right (Bear et al., 2007). The left oculomotor (III) nerve also receives projections from the ipsilateral vestibular nucleus. These two connections to the left oculomotor (III) nerve collectively facilitate the leftward movement of the left eye. Inhibitory connections are also provided to the lateral rectus of the left eye and the medial rectus muscles of the right eye (Bear et al., 2007). Figure 10 displays a visual representation of the connections involved in the VOR.

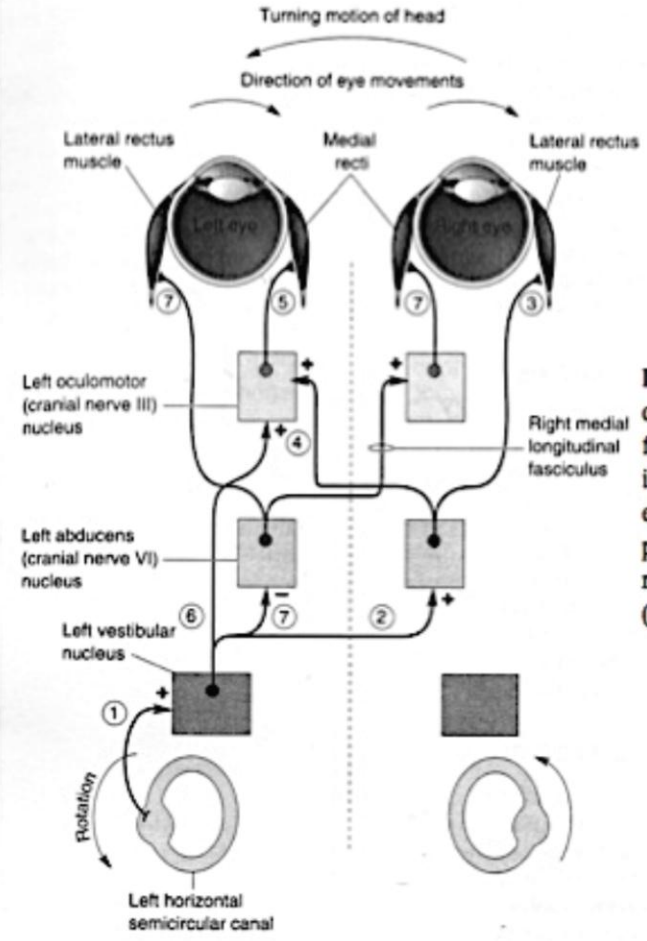


Figure 10. Afferent and efferent neural connections involved in the VOR. This figure describes the VOR in response to input from the horizontal SCCs. The excitatory responses are denoted by the plus signs (+) and the inhibitory responses are denoted by the minus signs (-). Obtained from Bear et al. (2007).

Efferent connections from the vestibular nuclei also project to the vestibulo-spinal pathways, which send information regarding equilibrium to the muscles that keep the head and body stable. These motor pathways facilitate the appropriate compensatory body or head movements to maintain stability (Lysakowski, 2005). As previously described in the discussion of the anatomy of the central vestibular system, the medial and lateral vestibulo-spinal tracts, collectively aid in stabilizing the eyes during head movements (along with the VOR), stabilizing the head on the shoulders by the activation of neck muscles, and maintaining posture (Lysakowski, 2005). For example, if one were to trip and begin to fall, the vestibular system provides input regarding balance and the muscles of the legs or arms could be activated to catch the fall.

From its most peripheral portions and above to the centrally integrated information of the vestibular, visual, and somatosensory systems, the vestibular system is anatomically and physiologically complex. However, it provides some of the most essential human functions, such as keeping the eyes in focus and the body and head balanced. An understanding of the basic anatomy and physiology of the peripheral and central vestibular systems is essential in understanding test results from various vestibular assessments in order to accurately describe sites of various vestibular lesions.

Introduction to the Vestibular Evoked Myogenic Potential (VEMP)

The VEMP is a myogenic response that reflects activity from the vestibulo-collic reflex. A discussion of this reflex will follow. Currently, this myogenic potential is elicited by either high intensity click and/or low frequency tone burst stimuli and recorded from active electrode sites located on the sternocleidomastoid (SCM) muscles. It can be recorded either unilaterally or bilaterally, from either one or both activated SCM muscles, respectively. The labeling system used in discussing myogenic potentials was established by Yoshie and Okudaira (1969), who used the lowercase letters "p" and "n," followed by the mean peak latency in milliseconds, to denote the positive peaks relative to the zero baseline and the negative peaks relative to the baseline in order to differentiate these myogenic responses from neural responses that are recorded from the scalp.

As shown in Figure 11, the response waveform in an adult with a normally functioning vestibular system, when measured from electrodes placed on the SCM, is characterized by a positive peak occurring at an approximate latency of 13 milliseconds (known as p13), followed by a negative peak occurring at a latency of roughly 23 milliseconds (known as n23) (Colebatch & Halmagyi, 1992). These components are often described as the p13-n23 complex. It was possible to successfully record p13-n23 responses in all subjects with normally functioning vestibular systems who were included in the study documented by Colebatch, Halmagyi, and Skuse (1994).

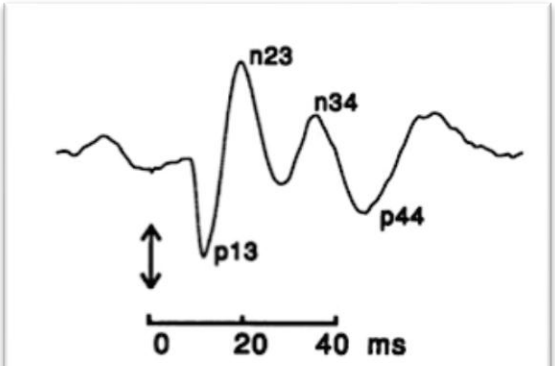


Figure 11. Normal VEMP response waveform obtained from the ipsilateral side to stimulation in a healthy adult subject. Obtained from Colebatch et al. (1994).

In some subjects with normal vestibular function, another waveform complex, known as the n34-p44 complex follows the p13-n23 complex, as seen in the negative and positive peaks following wave n23 in Figure 11 (Colebatch et al., 1994a). Colebatch et al. (1994a) estimated that the second complex, n34-p44, is present in approximately sixty percent of normal subjects. Welgampola and Colebatch (2005) reported that the n34-p44 complex has a much lower response threshold and is most likely cochlear, rather than vestibular, in nature. Due to the inconsistency in the presence of the n34-p44 complex and its possible non-vestibular nature, most of the literature in this area tends to focus on the p13-n23 response. The p13-n23 response is more recently referred to as the VEMP.

It is important to not only have a basic understanding of the vestibulo-collic reflex pathway but also to appreciate the history of how investigators determined the optimal stimulus and recording parameters for obtaining the VEMP. Both of these topics will be covered in the next 2 sections of this literature review.

Vestibulo-collic Reflex Pathway

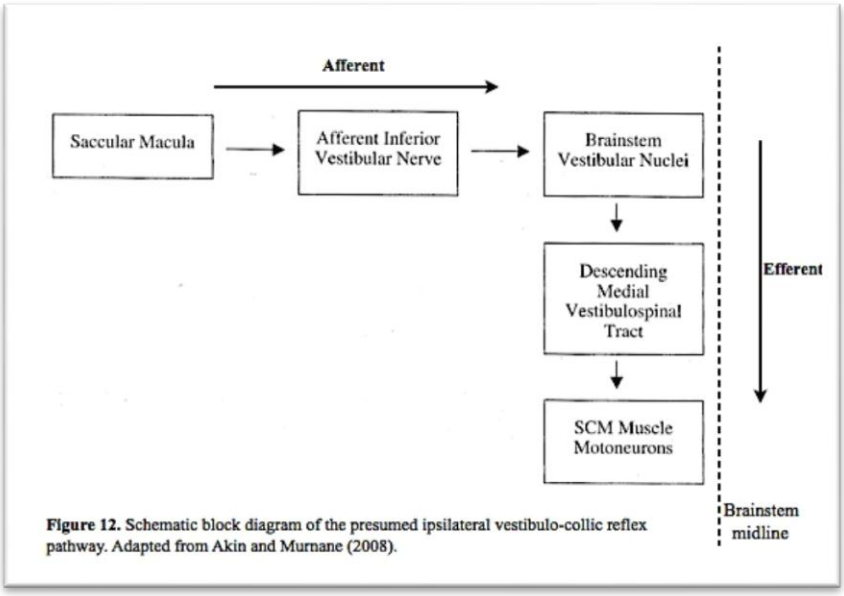


Figure 12. Schematic block diagram of the presumed ipsilateral vestibulo-collic reflex pathway. Adapted from Akin and Murnane (2008).

The myogenic response, which underlies the VEMP, reflects activity from the vestibulo-collic reflex, a pathway that is less often studied in the literature versus the well-studied VOR (Colebatch et al., 1994a). The vestibulo-collic reflex involves the activation of the two Otolith organs, their corresponding vestibular nerve afferent fibers, the vestibular nuclei, the efferent medial vestibulo-spinal tract, accessory nucleus, the accessory cranial nerve (IX), and the SCM muscle (Rosengren, Welgampola, & Colebatch, 2010). Mudduwa, Kara, Whelan, and Banerjee (2010), in their review of the literature, reported that the vestibulo-collic reflex allows for the stability of the head in space, as it acts directly upon the neck muscles. The presumed disynaptic vestibulo-collic reflex pathway has proven to be complex in nature, and there is still information yet to be learned in regards to the exact anatomical pathway of the afferent, and especially efferent projections (Wilson & Schor, 1999). Figure 12 illustrates a schematic diagram of the presumed vestibulo-collic reflex pathway.

Afferent Pathway

Investigators have stated that the saccule is sensitive to high intensity acoustic stimuli (Townsend & Cody, 1971). McCue and Guinan (1994) proved through animal experimentation on cats that the mammalian saccule is responsive to acoustic stimuli within the frequency and intensity range of human hearing, a response that was measured in the inferior vestibular nerve. Similar results were documented in an animal study of guinea pigs.

In humans, it has been documented that VEMPs can be measured from the SCM muscles in patients with cochlear abolition but not in patients with deafferentation of the vestibular nerve (Colebatch & Halmagyi, 1992; Colebatch et al., 1994a). Halmagyi and Colebatch (1995) recorded VEMPs in patients with absent caloric responses (n=6), and found that the VEMP response was present in some of these patients. This finding indicates that the VEMP response is not dependent on the integrity of the SCCs, but rather appears to have an Otolith origin (Halmagyi & Colebatch, 1995). In an earlier report in the literature, Townsend and Cody (1971) demonstrated the saccular origin of the inion response. More recently, Robertson and Ireland (1995) suggested that the saccule was the generator of the p13-n23 response recorded at the SCM. In support of these findings, Sheykholeslami and Kaga (2002) provided more direct evidence that the acoustically sensitive saccule was responsible for the generation of the vestibulo-collic reflex in their recording of VEMP responses in patients with varying inner ear anomalies (n=7). It is widely known that the inferior vestibular nerve innervates the saccule, projects ipsilaterally, and terminates in the vestibular nuclei (Lysakowski, 2005).

Therefore, it is presumed that the saccule and the inferior vestibular nerve comprise the afferent pathway of the vestibulo-collic reflex pathway.

Efferent Pathway

The anatomical structures contributing to the efferent pathways of the vestibulo-collic reflex are less certain. Robertson & Ireland (1995) reported that the likely efferent pathway contributing to the p13-n23 response involved the lateral vestibular nuclei as well as the lateral vestibulo-spinal tract, which was thought to innervate the SCM muscles. It is likely that the SCM muscles are innervated by the motor neurons of the lateral vestibular nucleus. Lysakowski (2005) reported that the lateral vestibular nuclei were responsible for the generation of the vestibulo-collic reflex, as well as the medial and lateral vestibulo-spinal tracts. This investigator also reported that the medial vestibulo-spinal tract terminated in the cervical region of the spinal cord, and that its function was to stabilize the head in space by producing muscle contractions in the neck to resist reflexive head movements (Lysakowski, 2005).

This function aligns with the aforementioned function of the vestibulo-collic reflex, suggesting that it is the medial, rather than the lateral vestibulo-spinal tract, that is responsible for the efferent projections of the vestibulo-collic reflex pathway. Most schematic diagrams that are documented in the literature since this time (i.e., Akin & Murnane, 2008; Mudduwa et al., 2010) include the medial, rather than the lateral vestibulo-spinal tract in the presumed efferent pathway projecting from the innervating the motoneurons in the SCM muscle. Rosengren et al. (2010) included the accessory nucleus and accessory cranial nerve (XI) in their description of the efferent pathway of

the vestibulo-collic reflex; however, the principal investigator of the current study did not encounter this in the description of the pathway elsewhere in the literature. The response must be recorded from an activated SCM muscle, as the VEMP is an inhibitory response, resulting in the relaxation of the SCM (Brantberg, Bergenius, & Tribukait, 1999; Wilson & Schor, 1999).

History of the VEMP

In 1958, Geisler, Frishkopf, and Rosenblith (1958) were the first investigators to report that a short-latency response occurs in response to presentation of supra-threshold acoustic clicks when electrodes are located on the inion region on the scalp. As shown in Figure 13, these investigators recorded inion responses to click stimuli of several different presentation levels. These levels included a no stimulus condition (baseline) as well as levels close to psychophysical threshold and supra-threshold presentation levels in one subject with normal vestibular function. Geisler and colleagues (1958) found that the amplitude of the inion response was greatest at supra-threshold levels, as indicated by

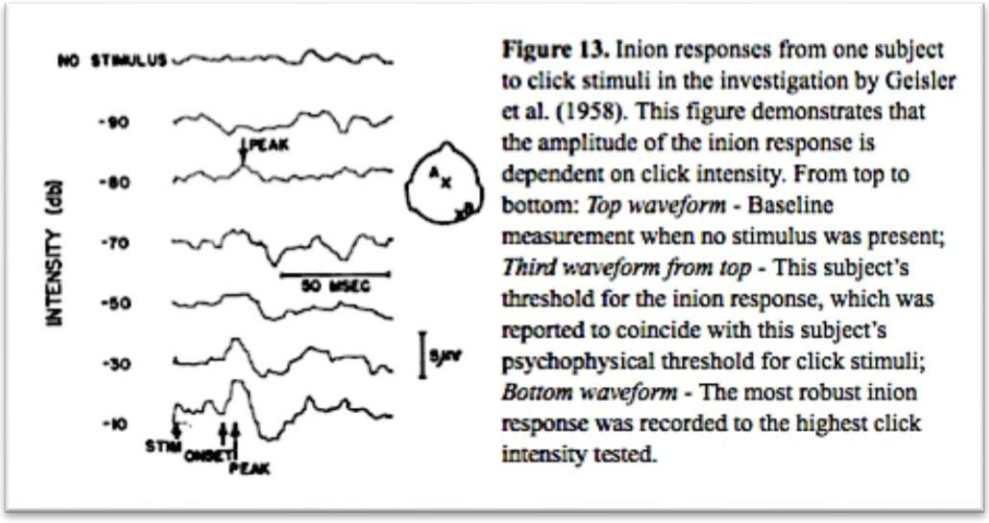


Figure 13. Inion responses from one subject to click stimuli in the investigation by Geisler et al. (1958). This figure demonstrates that the amplitude of the inion response is dependent on click intensity. From top to bottom: *Top waveform* - Baseline measurement when no stimulus was present; *Third waveform from top* - This subject's threshold for the inion response, which was reported to coincide with this subject's psychophysical threshold for click stimuli; *Bottom waveform* - The most robust inion response was recorded to the highest click intensity tested.

the -30 and -10 intensity levels shown in Figure 13. These investigators suggested that the origin of the response was cortical, specifically reflecting activity from the auditory cortex (Geisler et al., 1958).

Several years later, Bickford, Jacobson, and Cody (1964) suggested that this short latency response was actually myogenic in nature, as the amplitude of the response varied with varying neck muscle tension. For example, the subjects included in this report were asked to flex their neck in a forward direction, which increased the amplitude of the response. In contrast, pressing on the forward region of the scalp backwards resulted in relaxed neck muscles and abolished the response. In their report, Bickford and colleagues (1964) also suggested that other extracranial muscles, such as temporal, trapezius, as well as muscles in the arms and legs, showed similar responses (Bickford et al., 1964).

Bickford and his colleagues (1964) were interested in determining whether this myogenic response was cochlear or vestibular in nature, as the response was recorded to acoustic stimulation. These investigators answered this question by studying the properties of this myogenic response in three patients with either cochlear or vestibular lesions. The first patient presented with a complete unilateral sensorineural deafness and normal caloric responses bilaterally. This patient had normal myogenic responses recorded from the inion bilaterally. Since the cochlea was completely impaired on one side, the presence of a myogenic response for this ear was suggestive of a vestibular origin for this response (Bickford et al., 1964).

The second patient presented with complete bilateral sensorineural deafness and normally functioning vestibular labyrinths, bilaterally. Normal myogenic responses were

found bilaterally in this patient. This finding is again indicative of a vestibular origin for the inion response. Lastly, the third patient presented with a complete unilateral sensorineural deafness and loss of vestibular labyrinthine function in one ear, whereas he/she had normal cochlear and vestibular labyrinthine function in the opposite ear. The response was only present in the normally functioning inner ear. Based on these response patterns, Bickford and his colleagues (1964) concluded that "the response to sound-click input is initiated via the vestibular system rather than being cochlear in origin" (p. 213).

In subsequent studies it was discovered that it was possible to record this myogenic potential from electrodes located on the post-auricular muscle (PAM) as well as from electrodes located on the inion (Cody & Bickford, 1969; Townsend & Cody, 1971). These two groups of investigators were interested in determining the differences, if any, between the myogenic responses recorded from electrodes located on the inion versus electrodes located on the postauricular region of the scalp. Cody and Bickford (1969) reported that there was greater variability in the latency measurements for the responses recorded from the postauricular region in comparison to those recorded from the inion region of the scalp. These investigators also reported that the amplitude of the postauricular response was very sensitive to changes in neck tension, and the response detectability was less for responses recorded from the postauricular region versus responses recorded from the inion region of the scalp. Specifically, responses recorded from the postauricular region could only be detected in 68 percent of their 60 normal subjects (p. 404), whereas responses recorded from the inion could be detected in 90 percent of approximately 60 normal subjects (p. 406) (Cody & Bickford, 1969).

Lastly, Townsend and Cody (1971) also compared the inion response and the postauricular response examined in several subjects with various vestibular disorders. The first subject had bilateral vestibular neuronitis and normal hearing sensitivity in both ears. This subject had hypoactive caloric responses, indicating a loss or reduction in vestibular input from both ears. This individual had a normal postauricular response and an absent inion response, suggesting that the postauricular response is cochlear in nature. The second patient had a unilateral vestibular nerve section in the left ear and normal hearing sensitivity and vestibular function in the right ear. For this individual, the inion response was present in the normally functioning right ear but absent in the left ear, which underwent surgery (Townsend & Cody, 1971). This again confirmed the vestibular nature of the inion response.

Lastly, a third subject presented with complete bilateral sensorineural deafness due to labyrinthine otosclerosis. Caloric responses from this subject were borderline hypoactive; however, an inion response was present, but it was small in amplitude. Based on this evidence, Townsend and Cody (1971) concluded that the inion response was mediated by the vestibular system, while the postauricular response was mediated by the cochlea.

In their 1971 study, Townsend and Cody were also interested in trying to determine which anatomical structures in the peripheral vestibular system (i.e., the SCCs or the Otolith organs) were responsible for the inion response. These investigators reported that a subject who had normal hearing but complete absence of caloric responses to ice water stimulation in both ears due to streptomycin toxicity had normal inion responses bilaterally. This finding suggests that the presence of the inion response is

dependent on normal Otolith function rather than normal SCC function. These investigators also reported that in an earlier study by McGee and Olszewski (1962), when streptomycin was introduced into cats, it had a toxic effect on the hair cells in the cristae ampullaris of the SCCs, but only a minor effect, if any, on the hair cells in the macula of the Otolith organs. Collectively, these two pieces of evidence related to the streptomycin toxicity of the SCCs suggest that the inion response is dependent of healthy, intact function of the Otolith organs.

The next question addressed in the literature by investigators was which Otolith organ (i.e. the utricle or the saccule) is responsible for the generation of the inion response (Lindsay, Kohut, & Sciarra, 1967; Townsend & Cody, 1971). Lindsay, Kohut, and Sciarra (1967) conducted a histopathological study in individuals with Meniere's disease. These investigators discovered that the disease, especially in its end stages, had the greatest pathological effect on two anatomical inner ear structures. These two structures were found to be the cochlear duct and the saccule.

Based on these findings, Townsend and Cody (1971) recorded the inion response in patients with Meniere's disease (n=22) to determine whether the response was generated by the utricle or the saccule. A subject with unilateral endolymphatic hydrops in his left ear presented with normal caloric function in both ears. An inion response could only be evoked in the normal right ear. A second subject had bilateral endolymphatic hydrops and normal caloric responses in both ears. Inion responses could not be evoked in either ear of this subject. These three pieces of evidence, which include the response patterns for the inion responses reported in the two case studies by Townsend and Cody, (1971) along with the fact that the saccule is usually damaged in

endolymphatic hydrops while the utricle remains intact, strongly suggest that the saccule is the Otolith organ that is responsible for the generation of the inion response.

Townsend and Cody (1971) were interested in further confirming that the utricle was not responsible for the generation of the inion response. In order to accomplish this goal, the inion response was recorded in six subjects that were diagnosed with benign-paroxysmal positional vertigo (BPPV). Dix and Hallpike (1952) and Cawthorne and Hallpike (1957) had previously reported that in this type of positional vertigo, the SCCs and the saccule tend to remain intact, whereas these patients displayed an absence of the utricular membrane and disorganization of the sensory epithelium is found in the utricle in subjects with BPPV. Schuknecht (1962) also reported that BPPV results from the utricular otoconia becoming dislodged and ending up in the cristae ampullaris of the posterior SCC.

The subjects in the study by Townsend and Cody (1971) who all had been diagnosed with BPPV, all demonstrated normal hearing sensitivity and normal caloric responses. Therefore, it was interpreted that all of these subjects had normally functioning cochleae and horizontal SCCs. These investigators were successful in recording the inion response in five out of the six (or 83%) total subjects. Due to the previous reports suggesting that the genesis of BPPV took place in the utricle, and because most of these subjects had intact inion responses, Townsend and Cody (1971) strongly suggested that the utricle was not responsible for the generation of the inion response.

The suggested saccular origin of the VEMP is proven further through anatomical reasoning. The saccule is located below the stapes footplate, in position to be sensitive to acoustic energy at high stimulus intensities (Zhou & Cox, 2004). Because the VEMP is evoked by the presentation of loud acoustic stimuli, the anatomical location of the saccule only provides further evidence that the saccule is a generator of the response.

Reports regarding this response dwindled in the literature for roughly two decades, until Colebatch and his colleagues revisited the topic in the early to mid-1990s (Colebatch & Halmagyi, 1992; Colebatch et al., 1994a). Colebatch and Halmagyi (1992), in their presentation of a case study, recorded the p13-n23 response with an electrode placed on each SCM in a subject before and after undergoing a voluntary unilateral vestibular nerve section due to symptoms of Meniere's disease. Responses were present in both ears before the sectioning of the vestibular nerve. After the surgical procedure, the recordings from this subject were absent on the side of the nerve section; however, they were still present in the opposite ear (Colebatch & Halmagyi, 1992).

These investigators suggested that short-latency vestibular evoked potentials recorded to high intensity (e.g. 95 or 100 dB HL) click stimuli measured from the SCM could be used to evaluate the functioning of the vestibular afferents originating in the saccule, as well as the vestibulospinal projections from the vestibular nuclei to the neck muscles (Colebatch & Halmagyi, 1992). These investigators concluded that recording of the p13-n23 response from the SCM muscles was relatively simple to achieve and should be employed into routine clinical practice of patients with complaints of hearing difficulty or balance pathologies (Colebatch & Halmagyi, 1992).

Colebatch et al. (1994a) described the first documented account of the clinical VEMP procedure as we practice it today. These investigators reported that consistent VEMP responses to click stimuli could be successfully recorded at symmetrical electrode sites located on the upper half of the SCM muscles in normal adult subjects (n=10). Colebatch and colleagues explained their rationale for choosing the SCM on each side as their recording electrode sites with the reference electrode site over the sternum. They chose these sites over the use of the inion "to allow greater certainty as to the specific muscles likely to be generating the response seen" and "to avoid the uncertainties inevitably associated with the use of a midline recording site when investigating the effects of unilateral stimuli" (Colebatch et al., 1994a, p. 191). Bickford et al., (1964; 1969) as well as Townsend and Cody (1971), had reported that a bilateral inion response could be recorded as a result of unilateral auditory stimulation. Colebatch et al. (1994a) also found that the p13-n23 response was present in some subjects when measured from both SCM muscles (i.e., ipsilateral and contralateral) in response to monaural stimulation using click stimuli, with the ipsilaterally recorded VEMP producing larger p13-n23 amplitudes versus the contralateral response (Colebatch et al., 1994a).

In the 1994 study by Colebatch and colleagues, the p13-n23 response was present bilaterally in all normal subjects, and the amplitude was dependent on the level of tension in the SCM muscles as well as the intensity of the stimulus. Attempts were made to regulate the electromyographic (EMG) activity corresponding with neck tension in each subject. Each subject was instructed to press their foreheads against a padded bar until the desired level of tension was achieved, and they were asked to hold that position for the length of the recording. Even with attempts made to normalize the p13-n23 response in

terms of neck muscle activation, the amplitude of the response still varied widely across normal subjects. For example, when the mean EMG activity was 60 μV, responses to 95 dB click stimuli varied in amplitude from 18.3 to 137.1 μV. The presentation levels used in this study were relative to the average reference threshold for click stimuli (which was approximated to be 45 dB SPL) and were either presented at 95 or 100 dB above the reference (140 and 145 dB SPL, respectively).

Colebatch and colleagues (1994) found that these responses could only be measured at high click intensities, and found that the threshold for these responses ranged from 75 to 80 dB in normal subjects. The mean latency of p13 was 13.3 ms (SD=1.5) and for n23 the mean latency was 22.6 ms (SD=2.4) in twenty normal ears. Colebatch and colleagues (1994) also evaluated patients with complete sensorineural deafness with normal vestibular function. They reported robust p13-n23 responses in these patients. In contrast, patients with voluntary vestibular nerve section presented with absent p13-n23 responses. These findings were consistent with aforementioned studies (Bickford et al., 1964; Cody & Bickford, 1969; Townsend & Cody, 1971). It was also reported that the successful recording of the p13-n23 response requires the absence of a conductive pathology, as it was found that an occlusion of the ear canal or middle ear pathology either attenuated or eliminated the response.

A year later, Robertson and Ireland (1995) coined the term “vestibular evoked myogenic potentials” or VEMP. These investigators sought to re-assess these potentials after the electrode site was modified as established by Colebatch and his colleagues (1992; 1994a). They too recorded the VEMP response in healthy adults free of audio-vestibular dysfunction (n=7). They also recorded robust myogenic responses from SCM

muscles with a positive deflection relative to the baseline at an approximate latency of 13 ms and a negative deflection relative to the baseline at an approximate latency of 23 ms.

Since the reports in the early-to-mid 1990s, VEMP literature has increased in prevalence and has focused on the variations in the normal VEMP response, optimal stimulus and recording parameters to successfully record the VEMP response, as well as various clinical applications of the VEMP assessment. These topics will be discussed in the next section of the literature review.

Response Indices

Latency

The VEMP is a short-latency EMG recording, and the waveform components, as previously mentioned, are named for the polarity of the peak denoted with a lower-case 'p' or 'n,' followed by the approximate latency of the peak in ms. Colebatch and Halmagyi (1992) were the first to describe this 'p13-n23' response in several patients who underwent unilateral vestibular nerve sections. Colebatch et al. (1994a) first described the p13-n23 response in individuals with normal auditory and vestibular function, and indicated that the mean latencies (and standard deviations [SD]) for p13 and n23 were 13.3 ms (SD = 1.5) and 22.6 ms (SD = 2.4), respectively, when elicited with click stimuli at presentation levels of 100 dB nHL. It has been shown that response latency does not significantly vary with differing presentation levels of click stimuli (Akin, Murnane, and Proffitt, 2003; Lim, Clouston, and Sheean, 1995), or with varying tonic EMG level (Lim et al., 1995).

It was reported that the latencies of the VEMP waveform components were dependent on the stimulus type and frequency (Akin & Murnane, 2001). Basta, Todt, and Ernst (2005) assessed response latencies when 500 Hz tone bursts presented at 95 dB HL were used to elicit VEMPs in adult subjects free of remarkable otologic histories (n=64). Mean p13 latencies (and standard deviations) were 16.1 ms (SD = 2.1), and mean n23 response latencies were 23.8 ms (SD = 2.2). These latency values were slightly longer than the latency values obtained from VEMP responses to click stimuli as reported by Colebatch et al (1994a).

The measured latencies to these tone burst stimuli did not show any significant changes with increasing age. Wang et al. (2008), who also used 500 Hz tone bursts in stimulation, determined the normal adult p13 latency range to be 11.9 ms to 15.9 ms and the normal adult n23 latency range to be 18.7 ms to 23.1 ms (n=14). Due to many possible confounding stimulus and/or recording effects, it is important that normative latency values be established in clinics where VEMPs are included as part of the regular vestibular test battery.

Amplitude

A large intersubject variance in VEMP amplitude values has been reported, which has largely been attributed to stimulus intensity and tonic EMG level (Akin et al., 2003; Colebatch et al., 1994a; Lim et al., 1995). In the study put forth by Akin and colleagues (2003), tonic EMG level was controlled for and click stimuli were presented at 100 dB nHL. It was found that p13-n23 amplitudes ranged from 16 to 179 µV, and the mean peak-to-peak amplitude was reported to be 60 µV. Colebatch et al. (1994a) demonstrated

higher response amplitudes with higher levels of stimulation with click stimuli and greater tonic EMG levels.

When response amplitudes were normalized at a 60-µV tonic EMG level and clicks were presented at 95 dB nHL, amplitudes ranged from 18.3 to 137.1 µV in healthy adult subjects (n=10). Mean response amplitudes recorded from the right and left ears of the same subjects were reported to be 88.8 and 81.8 µV, respectively (Colebatch et al., 1994a). It is clear from these findings that even when stimulus intensity and tonic EMG level are controlled for, large intersubject variability can be expected when measuring VEMP amplitude in normal adult subjects.

Lim and colleagues (1995) demonstrated that the synergistic effect of the intensity of the click stimulus and the level of tonic EMG accounted for the majority (approximately 70%) of the variance in VEMP amplitude. These investigators recommended that both stimulus intensity and tonic EMG level should be controlled for when obtaining VEMP recordings due to the documented linear effect of stimulus intensity and tonic EMG level on VEMP amplitudes (Lim et al., 1995).

Welgampola and Colebatch (2001a) reported that when evoked to a 123.5 dB SPL (110 dB HL) 500 Hz tone burst stimulus, the mean VEMP amplitude value was 91.4 µV. In another study, it was reported that the mean peak-to-peak amplitude value of waves p13-n23 was 112 µV in response to a 120 dB peak SPL 500 Hz tone burst stimulus. Janky and Shepard (2009), who recorded VEMP responses to a 500 Hz tone burst stimulus at maximum output of their equipment (i.e., 123 peak SPL), recorded a mean

peak-to-peak amplitude value of p13-n23 at 66.12 µV (SD = 31.49) in their young adult group (ages 20-29).

Response amplitude measurements were taken on VEMPs recorded to 500 Hz tone burst stimuli presented at 95 and 90 dB nHL from the SCM muscles of healthy adult subjects in the current study. Based on the aforementioned literature, it was expected that the normal amplitude range would roughly fall between 15 and 180 µV, with mean response amplitudes ranging from 60 to 80 µV.

Interaural Asymmetry Ratio

Based on the large intersubject variability in terms of amplitude found in the literature, some investigators have examined the use of an interaural asymmetry ratio for amplitude to help distinguish pathological from non-pathological VEMP waveforms. The use of this type of ratio allows for the individual to act as their own control, by comparing amplitudes obtained from SCM muscles on each side of the neck. More specifically, the asymmetry ratio calculated from VEMP waveform amplitudes will assist in the identification of unilateral vestibular lesions that affect the VEMP pathway. The asymmetry ratio is expressed as a percentage and can be calculated by finding the difference between the absolute amplitude measures found on each side (an absolute value), dividing this value by the total amplitude found on each side, and multiplying this value by 100 to express this value as a percent. The equation can be found below.

$$\frac{Ampl.\ of\ greater\ side\ (\mu V) - Ampl.\ of\ smaller\ side\ (\mu V)}{Ampl.\ of\ greater\ side\ (\mu V) + Ampl.\ of\ smaller\ side\ (\mu V)} \times 100 = Asym.\ Ratio\ (\%)$$

Young, Huang, and Chen (2003) noted that the mean asymmetry ratio calculated from recordings in normal subjects (n=10) was 13% (SD= 10), and established the normative cutoff to be 33%. Elsewhere, the mean asymmetry ratio noted in normal adult subjects was reported to be 10.8% (SD = 27.3) (Li, Houldon, & Tomlinson, 1999). Two groups of investigators suggested that the variation of VEMP amplitude between sides should not exceed 30% in most normal adult subjects (Halmagyi, Colebatch, & Curthroys, 1994; Brantberg & Fransson, 2001). The asymmetry ratio has been documented as being a clinically significant response measure in determining the stage of Meniere's disease (Young et al., 2003).

In the current study, the interaural asymmetry amplitude ratio was calculated in each participating subject when evoked to a 500 Hz tone burst stimuli. These mean data assisted in establishing a normal cutoff value for this response index in our clinic.

Threshold

Colebatch and his colleagues (1994a) indicated that the threshold for the p13-n23 response varied between 75 and 85 dB nHL across healthy adult subjects (n=10) when VEMPs to click stimuli were recorded from SCM muscles. Ochi, Ohashi, and Nishino (2001) reported that the mean threshold for their group of normal subjects (n=18) was 87.78 dB (SD = 4.54). Akin and colleagues (2003) found VEMP thresholds to clicks in healthy adult subjects (n=19) ranged between 80 and 100 dB nHL, with the mean threshold at 91 dB nHL (SD=5.2).

It has been reported that normal VEMP thresholds are lower in intensity when evoked with tone bursts rather than click stimuli (Rauch, 2006). Janky and Shepard

(2009) determined VEMP thresholds to click and tone burst stimuli (250, 500, 750 and 1000 Hz) in subjects with normal audio-vestibular function (n=46). They found that the thresholds for VEMPs evoked to click stimuli were significantly higher than the VEMP thresholds reported to 500 Hz tone bursts across subjects aged 20 to 76 years. Overall, subjects had a mean threshold level of 122.17 dB SPL (SD = 4.09) to click stimuli, and 114.16 dB SPL (SD = 6.45) to 500 Hz tone bursts. Several other investigators have also reported similar findings. For example, Akin et al., 2003; Murofushi, Matsuzaki, and Wu, 1999; Rauch, 2006; and Welgampola & Colebatch, 2001a reported that the VEMP response had lower thresholds to tone burst versus click stimuli and the lowest VEMP thresholds were elicited with tone bursts between 500 and 1000 Hz.

Decreased VEMP thresholds (approximately 70 dB nHL) have been used as diagnostic criteria for identifying patients with superior semicircular canal dehiscence (SCD) and other third window syndromes (Brantberg, Bergenius, & Tribukait, 1999; Colebatch, Rothwell, Bronstein, & Ludman, 1994b; Minor, 2005). In the current study, we estimated VEMP threshold on each side of each of the participants. Based on the threshold literature, it was our expectation that our subjects with normal audio-vestibular function would likely have VEMP thresholds to the 500 Hz tone burst stimuli at presentation levels of approximately 75 to 90 dB nHL.

Stimulus Parameters

In order to successfully record the VEMP response, one must understand the optimal stimulus and recording parameters, as well as subject factors used to achieve the short-latency EMG response. Reports in the VEMP literature include various modes of

stimulation to evoke the short-latency VEMP response. However, this section of the literature review will solely focus on the stimulus and recording parameters that were used to collect normative data for our clinic.

The stimulus parameters that will be discussed in the following section include effects of stimulus type, intensity, rate, and duration. The discussion of recording parameters will include the following: filter settings, electrode montage, the necessary number of sweeps or trials to achieve the recording, effects of muscle tension, and the effects of unilateral versus bilateral recording of the VEMP. Subject factors that influence the recording of the VEMP are related to the age and attention or cooperation of the subject.

Stimulus Type: Clicks versus Tone Bursts

Several groups of investigators (Colebatch & Halmagyi, 1992; Colebatch et al., 1994a; Lim, Clouston, & Sheean, 1995; Robertson & Ireland, 1995) have reported in the literature that VEMPs can be evoked with the use of click stimuli in healthy adult vestibular systems. McCue and Guinan (1997) discovered that vestibular responses could be recorded to both click and tonal stimuli in cats. Several groups of investigators (Cheng, Huang, & Young, 2003; Murofushi et al., 1999; Welgampola & Colebatch, 2001a) researched the difference between the uses of click versus tone burst stimuli to evoke the VEMP response. Based on the reports from these investigators, there is some degree of controversy in the literature regarding the optimal stimulus type for eliciting the VEMP response.

Cheng and colleagues (2003) aimed to indicate whether clicks or 500 Hz tone bursts could elicit a higher VEMP response detection rate in normally hearing individuals (n=29). It was found that 98% of subjects had present responses to clicks and 88% had present responses to tone bursts, a difference that was found to be significant. VEMPs evoked by clicks also exhibited larger p13-n23 amplitudes as well as shorter peak latencies. Clicks were deemed superior to tone bursts and were recommended to elicit VEMPs in adults with suspected vestibular disorders (Cheng et al., 2003). These results were inconsistent with those of Akin et al. (2004), who found that 500 Hz tone bursts elicited higher VEMP amplitudes when compared to those elicited with clicks.

Murofushi and colleagues (1999) were interested in determining whether tone bursts, as well as click stimuli, could be used to evoke similar vestibular responses when recorded at electrode sites located on the SCM muscles of normal adult human subjects (n=6). These investigators presented both stimulus types to individuals with vestibulocochlear disorders (n=30; 34 affected ears), to explore the VEMP response pattern that clicks and tone bursts evoked. It was found that the results of the VEMP recorded to tone burst and click stimuli were similar in 88% of the subjects in determining whether the responses were normal or abnormal response (Murofushi et al., 1999). In 4 subjects with complete loss of cochlear function, VEMP responses were present to 500 Hz tone bursts. In contrast, in 4 subjects with decreased or absent caloric response, VEMPs were found to be decreased or absent to the 500 Hz tone burst stimuli. This finding confirms that the response elicited by the tone burst is probably vestibular in origin, similarly to the response elicited by the click (Murofushi et al., 1999).

Welgampola and Colebatch (2001a) also reported that the VEMP responses obtained with clicks and with tone bursts were quite similar, and agreed that the VEMP response obtained with tone bursts assessed the integrity of the same pathway as the response obtained with clicks. It was decided that in this current study, the investigators used a 500 Hz tone burst stimulus to elicit the VEMP response.

Tone Bursts: Optimal Frequency for Stimulation

McCue and Guinan (1997), who first discovered that VEMPs can be evoked to tonal stimuli in cats, reported the most robust VEMP responses were recorded from the vestibular systems of cats to tone bursts with frequencies between 500 and 1000 Hz. Murofushi et al. (1999) elicited VEMPs in human adults with normal auditory and vestibular function to 500, 1000, and 2000 Hz tone bursts to determine which stimulus frequency should be used to evoke the largest VEMP amplitude. It was found that the 500 Hz tone burst elicited the highest VEMP amplitudes, while the 2000 Hz tone burst yielded the responses with the smallest amplitudes. VEMP response waveforms obtained from recordings evoked with tone bursts of different frequencies in normal subjects are shown in Figure 14.

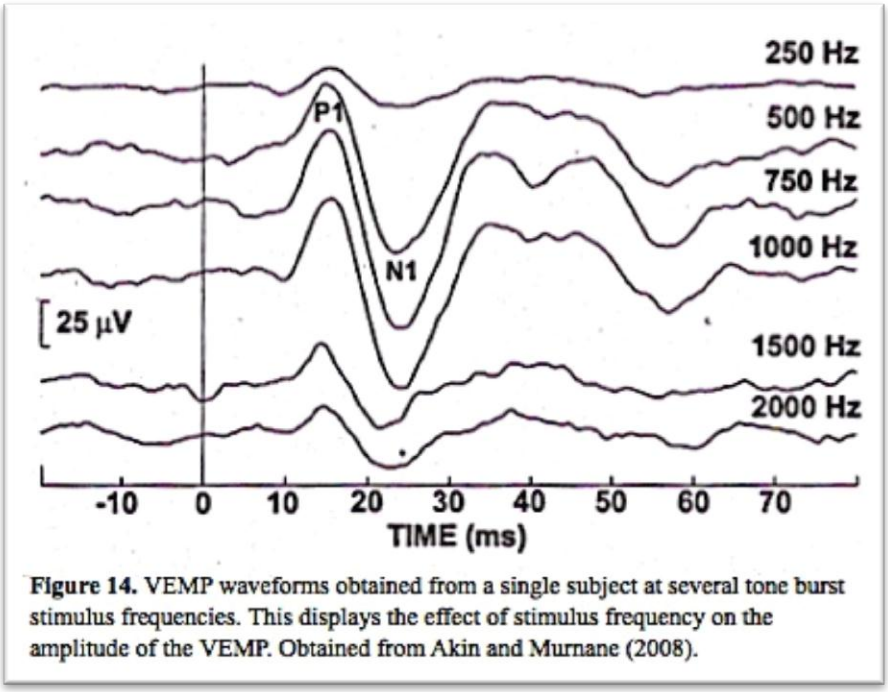


Figure 14. VEMP waveforms obtained from a single subject at several tone burst stimulus frequencies. This displays the effect of stimulus frequency on the amplitude of the VEMP. Obtained from Akin and Murnane (2008).

Welgampola and Colebatch (2001a) reported similar findings: the 500 Hz tone burst elicited the response with the largest amplitude in 7 normal adult subjects, while the 1000 Hz tone burst elicited the VEMP response with the largest amplitude in 5 normal adult subjects. Responses from a selected group of the subjects in their study (n=6) were then elicited with tone bursts from 200 Hz to 1000 Hz in 100 Hz intervals. The results from this study indicate that 700 Hz is the optimal stimulus frequency for eliciting the VEMP using tone bursts. These investigators ultimately recommended the use of both 500 and 1000 Hz tone bursts to ensure a robust response. It was also concluded that there is little evidence indicating differences between responses evoked by clicks versus tone bursts (Welgampola & Colebatch, 2001a).

As previously mentioned, 500 Hz tone burst stimuli were used to evoke the VEMP response in the current study. These tone bursts were used as it was recommended

in the aforementioned literature and allows for straightforward comparison between the current data in the current study and the published normative data.

Stimulus Intensity

Akin and colleagues (2003) report that the intensity of the stimulus has significant effects on the response amplitude of the VEMP, but no significant effects were found on response latency. They determined this by recording the VEMP to click intensities ranging between 80 and 100 dB nHL in 5-dB increments (Akin et al., 2003). The effect of stimulus intensity on the amplitude of the VEMP response has been documented by several groups of researchers (Colebatch et al., 1994a; Lim et al., 1995). Colebatch et al. (1994a) noted that the average normal perceptual threshold for click stimuli was approximately 45 dB SPL. This level was used as the reference (equivalent to 0 dB nHL) for presentation level of click stimuli to obtain the p13-n23 response. This is also the same reference used in presenting click stimuli during auditory brainstem response (ABR) testing. In their study, clicks at intensities as high as 100 dB nHL (145 dB SPL) were administered to each normal subject (n=10) (Colebatch et al., 1994a).

Both groups of investigators (Colebatch et al., 1994a; Lim et al., 1995) noted that the p13-n23 response was only present when evoked by high-intensity click stimuli. It also has been noted that the amplitude of the response increases as a function of click intensity (Akin et al., 2003; Colebatch et al., 1994a; Lim et al., 1995). Mean normative thresholds for the response were found to be between 75 and 80 dB nHL in the Colebatch et al. (1994a) study.

This data was consistent with that documented by Lim and colleagues, who estimated the threshold of this response at 78 dB nHL in normal subjects (n=10). Effects of click intensity on response amplitude appeared to be linear in both studies (Colebatch et al., 1994a; Lim et al., 1995). Response amplitudes were found to be 36% larger when evoked with 100 dB nHL clicks versus with 95 dB nHL clicks in the study by Colebatch and colleagues (1994a), a percentage figure similar to the 30% estimated by Lim and his fellow investigators using similar test parameters (1995). Figure 15 illustrates the effect of stimulus intensity on the response waveform.

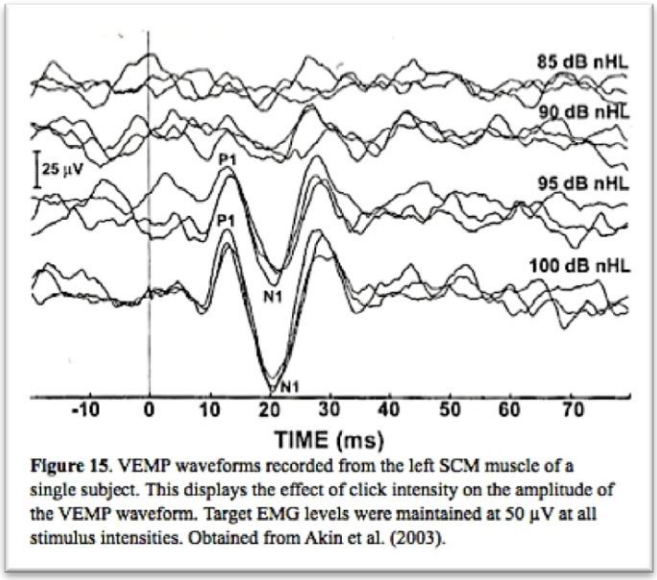


Figure 15. VEMP waveforms recorded from the left SCM muscle of a single subject. This displays the effect of click intensity on the amplitude of the VEMP waveform. Target EMG levels were maintained at 50 µV at all stimulus intensities. Obtained from Akin et al. (2003).

Due to the results of this documented research, the current study presented 500 Hz tone burst stimuli at 95 and 90 dB nHL to elicit robust VEMP waveforms in normal adult subjects. Also, VEMPs were recorded at a presentation level of 70 dB nHL and increasing in 5-dB steps up to 85 dB nHL to determine the VEMP threshold for the right and left ears of each individual subject.

Stimulus Rate

Wu and Murofushi (1999) investigated the effects of click repetition rate on the VEMP in order to select the optimal stimulus rate for clinical use. This investigation included VEMP responses in normal adult subjects (n=12) recorded unilaterally from electrodes located on contracted SCM muscles to 0.1 ms rarefaction click stimuli presented at 95 dB nHL. Click repetition rates used in the experiment were 1, 5, 10, 15, and 20 Hz. At click rates greater than 10 Hz, response detectability decreased. Peak-to-peak amplitudes of the VEMP response generally decreased as repetition rate increased, with the largest amplitudes found at a rate of 5 Hz. Differences in VEMP amplitudes found at the various stimulation rates were not considered to be significant.

The goal of selecting an appropriate stimulus rate is especially important in the recording of the VEMP because of the potential of muscle fatigue. It is important that the amplitude of the response is not sacrificed in the process. Wu and Murofushi (1999) have reported that the 5 Hz click repetition rate is the optimal rate to use in stimulation when recording the VEMP to click stimuli. In addition, Welgampola and Colebatch (2001a) reported that 5 Hz is also an appropriate repetition rate for use with tone bursts. Therefore, in the current study, we used a 5 Hz rate in presenting the 500 Hz tone burst stimuli.

Stimulus Duration

Huang, Su, and Cheng (2005) investigated the effects of the duration of click stimuli on the VEMP waveform. They recorded VEMPs in adults with normal otologic history (n=17) to clicks that were 0.1, 0.2, 0.5, and 1.0 ms in duration to determine the optimal click duration to use when recording the VEMP. Mean VEMP latencies were

significantly prolonged with each increase in click duration. Latency measures of VEMPs recorded to the 1.0 ms click duration were found to have the largest interaural and intersubject variability of all of the click durations employed in the study. VEMP amplitudes were the largest when 0.5 and 1.0 ms click durations were used in stimulation. It was found that the VEMP amplitudes were not significantly different from one another when either 0.5 or 1.0 ms click durations were used in stimulation. Because the 0.5 ms click duration yielded smaller variability in the latency measures of the VEMP recordings, it was considered to be the optimal click duration to use in recording the VEMP.

Welgampola and Colebatch (2001a) investigated the effects of stimulus duration when tone bursts were used to elicit the VEMP. Normal adult subjects (n=12) were assessed using tone bursts of the frequency that elicited the response with the largest amplitude (i.e., 500 or 1000 Hz) in each individual. The effects of tone burst duration were evaluated using tone bursts of 1, 3, 5, 7, 10, and 20 ms in duration. The optimal stimulus duration was found to be 7 ms in 10 subjects and 10 ms in the remaining 2 subjects. Significant differences were found between the response amplitudes found at the 6 stimulus durations that were assessed. Mean p13-n23 latencies were also found to be significantly different between the stimulus durations. Latencies of these peaks generally increased with an increase in stimulus duration, but were most prolonged at 7 ms for p13, and at 10 ms for n23. These investigators recommended the use of a tone burst that is 7 ms in duration in order to achieve an optimal response (Welgampola & Colebatch, 2001a). Based on the evidence, the investigators in the current study used a 7 ms stimulus duration with the use of tone burst stimuli.

Recording Parameters

Unilateral versus Bilateral Recording

In their original report on VEMPs as they are currently practiced, Colebatch et al. (1994a) recorded the VEMP response to high-intensity click stimuli using two different recording conditions in healthy adult subjects (n=10). These were (1) a unilateral recording from the contracted SCM muscle ipsilateral to the stimulated ear; and (2) a bilateral recording of both contracted SCM muscles with both ears being stimulated. Responses recorded in each condition were roughly symmetrical, suggesting that the method of recording the response does not affect the overall response measures for the VEMP.

Several advantages and disadvantages of recording VEMPs to monaural or binaural stimuli have been documented in the literature (Ferber-Viart, Duclaux, Colleaux, & Dubreuil, 1997; Li et al., 1999). Conducting bilateral VEMP recordings to binaural stimulation may be a desired method of recording because it reduces the test session time by half; therefore subjects only have to contract their SCM muscles for half the recording time than required for unilateral stimulation/recording. This reduces the potential risk for muscle fatigue during VEMP testing. A second reason bilateral recordings of VEMPs have also been favored is because amplitudes tend to be greater to binaural stimulation than in recordings elicited with unilateral stimulation with click stimuli (Ferber-Viart et al., 1997).

The primary disadvantage of recording VEMPs bilaterally to bilateral stimulation is that it is susceptible to myogenic or evoked activity crossing the midline (Li et al.,

1999). These investigators reported that this midline myogenic activity could alter the response measures and morphology of the recorded VEMP waveforms or could make it more difficult to determine the side of the vestibular lesion, if one exists (Li et al., 1999).

In contrast to the findings by Ferber-Viart and colleagues (1997), more recently Bhagat (2006) found that VEMP amplitudes recorded from bilateral SCM active electrode recording sites to bilateral stimulation with tone bursts presented at 95 dB nHL were smaller than those obtained when recorded from active electrodes located on the contracted SCM on the ipsilateral side to monaural tone burst stimulation at the same level. This difference may be due to the difference in stimulus type (i.e., click versus tone burst) between the two studies. While monaural stimulation with unilateral recording offers side-specific information, bilateral recording to bilateral stimulation may offer a faster test time, and may be more ideal for patients in which the contraction of the SCM proves difficult or fatigues easily.

In the current study, we solely recorded VEMPs unilaterally to ipsilateral stimulation (right and left). In each of these recording conditions, we recorded the VEMP to 500 Hz tone burst stimuli.

Electrode Montage

Colebatch and his colleagues (1992; 1994a) were the first investigators to report the active electrode site location for the VEMP to be on the SCM muscle. Since these initial reports were published in the literature, several groups of investigators have explored the effects of the active electrode site location on VEMP response characteristics. Ferber-Viart et al. (1997) proposed the recording of VEMPs from

electrodes located on the trapezius muscles, and compared these responses to those obtained on the SCM of the same healthy subjects (n=16). They found significant differences between the amplitudes and latencies of the responses obtained from each muscle. Responses from the trapezius muscle were always significantly longer in latency and significantly greater in amplitude in comparison to the responses recorded from the SCM muscles. Ferber-Viart et al. (1997) suggested that because of the differences in response latency between the measurements obtained from each electrode site, recording from the trapezius muscle assessed the integrity of a different pathway than recording from the SCM muscle.

Ferber-Viart et al. (1997) reported that the response latencies and amplitudes measured from responses obtained from the SCM muscle were consistent with those of Colebatch and his colleagues (1992; 1994a). To allow for ease of comparison between the normative responses obtained in this study and those in the documented literature, the

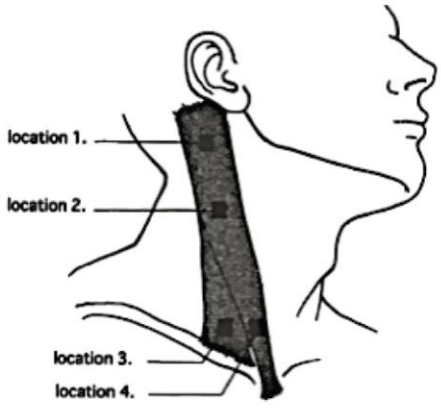


Figure 16. The four electrode locations used to identify the location that produced the most robust VEMP waveforms in the study by Sheykholeslami et al. (2001). Location 2 was found to be the optimal recording location. *Location 1:* the upper part of the SCM muscle at the mandibular angle; *Location 2:* middle part of the SCM muscle; *Location 3:* Directly above the clavicular origin of the SCM muscle; *Location 4:* Directly above the sternal origin of the SCM muscle. Obtained from Sheykholeslami et al. (2001).

active electrode site was located on the SCM muscles in the current study.

The effect of the location of the active electrode site on the SCM muscle on the VEMP has also been studied in normal subjects (n=15) (Sheykholeslami, Murofushi, & Kaga, 2001). Figure 16 shows the four electrode locations that were used in recording VEMPs from each subject in this study. It was found that the mean response amplitudes were highest (227.46 μV) when recorded from the upper portion of the SCM muscle (location 1). However, recording from the middle portion of the SCM muscle (location 2) was ultimately recommended because mean response amplitudes were still high (169 μV), and showed less variation in the data (SD = 116) versus recording from the upper portion of the SCM muscle (SD = 215). The recording at locations 3 and 4 were consistently lower in amplitude versus locations 1 and 2 (Sheykholeslami et al., 2001).

Per the recommendations from Sheykholeslami and colleagues (2001), the active electrodes were located on the middle of each of the SCM muscles of healthy subjects in the current study. Reference electrodes were placed on the upper sternum, and ground electrodes were placed on the forehead. This electrode montage is consistent with those reported in the documented normative VEMP literature (Colebatch et al., 1994a; Robertson & Ireland, 1995; Sheykholeslami et al., 2001).

EMG Bandpass Filter Settings

There is a great deal of variation in the bandpass filter settings used for recording VEMPs in the normative literature. Akin and Murnane (2008) stated that the dominant energy of an EMG signal is found between 40 through 150 Hz; therefore, the bandpass filter should be set using a high-pass cutoff between 5 and 20 Hz and a low-pass cutoff

between 1 and 2 kHz. This recommendation is consistent with the bandpass filter settings used in the normative VEMP literature. Numerous bandpass filter settings from various studies that have successfully recorded VEMPs in healthy individuals can be found in the Table 1. In the current study, a bandpass filter setting of 50-1500 Hz was employed, due to the availability of filter settings that exist in the equipment that was used in recording the VEMP response. This filter setting encompasses the desired EMG signal and is consistent with the literature.

Table 1. Bandpass filter settings from a variety of documented normative VEMP studies.

Study	Bandpass filter setting
Colebatch et al. (1994a)	8 Hz - 1.6 kHz
Robertson & Ireland (1995)	20 Hz – 10 kHz
Basta et al. (2005)	20 Hz – 1.5 kHz
Ferber-Viart et al. (1997)	5 Hz – 1 kHz
Wang et al. (2008)	30 Hz – 3 kHz
Sheykholeslami et al. (2001)	20 Hz – 2 kHz
Welgampola & Colebatch (2001b)	8 Hz – 1.6 kHz

Laterality of VEMPs

There are inconsistencies in the literature when it comes to establishing the laterality of the VEMP. Colebatch and his colleagues (1994a) found that the click-evoked VEMP was largely ipsilaterally dominant in his group of normal adult subjects (n=10). A year later, Robertson and Ireland (1995) documented that in their group of normal adult subjects (n=7), the amplitude of the responses recorded from the ipsilateral and contralateral pathways when elicited with monaural stimulation were approximately

equal, suggesting bilateral involvement in the VEMP pathways. Ferber-Viart et al. (1997) noted that contralateral response amplitudes tended to be greater than ipsilateral response amplitudes to monaural stimulation of high-intensity clicks in healthy adults (n=16).

More recently, the consensus in the research seems to support the ipsilateral nature of the VEMP that was recorded using monaural stimuli. Some researchers believe the response to unilateral stimuli to be purely ipsilateral in nature in healthy adult subjects (Akin & Murnane, 2001; Li, Houldon, and Tomlinson, 1999), and some have found that the response was greater in amplitude on the side ipsilateral to stimulation; however, a response was still present when recorded from the contralateral SCM muscle (Akin et al., 2003; Murofushi, Ochiai, Ozeki, & Iwasaki, 2004). In the current study, used a two-channel recording (ipsilateral and contralateral) for our VEMP recordings to unilateral stimulation to investigate whether differences are found between the waveforms obtained from each SCM muscle.

Number of Trials/Sweeps

It is important to establish an estimate of how many sweeps or averages are needed to obtain a clear VEMP waveform, especially in the light of patient comfort and the potential of SCM muscle fatigability. The amplitude of this myogenic response is much larger relative to the cranial responses obtained during assessments such as the auditory brainstem response; therefore less sweeps/averages would be needed to record clear VEMP waveforms than would be required to obtain a clear ABR waveform. Akin and Murnane (2008) estimated that the amplitude of VEMPs were approximately 60 to 800 times larger than the amplitudes of the ABR components. Refer to Table 2 for a

summary of the number of trials, number of replications, and total number of sweeps included in the averages in several studies where the VEMP has been successfully recorded.

Table 2. Number of sweeps, trials, equating to the total number of sweeps contributing to the averaged VEMP waveforms reported in the normative literature.

Study	# of sweeps per tracing	# of trials	Total # of sweeps contributing to avg.
Akin et al. (2003)	128	3	384
Basta et al. (2005)	130	2	260
Cheng et al. (2003)	128	2	256
Colebatch et al. (1994a)	NR	NR	512
Murofushi et al. (2004)	100	2	200
Sheykholeslami et al. (2001)	200	2	400
Welgampola & Colebatch (2001b)	256	1	256

Note. NR = not reported

In the current study, we set the analysis to run 128 sweeps per trial, and two trials were run for each stimulus condition to ensure the replicability of each response. Subsequent trials were added on an as needed basis if the response proves to not be replicable after two trials. Even so, a total of 256 sweeps was used to contribute to the averaged VEMP waveform for each stimulus condition in each of the included participants. Subjects were allowed resting time in between each trial, in order to prevent fatigue of the SCM muscles.

Analysis Window

Because the prominent peaks of the VEMP occur at approximately 13 to 23 ms, a post-stimulus analysis window beyond this time frame must be used to capture the VEMP. Many studies have used post-stimulus analysis time windows between 50 and 100 ms (Akin et al., 2003; Basta et al., 2005; Colebatch et al., 1994a; Murofushi et al.,

2004; Sheykholeslami et al., 2001; Welgampola & Colebatch, 2001b). Therefore, a post-stimulus analysis time window of 60 ms was used in the current study, as it captured the prominent peaks of the VEMP and is consistent with the literature.

Subject Factors

Tonic EMG level/Subject's Position During Testing

It has already been mentioned briefly in the discussion on response amplitude, that the tonic EMG level, which is related to the strength of the SCM muscle contraction, is highly correlated to response amplitude. Lim et al. (1995) found a significant strong positive correlation between the two variables (r = 0.91-0.94). Akin et al. (2004) explored the effects of tonic EMG level on the unilaterally recorded VEMP response to both clicks and tone bursts presented to high intensities in healthy adult participants (n=11). They recorded VEMPs unilaterally at target EMG levels of 0, 30, 50, 70, and 90 μV in each of the normal adult participants. They found significant coefficients of determination between tonic EMG level and VEMP amplitude values for both click and tone burst stimuli, with VEMP amplitude values increasing with increases of tonic EMG level (r^2=0.98) (Akin et al., 2004). Figure 17 shows the influence of tonic EMG level on the amplitude of VEMP waveforms recorded to both click and tone burst stimuli.

However, at the higher tonic EMG levels, higher variability in the amplitude data was observed. Therefore, these investigators recommended the use of target EMG levels of 30 to 50 µV. Other VEMP studies have documented the use target EMG levels of 50 to 60 µV to successfully record VEMP responses (Akin et al., 2003; Colebatch et al., 1994). These groups of investigators, as previously mentioned, recommended that clinicians and researchers should control for the tonic EMG level in their subjects, as this could influence the VEMP amplitudes obtained in subjects, making it more difficult to ensure that a response should be classified as pathological or non-pathological (Akin et al., 2004; 2003; Lim et al., 1995).

Several methods of normalizing tonic EMG level across subjects have been documented for both the unilateral and bilateral recording of VEMPs. Unilateral recording of VEMPs has largely been achieved by turning the head towards the side contralateral to where the recording is taking place (Akin et al., 2004; Colebatch et al., 1994a). This contracts the SCM muscle ipsilateral to the ear that is being stimulated. Akin and Murnane (2008) reported that because a portion of the clinical interpretation of

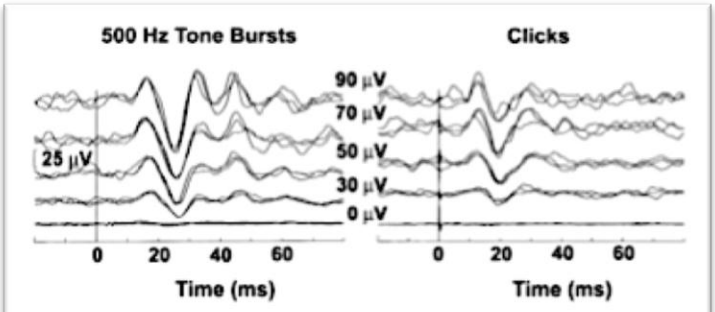


Figure 17. VEMP waveforms obtained from a single subject recorded to both 500 Hz tone bursts (left) and click stimuli (right) at varying target EMG levels (indicated in center of figure). This illustrates the effect of target EMG level of the VEMP waveform amplitudes. Obtained from Akin et al. (2004).

the unilaterally recorded VEMP involves the interaural amplitude comparison of VEMP responses, differences in tonic EMG level of the SCM muscles between the sides need to be regulated. This should be considered in order to ensure accurate interpretation of the interaural amplitude comparisons (Akin & Murnane, 2008).

Colebatch and his colleagues (1994a), in their successful bilateral recording of the VEMP, included two methods for achieving bilateral SCM muscle contraction in their normal subjects. These were (1) pressing their forehead against a bar in front of them; and (2) lifting their heads against gravity while in a supine position. Welgampola and Colebatch (2001b) also successfully recorded VEMP responses using the second bilateral method in normal individuals (n=70).

Another issue in the tonic EMG level of the contracted SCM muscles is the manner in which the level of the contraction is regulated or monitored. Investigators have most frequently used some form of visual feedback method in order for subjects to maintain target EMG levels (Akin & Murnane, 2001; Akin et al., 2003; Colebatch et al., 1994; Ferber-Viart et al., 1997; Li et al., 1999; Welgampola & Colebatch, 2005). These visual feedback systems have consisted of stand-alone differential electrodes recorded from the SCM muscles to commercially available systems (Akin et al., 2004; 2003; Li et al., 1999), pressing the jaw against a rubber ball connected to an EMG level recording device (Ferber-Viart et al., 1997) and pressing the chin against a blood pressure manometer (Vanspauwen, Wuyts, & Van de Heyning, 2006). Vanspauwen et al. (2006) reported that more reliable VEMP responses were obtained when visual feedback was provided for normal subjects (n=10).

A commercially available EMG monitoring system may not be available in some clinics that offer VEMP testing, as it adds an extra cost to clinics that would most likely only use it for VEMP testing. The amount of VEMP assessments performed may not outweigh the cost of the equipment, and for this reason a more practical method should be developed. Patients undergoing VEMP testing at a local clinic are instructed hold a tennis ball between their chin and chest and the tonic EMG level of the SCM muscle on each side is monitored by visual examination by clinicians (S. Pallett, personal communication, 2011). The symmetry of the SCM contractions on each side is an important factor in assessing VEMP responses, as previously mentioned; therefore, as long as the SCM muscle contractions appear to be equivalent on each side, this may offer a more practical method to monitor EMG level in audiological clinics who regularly assess VEMPs in patients with suspected vestibular lesions.

The current investigators monitored the tonic EMG level using a commercially available EMG monitoring system (Delsys Bagnoli 2 EMG monitoring system). Subjects made natural unilateral SCM muscle contractions and an EMG target level was measured based on this natural level of the contraction. With use of visual feedback on the computer monitor, subjects were asked to maintain this tonic EMG level within 10% of the EMG target level during each recording trial. This was conducted to ensure that tonic EMG levels were not contributing to differences in amplitude across recording trials and from each side of the participant. In contrast to the aforementioned studies in the literature, a constant EMG level was not used across all subjects. As previously mentioned, target EMG levels were based on the tonic EMG levels of individual SCM contraction.

Age-related Factors

Su, Huang, Young, and Cheng (2004) explored the effects of age on the response parameters of the VEMP using click stimuli to evoke the responses. Significantly prolonged latencies were found for component n23 as age increased in a group of healthy individuals (n=80) of varying ages. Latencies of the p13 component tended to be prolonged, but the effect was not significant (Su et al., 2004). Lee, Cha, Jung, Park, and Yeo (2008), who assessed age-related changes in VEMP parameters in participants aged 12 to 77 years when evoked by click stimuli presented at 95 dB nHL (n=97), found significant increases in latency of both the p13 and n23 peaks when age groups above 60 years were compared with each of the younger age groups.

Response amplitudes have been found to be significantly decreased in the subjects above age 60 years in comparison to younger subjects, causing a decrease in VEMP response rate (Su et al., 2004). Welgampola and Colebatch (2001b) found clearly decreased amplitudes in normal adult subjects aged between 50 and 60 years and older in comparison with younger age groups (n=70) when elicited with click stimuli. Lee et al. (2008) found significant differences between the 10 to 19 year age group and any of the 10-year age groups beginning at age 30 to 39 years.

Increases in VEMP threshold with increases in age were also documented in the literature (Janky & Shepard, 2009; Welgampola & Colebatch, 2001b). Welgampola and Colebatch (2001b) reported that in their group of normal subjects, mean VEMP thresholds to click stimuli increased from 85 dB nHL between participants aged 20 and 29 years to 96 dB nHL in participants aged 70 and older. In contrast, Janky and Shepard

(2009) did not find significant increases in VEMP threshold with increasing age to click stimuli; however, there were significant positive correlations between age and VEMP threshold when elicited to tone bursts at 250, 500, 750, and 1000 Hz.

Su and colleagues (2004) recommended establishing normative data according to age group in any clinic where the VEMP is a part of regular clinical practice. Lee et al. (2008) suggested establishing different normative data for individuals younger than those below 60 years old and those above 60 years old. The individuals included in the current study were limited to young adults (i.e., between 20 and 30 years) with normal audio-vestibular function. Thus, age-related effects on the VEMP are not expected.

Subject State

Because the tonic contraction of the SCM muscles is required to successfully record VEMPs, it is necessary that subjects undergoing this assessment be awake and cooperative when asked to contract their SCM muscles during recording analysis time.

Conductive Hearing Loss

It has been reported that the presence of conductive hearing loss due to the dysfunction of the middle ear can abolish or attenuate the VEMP (Bath, Harris, McEwan, & Yardley; Colebatch et al., 1994a; Rosengren et al., 2010; Welgampola & Colebatch, 2001b). Bath et al. (1999) indicated that air-bone gaps as small as 9 dB HL and as large as 40 dB HL resulted in absent VEMP responses in individuals with no history of audio-vestibular dysfunction apart from middle ear pathologies (n=32). Sheykholeslami, Murofushi, Kermany, and Kaga (2000) reported that VEMP responses could be elicited through bone conduction in individuals with conductive hearing loss as a result of middle

ear dysfunction. All stimuli in the current study were transmitted through air conduction via ER-3A (Etymotics Research) insert earphones. Therefore, any individual that had presented with abnormal tympanometric results and/or absent ipsilateral acoustic reflexes would have been excluded from the current study. All of our subjects demonstrated both normal tympanometric and acoustic reflex results.

Clinical Applications of the VEMP

In the current study, the participants included individuals with no history of auditory or vestibular pathologies in order to establish normative absolute latency values for waves p13 and n23, peak-to-peak amplitude of p13-n23, interaural asymmetry ratio for amplitude and VEMP threshold data for our clinic. While it is important for every clinic with the VEMP included in their vestibular test battery to establish normative data specific to their set of protocols and equipment, the VEMP is not typically assessed in individuals with normal audio-vestibular function. Typically, VEMPs are ordered for patients with suspected vestibular pathologies. It is important for clinicians and researchers to be aware of the clinical populations in which VEMP parameters can be used as diagnostic criteria. A brief discussion of several vestibular pathologies in which the VEMP assists in the diagnosis will ensue.

Third Window Syndrome or Tullio's Phenomenon

Patients with superior semicircular canal dehiscence (SCD) experience high intensity sound-induced vertigo and oscillopsia (Tullio's phenomenon) (Bronstein, Faldon, Rothwell, Gresty, Colebatch, and Ludman, 1995; Minor, Cremer, Carey, Della Santina, Streubel, & Weg, 2001). Symptoms of vertigo and oscillopsia can also be

induced by pressure changes, either intracranial or middle ear, and also can be evoked with Valsalva maneuvers. Patients also experience autophony. Specifically, patients with SCD have reported that they can hear the motion of their eyes or their pulse (i.e., pulsatile tinnitus) (Minor et al., 2001), the sound of their own voice louder is in the dehiscent ear, and lastly that the sound of their own feet when they are running is louder in the dehiscent ear (Minor, 2005). The clinical manifestation of SCD has been noted to be heterogeneous in nature, with a majority of patients reporting vestibular manifestations and fewer patients reporting solely auditory symptoms. Minor (2005) reported that of 65 subjects with diagnosed SCD, 60 (92%) of them experienced vestibular manifestations of the inner ear pathology, and 5 (7%) of them experienced auditory manifestations only.

Audiologic testing in these patients typically reveals an air-bone gap, indicating an apparent middle-ear conductive involvement, of at least 10 dB and tends to be greatest in the low frequencies of the dehiscent ear (Minor, 2005). However, immittance testing in these patients typically shows normal middle ear function, as demonstrated by normal tympanograms and intact acoustic reflexes. This suggests that the conduction of sound through the middle ear space cannot explain the present air-bone gaps in these subjects. Further, it has been reported that in these patients, a demonstration of hypersensitivity to stimuli elicited by bone conduction typically occurs (Minor, 2005; Minor et al., 2001). For example, audiometric testing in a case study reported by Minor (2005) indicated bone conduction thresholds occurring at -5 and -10 dB HL at 250 and 500 Hz with air conduction thresholds occurring at 30 and 10 dB HL at these respective frequencies.

The aforementioned symptoms and clinical findings were reported to be a result of a dehiscence, or thinning, of the bony wall overlaying the superior SCC, which creates

a third mobile window and decreases the impedance to sound or pressure in the inner ear (Brantberg et al., 1999; Minor, 2005; Minor, Solomon, Zinreich, & Zee, 1998; Minor et al., 2001). Diagnosis of SCD has included the report of the above-mentioned symptoms, hypersensitivity of bone conduction to sound, presence of a vertical-torsional nystagmus on plane with the superior SCC (can be induced with Valsalva maneuvers), and by visualization of the dehiscence on CT (Computerized Tomography) to scan of the temporal bone (Minor et al., 2001). Recently, investigators have noted that VEMPs can be of diagnostic significance in patients with otherwise unexplained air-bone gaps and characteristic audio-vestibular symptoms (Brantberg et al., 1999; Minor, 2005; Minor et al., 2001; Watson, Halmagyi, & Colebatch, 2000).

Colebatch et al. (1994b) found abnormally lower VEMP thresholds (i.e., $\leq$ 70 dB nHL) to clicks in a patient experiencing Tullio's phenomenon of unknown etiology when compared to normal subjects (n=25). The case study presented by Bronstein et al. (1995) offered consistent results in a patient with Tullio's phenomenon also of unknown etiology.

After SCD was first documented in a report by Minor and colleagues (1998), Brantberg et al. (1998) assessed VEMPs in patients with SCD (n=3), and found similar pathologically low VEMP thresholds to tone burst stimuli. Minor (2005) proved this evidence further as he observed pathologically reduced VEMP thresholds in a larger sample of subjects with SCD (n=51 ears) in comparison to normal ears (n = 30 ears). The mean VEMP threshold to clicks for the SCD group was 81 dB nHL (SD=9), while normal controls exhibited a mean threshold of 98 dB nHL (SD=4) (Minor, 2005). In their review of the literature (1994-2006), Akin and Murnane (2008) noted that of 64 patients with SCD, all (100%) demonstrated decreased VEMP thresholds. Augmented VEMP

amplitudes have also been reported in this clinical population (Bronstein et al., 1995; Mudduwa, Kara, Whelan, & Banerjee, 2010).

Other pathologies that result in the experience of Tullio's phenomenon include a fistula of the horizontal SCC, perilymph fistula and Meniere's disease (Watson et al., 2000). Pathologically reduced VEMP thresholds have been reported in both patients with a perilymph fistula (Modugno, Magnani, Brandolini, Savastio, & Pirodda, 2006), and Meniere's disease (Akin & Murnane, 2008). All patients with vestibular symptoms should undergo VEMP testing to assess the integrity of the vestibulo-collic reflex pathway; however, the VEMP has been shown to be of diagnostic significance in identifying SCD and other pathologies that result in the experience of sound- or pressure-induced vertigo or oscillopsia.

These patients typically exhibit VEMP thresholds between 55 and 70 dB nHL (Mudduwa et al., 2010). Because reduced VEMP thresholds are a highly sensitive measure in diagnosis of SCD and other third-window syndromes, the VEMP should be used clinically with patients who report the aforementioned symptoms associated with this pathology and present with the characteristic audiometric data.

Meniere's Disease

Meniere's disease, a pathology within the inner ear structures, has been classified by the following classic symptoms: tinnitus, episodic rotatory vertigo, low-frequency hearing loss that tends to fluctuate in the early stages of the disease, becoming more stable with longer duration of the disease, and symptoms of aural fullness (de Waele, Tran Ba Huy, Diard, Freyss, & Vidal, 1999; Rauch, Zhou, Kujawa, Guinan, & Herrmann,

2004). It has been documented that patients with Meniere's disease suffer from cochleo-saccular hydrops (Lindsay et al., 1967; Rauch et al., 2004). Young et al. (2003) reported that in Meniere's patients, endolymphatic hydrops most frequently occurred in the cochlea, and secondly in the adjacent saccule. Meniere's disease can cause hair cell degeneration in the saccule, dilatation of the saccular membrane (Hong, Yeo, Kim, & Cha, 2008), and in the later stages of the disease, collapse of the saccular membrane on the saccular sensory epithelium, leading to its atrophy (Welgampola & Colebatch, 2005; Young et al., 2003).

Because VEMP testing assesses the integrity of the vestibulo-collic pathway, which depends on the proper functioning of the saccule, it is not surprising that VEMP responses would be altered in patients with Meniere's disease. The clinical interpretation of the VEMP in Meniere's disease has yielded controversy in the literature.

The heterogeneous nature of the clinical manifestation of Meniere's disease was illustrated in the review of the literature (1994-2006) offered by Akin and Murnane (2008), which summarized findings from VEMPs recorded in individuals with Meniere's disease (n = 320). Among those subjects, 63 of them (19.6%) had absent VEMP waveforms, 39 (12.1%) had decreased VEMP thresholds, and 37 (11.5%) had altered VEMP frequency tuning. These were the most common clinical manifestations of Meniere's disease in the VEMP responses; however, it was reported that several other subjects presented with increased VEMP amplitudes, decreased VEMP amplitudes, and increased response latency. In roughly half of the subjects with Meniere's disease (n=162), normal VEMP waveforms were found (Akin & Murnane, 2008) Given that this was a review of the literature, which only included the ways that the presence of

Meniere's disease manifests on the VEMP response, test results from other assessments were not included.

In attempts to identify the most sensitive VEMP measures to assist in the diagnosis of Meniere's disease, several groups of investigators have presented their observations from VEMP assessments of patients with Meniere's disease, with suggestions as to how to best identify the presence of the disease by assessing the patient using the VEMP. It has been reported elsewhere that the incidence of absent VEMPs in patients with Meniere's disease ranged from 18 to 54% of documented cases (de Waele et al., 1999; Murofushi, Shimizu, Takegoshi, & Cheng, 2001; Young et al., 2003). The incidence of abnormal VEMP waveforms in patients with Meniere's disease was reported to be as high as 69%, in a study in which investigators used the latency of VEMP components p13 and n23 and the interaural asymmetry ratio to explore response patterns in patients with vestibular neuritis (n=49), BPPV (n=16), and Meniere's disease (n=20) (Hong et al., 2008). These investigators concluded that the VEMP parameters, especially the interaural asymmetry ratio, were the most sensitive to the assessment of Meniere's disease of the three pathologies discussed (Hong et al., 2008).

Young and colleagues (2003) proposed the use of the VEMP in assessing which stage of the Meniere's disease the patient had based on the severity of their hearing loss (n=40). These investigators found that there were significant increases in the asymmetry ratio as the stage of the Meniere's disease progressed, suggesting that the use of this parameter, along with audiometric testing, can aid in the evaluation of the staging of Meniere's disease in an individual (Young et al., 2003). In the early stages of the disease,

when hearing thresholds and vertiginous symptoms are fluctuant in nature, pathologically increased asymmetry ratios were found (Young, Wu, & Wu, 2002; Young et al., 2003).

These large asymmetry ratios are due to increased VEMP amplitudes on the Meniere's-affected side (Young et al., 2002). The dilation of the saccular membrane, which extends to reach the stapes footplate in some hydropic ears, which results in the saccule being hypersensitive to sound, can explain the augmented VEMP amplitudes on the pathological side. This augmented VEMP recorded from the pathological side in patients in the early stages of Meniere's disease could be used to indicate that the saccular membrane has expanded and established contact with the stapes footplate (Young et al., 2002). Increased asymmetry ratios were also found in another study in Meniere's patients (n=29), with an incidence of 70% in these patients (Hong et al., 2008). In support of the findings documented by Young et al. (2002), the increased VEMP asymmetry ratio was more prevalent in younger Meniere's patients, whose experience with the disease was shorter in comparison to patients who had experienced the disease for a longer period of time (i.e., earlier staging of Meniere's disease).

Altered frequency tuning of the VEMP in Meniere's patients was also documented in the literature (Kim-Lee, Ahn, Kim, & Yoon, 2009; Rauch et al., 2004). Rauch et al. (2004) hypothesized that because of the altered mechanical function of the saccule in Meniere's patients with saccular hydrops, VEMPs recorded in these individuals would be altered in terms of their frequency tuning and threshold characteristics. Their findings from VEMPs recorded to tone burst stimuli (250, 500, 1000, 2000, and 4000 Hz) in Meniere's patients (n=34) and normal adult controls (n=14) support their proposed hypothesis (Rauch et al., 2004).

In this study, frequency tuning referred to the stimulus frequency that evoked the VEMP response at the lowest threshold level. Specifically, Rauch et al. (2004) reported that while the control group showed the best frequency tuning when the VEMP was recorded to 500 Hz tone bursts, Meniere's subjects showed altered frequency tuning, with the lowest thresholds recorded with 1000 Hz tone bursts. They noted that similarly altered tuning was found in the non-pathological side in unilateral Meniere's patients; however, this effect was less obvious in comparison to the pathological side. These investigators suggested that if the intention of distinguishing Meniere's disease from non-pathological recordings is the goal of VEMP testing, then it should be recorded to 500 Hz tone burst stimuli, because at this frequency the largest differences in threshold between these two groups were found (Rauch et al., 2004).

As a result of the findings from Rauch and his fellow investigators, Kim-Lee et al. (2009) recommended the use of a frequency peak amplitude (FPA) ratio with amplitudes measured from recordings to 1000 Hz tone bursts and 500 Hz tone bursts (i.e., 1/0.5 kHz FPA) as a diagnostic criteria for Meniere's disease. These FPA ratio values were significantly higher in patients with diagnosed Meniere's disease (n=26) versus in normal individuals (n=20), and a normative upper cutoff value was established at 0.7 (or 70%) when using this response measure as a diagnostic criterion (Kim-Lee et al., 2009).

As demonstrated in the aforementioned literature, the clinical interpretation of the VEMP waveform in individuals with Meniere's disease can manifest in a variety of the response parameters, and also can change with the fluctuant and progressive nature of the disease. In summary, absent/attenuated waveforms, augmented waveforms, abnormal

interaural asymmetry ratios, increased latencies, and altered frequency tuning have been found in VEMP recordings in Meniere's subjects.

Other Pathologies with Clinical Significance for VEMP testing

VEMP assessment has shown clinical significance in patients with other pathologies than the two previously mentioned. The following disorders have demonstrated VEMP abnormalities: BPPV, vestibular neuritis, cerebello-pontine angle tumors, noise-induced hearing loss, conductive hearing loss, disorders of the central nervous system such as multiple sclerosis, gentamycin therapy, and vestibular schwannomas (Akin & Murnane, 2008; Mudduwa et al., 2010).

It has been shown that most pathologies that tend to result in several possible VEMP response patterns, and VEMP response patterns could possibly be a result of several different pathologies (Akin & Murnane, 2008). Specifically, absent VEMP responses or increased VEMP amplitudes have been observed in patients with Meniere's disease, vestibular schwannomas, vestibular neuritis, sensorineural hearing loss and multiple sclerosis. Decreased VEMP amplitudes have been observed in patients with Meniere's disease and sensorineural hearing loss (Akin & Murnane, 2008). It has been noted that prolonged VEMP latencies, especially of p13, may be indicative of lesions in the vestibulospinal tract (Murofushi et al., 2001). Many patients with multiple sclerosis exhibit prolonged VEMP latencies (Akin & Murnane, 2008). Decreased VEMP thresholds have been found in patients with Meniere's disease, SCD, and other pathologies that result in Tullio's phenomenon (Akin & Murnane, 2008).

Due to the heterogeneous nature of the VEMP response patterns in certain audio-vestibular disorders, it is necessary that the VEMP test is not the only assessment used in the diagnosis of these pathologies. Case history, comprehensive audiometry, other vestibular assessments and possible radiographic studies (i.e., CT scan or MRI [Magnetic Resonance Imaging]) should be used in conjunction with VEMP assessment in making a clinical diagnosis.

Goal of Current Study

The primary goal of the current study was to establish normative data for the various response indices of the VEMP response so that this type of vestibular assessment could be offered at the Speech-Language Hearing Center at Towson University. A discussion of the specific response indices that were explored in the current study will follow in the methods section.

CHAPTER 3

METHODS

Methodology of Current Study

Subjects

Eighteen healthy adult participants were included in the current study. They were recruited via word-of-mouth and flyers located on campus (See Appendix A for flyer). The age range for the included participants was 20 to 30 years, with a mean age of 24 years (SD = 2.22). A total of 11 female subjects and 7 male subjects were included in the subject sample. For inclusion in the current study, it was necessary that participants meet the following three criteria: (1) normal mobility of both tympanic membranes, (2) intact ipsilateral acoustic reflexes, and (3) an unremarkable self-reported vestibular history bilaterally. All 18 subjects provided written informed consent for participation in the current study (See Appendix B for informed consent form).

In order to qualify for inclusion in the current study, the participants were required to demonstrate normal (Jerger Type A) tympanograms bilaterally, including a peak pressure values between ±149 daPa, static compliance values between 0.3 and 1.4 ml, and ear canal volumes between 0.5 and 1.6 ml (Jerger, 1970). Finally, the included participants were all required to demonstrate unremarkable vestibular histories through completion of our case history form (as seen in Appendix C). Otoscopy was also performed to ensure that visual inspection of the ear canal and tympanic membrane in each ear was unremarkable. Subjects were recruited via word of mouth and flyers displayed across campus.

Stimuli

A 500 Hz tone burst stimulus was used to assess the VEMP response. These tone burst stimuli were generated by the Intelligent Hearing Systems (IHS) Smart EP evoked potential system and delivered monaurally to the ears of each subject via ER-3A insert earphones. These stimuli were presented at presentation levels ranging from 70 dB nHL to 95 dB nHL. The sequence of presentation levels will be explained in a later portion of this methods section. The 500 Hz tone burst were presented at a stimulus rate of 5 Hz, as this rate was noted to be the optimal rate for VEMP recording (Welgampola & Colebatch, 2001a). The stimulus duration of the 500 Hz tone burst stimuli was 7 ms (Welgampola & Colebatch, 2001a), as this was determined in the literature to be the optimal stimulus duration for this type of stimulus.

Recording Parameters

Active electrodes were placed on the middle third of each SCM muscle in all participants. The reference electrode was located on the upper sternum, and the ground electrode was located on the forehead (Fpz). The skin at each of the electrode sites was prepared prior to electrode placement. Gold cup recording electrodes were secured to each electrode site using conductive paste and medical tape. All electrode impedance values were measured prior to recording the VEMP. Electrode impedances were all kept at $\leq$ 3000 Ohms.

The VEMP responses were all recorded to monaural stimulation of 500 Hz tone bursts to either the right or left ear. Two-channel recordings were employed in order to simultaneously record from the active electrode located on the contracted SCM muscle

ipsilateral to stimulation (ipsilateral recording) as well as the active electrode located on the non-contracted SCM muscle contralateral to stimulation (contralateral recording). This was true for VEMP recordings on both sides. A further discussion of the procedure associated with contracting the SCM muscle will follow in a later portion of this methods section.

The bandpass EMG filter setting was set at 50 to 1500 Hz as this filter setting encompasses myogenic responses (Akin & Murnane, 2008). The equipment gain setting was set to amplify by a factor of 50,000 (50 K). A total of 128 sweeps were completed during each trial. At least two replicable VEMP responses were recorded at each of the stimulus intensities. A total of 256 sweeps contributed to the average for each recording condition. The post-stimulus analysis window was set from 0 to 60 ms, as it encompassed the latency of the VEMP response.

Procedure for Screening for Inclusion in the Current Study

Otoscopy was performed to ensure that visual inspection of the ear canal and tympanic membrane in each test ear was unremarkable. This was true for all subjects. Tympanometry using a 226 Hz probe tone was performed to ensure normal mobility of the tympanic membrane of each test ear. This was achieved for 17 subjects and one ear of the remaining subject. In this case, the static compliance value was measured to be 1.7 ml in the subject's left ear. This tympanometric result was judged to be normal for this individual and she remained to be included as a participant in the current study.

Ipsilateral acoustic reflexes were assessed at 500, 1000, and 2000 Hz in each ear. All 18 subjects demonstrated present acoustic reflexes in both ears. The vast majority of

our subjects had acoustic reflex thresholds that were within the 90th percentile for individuals with normal hearing (Gelfand, Schwander, & Silman, 1990). These acoustic immittance measures were part of our inclusion criteria in order to ensure that there was no conductive pathology present in any of our subjects. A case history form with questions related to the participant's vestibular history was completed and checked for remarkable vestibular history prior to the test session. A self-reported unremarkable vestibular history was demonstrated for all 18 participants.

VEMP Procedure

Each subject was seen for one test session. Before each test session began, a subject number was randomly assigned and one of the sides of the neck was chosen as the ipsilateral condition to record the first set of VEMP recordings. This selection was alternated between each subject and roughly half of these 18 subjects began their recording on the right side of the neck and the remaining subjects began their recording on the left side of the neck.

For example, if the right side was chosen to be recorded first, the first half of the VEMP recordings consisted of the subject contracting their right SCM muscle. During these active SCM muscle contractions, the 500 Hz tone burst stimuli in this example would have been delivered to the right ear. VEMP responses were then recorded from an active electrode located on the contracted SCM on the right side of the neck, thus yielding the ipsilateral VEMP recording. The VEMP response was also recorded from the active electrode located on the non-contracted SCM muscle on the left side of the neck,

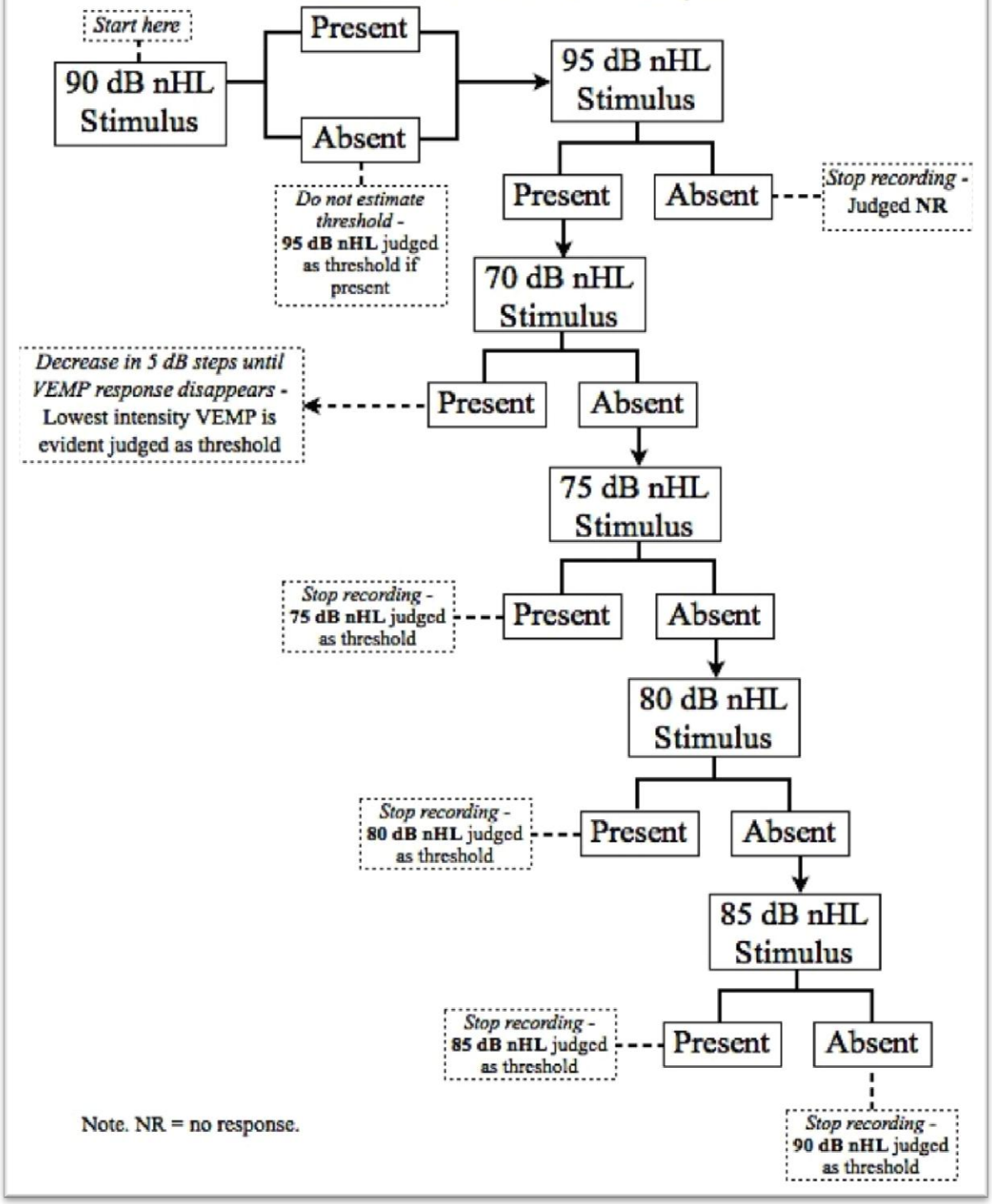
thus yielding the contralateral VEMP response. When these recordings from the right side of the neck were completed, the same procedure was employed for the left side.

For the recording of the VEMP responses on each side, various stimulus intensities were used to collect normative data at supra-threshold levels (i.e., 95 and 90 dB nHL) and to estimate each subject's VEMP thresholds. At these supra-threshold levels, the first intensity at which the 500 Hz tone bursts were presented was always 90 dB nHL. At least two replicable VEMP responses were obtained at this presentation level. The stimulus intensity was increased to 95 dB nHL and again at least one to two replicable VEMP responses were recorded. We followed this supra-threshold test protocol for all 18 subjects.

In order to estimate VEMP threshold for each side of these subjects, the stimulus intensity was then reduced to 70 dB nHL. If the VEMP response was judged to be replicable and present at this lower presentation level, the stimulus intensity was reduced in 5 dB steps until the VEMP response disappeared. None of our subjects had replicable VEMP responses at 70 dB nHL. In contrast, if the VEMP response was absent at 70 dB nHL, the presentation level of the 500 Hz tone burst stimuli was increased in 5 dB steps until a replicable response was evident. The lowest intensity that the VEMP response was judged as replicable and present was considered to be the VEMP threshold.

Once the VEMP responses at the two supra-threshold levels were obtained as well as VEMP threshold data for the first test ear, these same procedures were used to test the opposite ear. A schematic flowchart of these test procedures is found in Figure 18.

Figure 18. A flowchart illustrating the procedure employed to record the VEMP response to a 500 Hz tone burst in each ear for each of the 18 subjects.



Note. NR = no response.

Test Set-Up

All testing was completed in an Industrial Acoustics Company (IAC) double-walled audiometric test booth in the Towson University Speech, Language and Hearing Center located in Towson, Maryland. One of the investigators of this study was positioned on the tester side of the booth and operated the IHS system that recorded the VEMP responses. The other investigator was positioned in the patient side of the booth next to the participant, and operated the EMG monitoring system equipment (a further description of this will follow). The talk-back/talk-forward system configuration on the Madsen Astera audiometer allowed the two investigators and the participant to communicate throughout the test session, which was a necessity in the current study.

Test Instructions

After each participant was set up for recording, they were instructed on how to contract their SCM muscles. The participants sat in a recliner chair and were placed in the maximal reclining position. Instructions were modified based on which SCM was to be recorded from first. For example, to record the VEMP response from the right side, the subject was asked to lift their head off of the recliner and turn toward the left side, naturally contracting the SCM muscle on the right side of the neck. During this first active SCM muscle contraction, the VEMP responses were recorded to 90 dB nHL 500 Hz tone burst stimuli from the active electrode located on the contracted SCM muscle on the right side (ipsilateral recording) and from the active electrode located on the non-contracted SCM muscle on the left side of the neck (contralateral recording).

The subjects were asked to maintain the level of their SCM contraction until the stimulus stopped and then immediately to relax their SCM muscle. A more detailed description of how each subject maintained their SCM muscle contraction will follow in a later portion of this methods section. These set of instructions applied for all test conditions.

Timeframe of VEMP Recordings/Test Session

In between each test condition, a rest break of approximately one minute was given to each participant to prevent fatigue of the SCM muscles. If necessary, additional rest time was provided to each subject. Each recording trial consisted of 128 sweeps presented at a stimulus rate of 5 Hz. Therefore, the recording time for each trial was approximately 25.6 seconds. The recording time for each stimulus intensity varied depending on the number of trials needed to obtain two replicable VEMP responses. In some subjects, four to five trials were needed reach this goal. This obviously extended the overall recording time.

The overall recording time (excluding test set-up and rest breaks) was, at minimum, approximately 10 minutes. When test set-up and rest breaks were included, each VEMP test session took approximately 30 to 45 minutes. Tympanometry, acoustic reflex testing, and filling out the case history form were additional to this time frame. Each participant participated in one test session that lasted approximately one hour.

Recording of Tonic EMG Level of the SCM Muscle Contractions

It has been previously stated that the VEMP response is successfully recorded from electrodes located on a contracted SCM muscle (e.g. Colebatch et al., 1994a; Akin

& Murnane, 2008; Akin et al., 2004). In the current study, the investigators were interested in exploring the tonic EMG level of this muscle contraction, and thus the Delsys Bagnoli 2 EMG monitoring system was used to record and monitor this EMG activity. Akin and colleagues reported that successful VEMP recordings were achieved when subjects demonstrated tonic EMG levels ranging from 30 µV to 90 µV (e.g. Akin & Murnane, 2001; Akin et al., 2004). Therefore, Akin and colleagues have suggested maintaining a constant tonic EMG level of approximately 50 to 60 µV during VEMP recordings.

However, during our collection of pilot data, the current investigators discovered that successful VEMP recordings could not be obtained with tonic EMG levels within this reported 30-µV to 90-µV range. On five adult pilot subjects with a negative history of vestibular pathology, our tonic EMG levels were considerably larger (i.e., in one pilot subject, tonic EMG levels reached 750 µV) than these previously reported values and were quite variable across the 18 subjects. Therefore, based on this pilot data, the current investigators made the decision to measure the tonic EMG level of each subject's natural SCM contraction at the beginning of the VEMP test session and to use this level as their individual target EMG level.

Each subject was then asked to maintain this tonic EMG level for the remainder of their VEMP recordings. The Delsys system was used to ensure that each subject maintained their individual target EMG level (± 10%) during each VEMP recording. The subjects were asked to visually observe their actual tonic EMG level in comparison with their target EMG level. When necessary, the investigator who was operating the EMG

monitoring system instructed the subject to contract their SCM muscle more or less to maintain the target EMG level.

During each trial of the VEMP assessment, the tonic EMG level of the contracted SCM muscle was monitored using the Delsys EMG monitoring system equipment. This was done for the VEMP recordings from both the right and left sides of the neck. This EMG activity was recorded from an active sensor electrode, which was located directly inferior to the VEMP active recording electrode on the contracted SCM muscle. A reference electrode for this EMG monitoring system was placed on the back of each participant's left hand. A one-channel recording set-up was then used to record the tonic EMG level.

Response Measurements for VEMP and EMG Recordings

As previously reported, two replications (256 sweeps) contributed to the average VEMP waveform for each test condition. Several VEMP response indices were measured from each averaged waveform. These included the absolute latency values of waves p13 and n23 (in ms), the peak-to-peak amplitude values of p13-n23 (in µV) and the interaural asymmetry ratio values for amplitude (in %). To calculate the interaural asymmetry ratio, amplitude values from the waveforms obtained for each side were inserted into the following equation:

$$\frac{Ampl.\ of\ greater\ side\ (\mu V) - Ampl.\ of\ smaller\ side\ (\mu V)}{Ampl.\ of\ greater\ side\ (\mu V) + Ampl.\ of\ smaller\ side (\mu V)} \times 100 = Asym.Ratio\ (\%)$$

These response measurements were taken on the VEMP response waveforms in each ear at both supra-threshold presentation levels. VEMP thresholds (in dB nHL) were also

determined for each ear of all subjects. The detectability rate of the VEMP response in the ipsilateral and contralateral recording channels was explored to investigate the laterality of the VEMP response. Tonic EMG levels of the SCM muscle contractions were also explored and are discussed in further detail in the results section of the current study.

Statistical Analyses

Descriptive Statistics

Mean, standard deviation, median, and range values were calculated and reported for each of the previously mentioned response indices obtained to 500 Hz tone burst stimuli presented at 95 and 90 dB nHL in both ears of all subjects. These descriptive statistics are reported separately for each ear at each of the supra-threshold stimulus intensity. The response detection rate was calculated also separately for the 95 and 90 dB nHL responses. This response detection rate, expressed as a percentage, was calculated to investigate the laterality of the VEMP response.

Inferential Statistics

A series of one-way ANOVAs (n=7) were conducted to determine whether or not a significant ear effect existed for the various VEMP response indices. Each response index was evaluated separately. The response measures evaluated included the mean latency values of waves p13 and n23, the mean peak-to-peak amplitude values of p13-n23, and the VEMP threshold. These one-way ANOVAs were calculated separately for the 95 and 90 dB nHL responses, with the exception of the VEMP threshold values. The

within-subjects factor in each of these ANOVAs was the response for the right ear versus the response from the left ear.

If no significant ear effect was obtained for this specific response index, the data for this response index were then collapsed (averaged) across ears. Then, a second series of one-way ANOVAs (n=4) were performed to determine if a significant effect of stimulus intensity existed in this collapsed (averaged) data for this response index. The within-subject factor in this set of ANOVAs was the response at 95 dB nHL versus the response at 90 dB nHL. An alpha level of $p < 0.05$ was used to indicate statistical significance for all of the ANOVAs.

CHAPTER 4

RESULTS

VEMP responses were elicited to 500 Hz tone bursts presented at 95 and 90 dB nHL in eighteen subjects with normal vestibular function. All VEMP response measures were only taken from the summed (averaged) ipsilateral recordings at both stimulus intensities. In the contralateral recording, the investigators of the current study only judged the presence or absence of replicable VEMP responses at both stimulus intensities. All eighteen subjects had present VEMP responses bilaterally to the 95 dB nHL 500 Hz tone burst stimulus. However, only fifteen subjects had present VEMP responses bilaterally when a 90 dB nHL stimulus was used. Figure 19 displays a sample VEMP waveform from which the various response measures were measured. This VEMP waveform was obtained from the right ear of subject 2391 and was recorded to a 95 dB nHL 500 Hz tone burst stimulus. This subject's VEMP data is representative of what was seen in the VEMP recordings across the remaining 17 subjects.

The ipsilateral waveforms (stimulus delivered to the right ear and active electrode recorded from the contracted SCM muscle on the right side of the neck) are located in the top portion of Figure 19 (labeled 1 and 2). In contrast, the contralateral recordings (stimulus delivered to the right ear and active electrode recorded from the non-contracted SCM muscle on the left side of the neck) are located on the lower portion of Figure 19. The odd-numbered tracings (i.e., 1 and 3) represent the summed (averaged) VEMP waveform (total of 256 sweeps in each of these tracings), while the even-numbered tracings (i.e., 2 and 4) display the individual trials (trials 1 and 2) that contributed to the

summed (averaged) waveforms. These individual trials were averaged over a total of 128 sweeps per trial.

The response measures that were investigated included: (1) the absolute latencies of waves p13 and n23, (2) the peak-to-peak amplitude values of waves p13-n23, (3) the interaural asymmetry ratio for amplitude, and (4) the VEMP threshold. The laterality of the VEMP response and the level of the tonic EMG contraction of the SCM muscles were also explored. The organization of the results and discussion sections will discuss the findings for each one of these VEMP response measures in the sequence mentioned above. Below is a description of the individual as well as group findings for the absolute

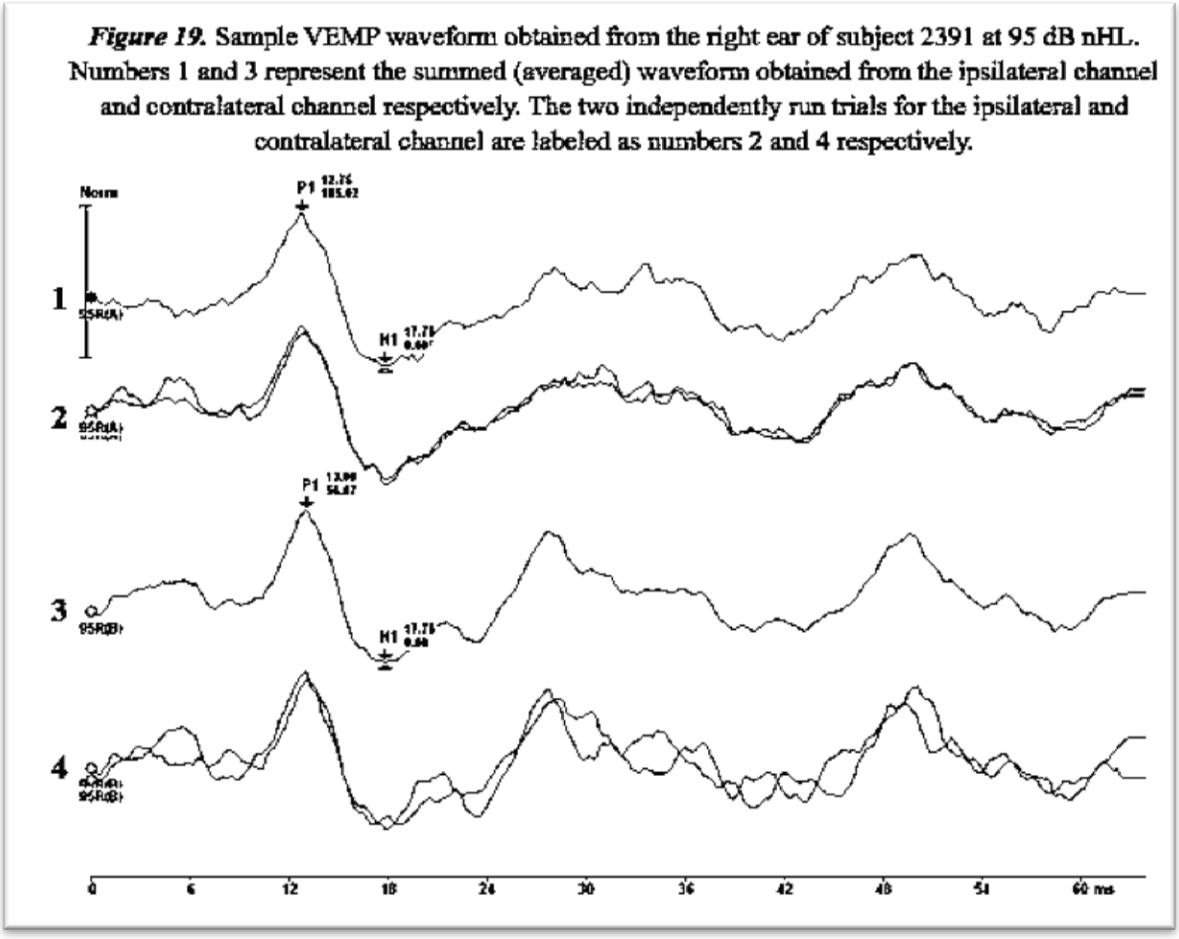


Figure 19. **Sample VEMP waveform obtained from the right ear of subject 2391 at 95 dB nHL. Numbers 1 and 3 represent the summed (averaged) waveform obtained from the ipsilateral channel and contralateral channel respectively. The two independently run trials for the ipsilateral and contralateral channel are labeled as numbers 2 and 4 respectively.**

latency values for waves p13 and n23.

Absolute Latency Value for Waves p13 and n23

The individual subjects' latency measurements for p13 and n23 at both stimulus intensities are displayed in Table 3 with a summary of their descriptive statistics. There were no obvious differences in the mean p13 latency measurements from the right versus left ears at each stimulus intensity. For example, at 95 dB nHL the mean p13 latency values were 13.59 ms and 13.40 ms for the right and left ears, respectively. The variance in the data, reflected in the standard deviation values, was similar across ears at both stimulus intensities (i.e., 1.22 and 1.24 for right versus left ear respectively at 95 dB nHL; and 1.29 and 1.38 for right versus left ear respectively at 90 dB nHL). There was minimal difference in central tendency of the data, as reflected in the median values, and was also similar between ears at both stimulus intensities. For example, the median values at 95 dB nHL were 13.32 ms and 13.13 ms in the right and left ears respectively. Similar ranges of p13 latency values were obtained between ears at both stimulus intensities. For example, at 95 dB nHL, the range of p13 latency values were 12.50-17.25 ms in the right ear and 10.13-15.25 ms in the right ear.

Again, there were no apparent differences in the mean n23 latency measurements between ears at either stimulus intensity For example, at 95 dB nHL, mean n23 latency values were 19.02 ms in the right ear and 19.31 ms in the left. At 90 dB nHL, the mean n23 latency values were 18.79 ms and 19.62 ms in the right and left ears respectively. The variance in the data, as noted in the standard deviation values, was similar between ears at both stimulus intensities. For example, the standard deviation values for n23

latency were 2.46 in the right ear and 2.12 in the left at 95 dB nHL. Similar values were reported at 90 dB nHL.

The central tendency, as reflected in the median values, was also similar across ears. For example, at 95 dB nHL, the median values were 18.69 ms in the right ear and 18.50 ms in the left. This finding was true at both stimulus intensities. Similarly to the findings for wave p13, the ranges of the absolute latency data for wave n23 were similar in the right and left ears. For example, at 95 dB nHL, n23 latency values ranged from 15.88-25.88 ms in the right ear and 16.25-23.13 ms in the left. Again, similar ranges were calculated at 90 dB nHL.

A series of one-way ANOVAs (n=4) were calculated to determine if there were significant ear effects for the p13 and n23 latency measurements. The results for each of the VEMP waveform components (i.e., waves p13 and n23) as well as the results at each of the supra-threshold stimulus intensities were evaluated separately. As expected, there were no significant ear effects for the mean latencies of wave p13 at 95 dB nHL [$F(1, 34) = 0.224$ ($p=0.639$)] or at 90 dB nHL [$F(1,28) = 0.246$, ($p=0.623$)]. No significant ear effects were also found for the mean latency values of wave n23 at 95 dB nHL [$F(1,34) = 0.145$, ($p = 0.705$)] or at 90 dB nHL [$F(1, 28) = 0.000$, ($p = 0.986$)].

Given that no statistically significant differences were found in the latency measurements between ears for either wave p13 or for wave n23, these data were then collapsed (averaged) across ear. These data were collapsed (averaged) for each VEMP component and for each of the stimulus intensities. These averaged data are reported in Table 4. Two additional one-way ANOVAs were performed in order to determine if there

was a statistically significant effect of stimulus intensity on this collapsed (averaged) data. Each of the VEMP waveform components was evaluated separately.

The results of these ANOVAs revealed no statistically significant difference between p13 latencies at 95 versus 90 dB nHL [F (1, 28) = 0.001, (p=0.977)]. Similarly, there was no significant effect of stimulus intensity for the n23 latency values [F (1, 28) = 0.000, (p = 0.986)]. One interesting pattern noted in the individual data related to stimulus intensity was the difference in response detectability at each of these stimulus intensities, which will be discussed in a later section of the results.

Table 3. Absolute Latency of waves p13 and n23 in milliseconds (ms) for 95 and 90 dB nHL presentation levels for 18 individual subjects. Three subjects did not have present VEMP responses in at least one ear at 90 dB nHL, and thus a no response (NR) is indicated for these individuals.

Subject #	Absolute Latency of p13				Absolute Latency of n23			
	95 dB nHL		90 dB nHL		95 dB nHL		90 dB nHL	
	Right	Left	Right	Left	Right	Left	Right	Left
3663	14.13	13.00	13.88	12.88	21.50	17.88	20.63	18.50
2569*	14.38	13.63	15.63	NR	19.25	18.50	19.13	NR
6471	14.75	14.63	14.25	14.75	25.88	22.75	23.00	22.88
1598	12.75	12.38	12.50	13.00	20.13	21.38	19.75	23.13
4216*	13.63	13.75	14.13	NR	16.38	16.25	17.38	NR
5157	12.75	13.13	13.63	12.38	15.88	19.88	16.63	20.63
8012	12.63	12.63	13.13	10.75	19.00	18.25	19.13	17.75
5391	13.75	15.25	12.88	15.38	20.75	21.63	20.50	22.13
4777	12.75	12.5	12.50	12.38	16.75	16.50	15.88	17.13
4519	12.50	10.13	13.25	13.25	19.50	18.38	18.00	18.00
6300	13.13	13.00	13.75	12.38	17.38	18.50	15.50	19.38
8388	12.75	13.13	12.63	12.88	16.63	18.5	17.00	19.50
2391	12.75	12.63	12.38	12.25	17.75	17.63	18.13	17.00
7825	15.13	13.75	13.25	13.75	18.38	18.50	18.50	18.13
4412*	13.63	13.63	NR	NR	19.88	23.13	NR	NR
8850	12.50	13.00	12.88	12.38	17.63	17.13	17.88	16.88
4326	17.25	14.63	17.63	14.50	21.88	21.13	22.88	21.50
5698	13.50	15.00	14.00	16.00	17.75	21.63	18.38	21.75
Mean	13.59	13.40	13.50	13.26	19.02	19.31	18.79	19.62
SD	1.22	1.24	1.29	1.38	2.46	2.12	2.25	2.21
Median	13.32	13.13	13.25	12.88	18.69	18.50	18.38	19.38
Range	12.50-17.25	10.13-15.25	12.38-17.63	10.75-17.63	15.88-25.88	16.25-23.13	15.50-23.00	16.88-23.13
n	18	18	15	15	18	18	15	15

Note. * = subject did not have a detectable VEMP response in at least one ear at 90 dB nHL.
NR = no response

Table 4. Descriptive statistics for collapsed (averaged) data of absolute latency measurements(ms) for waves p13 and n23 at both stimulus intensities.

	p13		n23	
	95 dB nHL	90 dB nHL	95 dB nHL	90 dB nHL
Mean	13.39	13.38	19.22	19.20
SD	1.16	1.11	2.03	1.96
Range	11.32-15.94	11.94-16.07	16.63-24.32	16.51-22.94
n	15	15	15	15

Peak-to-peak Amplitude Value of Wave p13-n23

The individual subjects' peak-to-peak amplitude measurements for wave p13-n23 at both supra-threshold stimulus intensities are displayed in Table 5 with a summary of the descriptive statistics on these response measures. There were no obvious differences in the mean p13-n23 amplitude measurements from the right versus left ears at both stimulus intensities. This is especially true for the mean amplitude values for the 90 dB nHL response, which were 48.98 µV for the right ear and 47.36 µV for the left ear. The variance in the data, reflected in the standard deviation values, was relatively similar across ears. For example, the standard deviation values were 31.75 for the right ear and 20.19 for the left in the 90 dB nHL responses.

The central tendency of the data, reflected in the median values was also similar between ears at both stimulus intensities. For example, for the 95 dB nHL response, the median values were 54.84 µV for the right ear and 53.49 µV for the left ear. Relatively larger ranges of p13-n23 peak-to-peak amplitude values (i.e., ranges of approximately 60-180 µV) were obtained for each ear at both stimulus intensities. It should be noted that as

expected, the variability in the amplitude data was considerably greater versus the latency data.

A series of one-way ANOVAs (n=2) were calculated to determine if significant ear effects existed for the p13-n23 peak-to-peak amplitude measurements. The amplitude results for each of the supra-threshold stimulus intensities were evaluated separately. There was no significant ear effect for the p13-n23 amplitude values at 95 dB nHL [F (1, 34) = 0.486 (p=0.491)]. Similarly, the p13-n23 amplitude values at 90 dB nHL were not significantly different between ears [F (1, 28) = 0.028, (p=0.869)].

Given that no statistically significant differences were found between p13-n23 amplitude measurements between ears, this data was collapsed (averaged) across ear. These averaged data for each of the stimulus intensities is shown in Table 6. An additional one-way ANOVA was performed in order to determine if there was a statistically significant effect of stimulus intensity on this collapsed (averaged) data. The results of the ANOVA revealed that the mean p13-n23 amplitude at 95 dB nHL (77.18 µV) is significantly larger than the mean p13-n23 amplitude at 90 dB nHL (48.17 µV) [F (1, 28) = 5.973, (p=0.021)]. A related amplitude measure that is calculated on the VEMP data is the interaural asymmetry ratio for amplitude, which is explored in the following section.

Table 5. Peak-to-peak amplitude measurements for waves p13-n23 in microvolt (μV) for 95 and 90 dB nHL presentation levels for eighteen individual subjects. Three subjects did not have present VEMP responses in at least one ear at 90 dB nHL, and thus a no response (NR) is indicated for these individuals.

Subject #	Peak-to-peak amplitude of p13-n23			
	95 dB nHL		90 dB nHL	
	Right	Left	Right	Left
3663	24.43	45.34	21.18	30.70
2569*	53.24	30.79	30.54	NR
6471	133.71	114.99	65.67	67.33
1598	44.90	40.49	30.93	23.14
4216*	6.79	3.25	2.94	NR
5157	11.58	38.72	13.40	29.62
8012	35.23	53.09	57.87	52.25
5391	38.93	42.37	27.81	13.37
4777	34.92	63.54	20.83	28.38
4519	89.89	49.06	46.63	46.75
6300	145.64	93.34	45.63	36.41
8388	76.17	111.48	33.34	76.72
2391	185.02	103.23	131.46	52.06
7825	59.44	83.76	31.26	52.40
4412*	12.62	9.92	NR	NR
8850	127.62	108.69	97.42	75.03
4326	56.43	53.88	42.59	50.94
5698	177.91	71.55	68.61	75.31
Mean	73.03	62.08	48.98	47.36
SD	57.21	34.13	31.75	20.19
Median	54.84	53.49	42.59	50.94
Range	6.79-185.02	3.25-114.99	13.40-131.46	13.37-76.72
n	18	18	15	15

Note. * = subject did not have a detectable VEMP response in at least one ear at 90 dB nHL. NR = no response

Table 6. Descriptive statistics for collapsed (averaged) data of peak-to-peak amplitude measurements (μV) for wave p13-n23 at both stimulus intensities.

	p13-n23	
	95 dB nHL	90 dB nHL
Mean	77.18	48.17
SD	39.80	23.00
Range	25.15-144.13	20.59-91.76
n	15	15

Interaural Asymmetry Ratio Value for Amplitude

Interaural asymmetry ratios for amplitude were calculated for each individual subject at both stimulus intensities. The results are displayed in Table 7 along with a summary of the associated descriptive statistics. There was no apparent difference in the mean interaural asymmetry ratio values at each of the supra-threshold stimulus intensities (i.e., 21.01% for the 95 dB nHL response versus 18.21% for the 90 dB nHL response). The variability in the data, as reflected in the standard deviation values, was similar across stimulus intensities. The median value at 95 dB nHL was slightly larger in comparison to the median value at 90 dB nHL. There were also no obvious differences in the range of the interaural asymmetry ratios found between the responses at each of the stimulus intensities.

In order to compare interaural asymmetry ratios for amplitude between the responses at each of the supra-threshold stimulus intensities, only the 15 subjects with calculable asymmetry ratios at both 95 and 90 dB nHL could be included in this analysis. The three subjects that were not included in the analysis were subjects 2569, 4216, and 4412. The descriptive statistics for this group of 15 subjects are displayed in Table 8.

Table 7. Interaural amplitude asymmetry ratios in percent (%) for 95 and 90 dB nHL for eighteen individual subjects. Three subjects did not have present VEMP responses in at least one ear at 90 dB nHL, and thus a no response (NR) is indicated for these individuals.

Subject #	Asymmetry Ratio	
	95 dB nHL	90 dB nHL
3663	29.97	18.35
2569*	26.72	NR
6471	7.53	1.24
1598	5.16	14.41
4216*	35.26	NR
5157	53.96	37.70
8012	20.22	5.10
5391	4.23	35.07
4777	29.07	15.34
4519	29.38	0.12
6300	21.88	11.24
8388	18.82	39.41
2391	28.37	43.27
7825	16.98	25.27
4412*	11.98	NR
8850	8.01	12.98
4326	2.31	8.93
5698	42.64	4.66
Mean	21.01	18.21
SD	14.33	14.51
Median	21.05	14.41
Range	2.12-53.96	0.12-43.27
n	18	15

Note. * = subject did not have a detectable VEMP response in at least one ear at 90 dB nHL. NR = no response

Table 8. Descriptive statistics for interaural asymmetry ratios (%) at both stimulus intensities for fifteen subjects with asymmetry ratios found at both 95 and 90 dB nHL.

	Asymmetry Ratio	
	95 dB nHL	90 dB nHL
Mean	21.01	18.21
SD	15.08	14.51
Median	20.22	14.41
Range	2.12-53.96	0.12-43.27
n	15	15

A one-way ANOVA was performed to determine whether there was a significant effect of stimulus intensity on the mean interaural asymmetry ratio measurements between the responses at 95 and 90 dB nHL. This ANOVA was performed on the data for these 15 subjects. Results of the ANOVA revealed no significant effect of stimulus intensity on these mean interaural asymmetry ratio values [$F(1, 28) = 0.03$ ($p = 0.776$)].

VEMP Threshold

Figure 20 displays an illustration to display how the VEMP threshold was determined for subject 2391. For this specific example, the averaged VEMP waveforms obtained at 90, 85, and 80 dB nHL from the right ear of this subject are displayed. In this case, the VEMP threshold was judged to be 85 dB nHL, as no replicable VEMP responses were evident at 80 dB nHL. The same procedure for judging VEMP thresholds was employed across subjects.

VEMP thresholds for each individual subject for the 500 Hz tone burst are displayed in Table 9, along with their associated descriptive statistics. Mean VEMP threshold values were very similar between ears (i.e., 83.61 dB nHL for the right ear and 84.72 dB nHL in the left ear). The variability in the data, reflected in the standard

deviation values, was also similar between the right versus left ears. The median values were exactly the same in the right and left ears. The ranges of VEMP thresholds were also exactly the same for each ear.

A one-way ANOVA was calculated to determine whether a significant ear effect existed in the VEMP threshold data. As expected, results of the ANOVA showed no significant difference in the VEMP threshold values for the right versus left ears [F (1, 34) = 0.339, (p=0.564)].

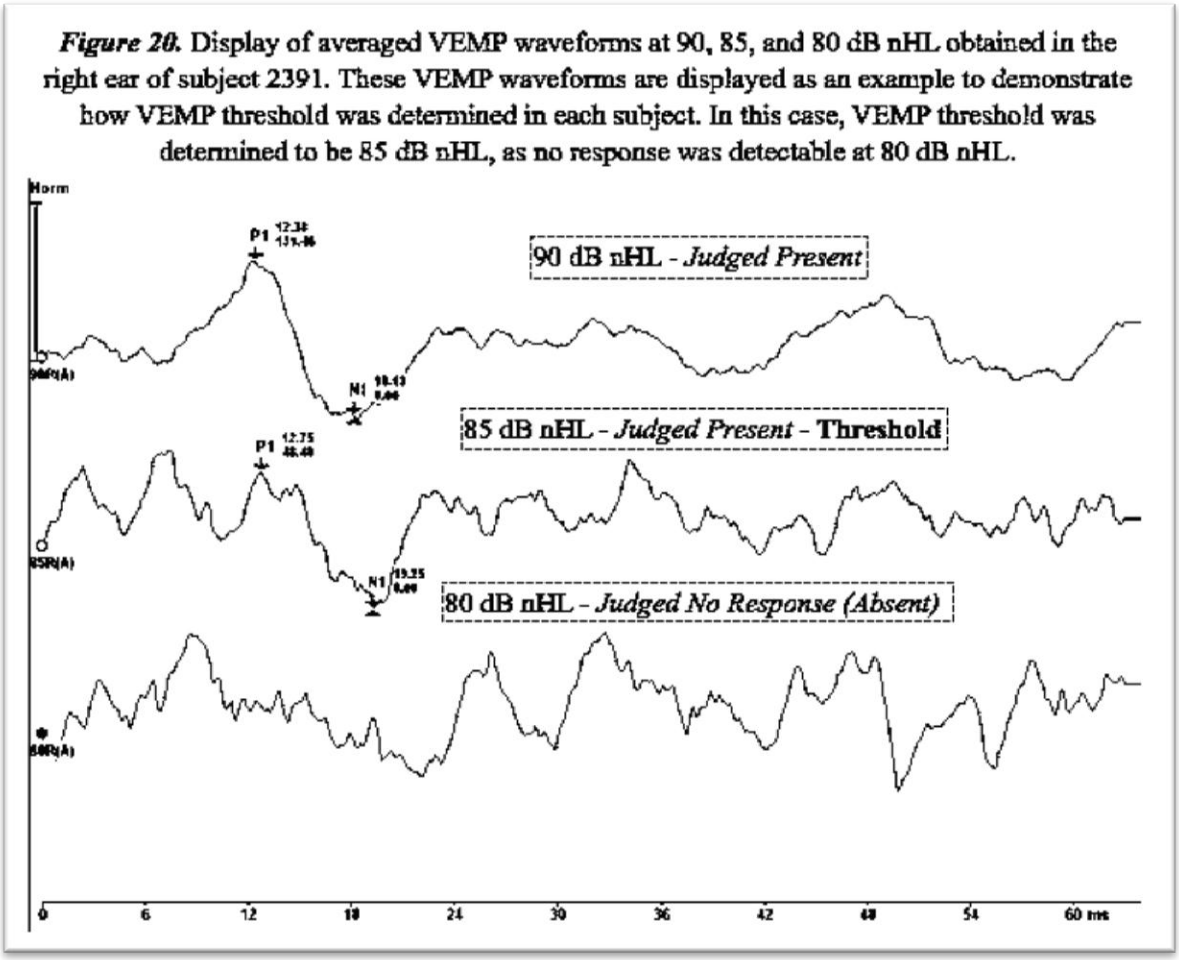


Figure 20. **Display of averaged VEMP waveforms at 90, 85, and 80 dB nHL obtained in the right ear of subject 2391. These VEMP waveforms are displayed as an example to demonstrate how VEMP threshold was determined in each subject. In this case, VEMP threshold was determined to be 85 dB nHL, as no response was detectable at 80 dB nHL.**

Table 9. VEMP threshold values (dB nHL) in each ear for eighteen individual subjects.

Subject #	Threshold	
	Right	Left
3663	85	80
2569	85	95
6471	80	85
1598	85	85
4216	90	95
5157	80	80
8012	75	75
5391	80	80
4777	85	80
4519	85	85
6300	85	90
8388	80	75
2391	85	90
7825	90	85
4412	95	95
8850	85	80
4326	80	85
5698	75	85
Mean	83.61	84.72
SD	5.09	6.29
Median	85	85
Range	75-95	75-95
n	18	18

Laterality of VEMP responses

In Table 10, the VEMP response detectability of data for each individual subject is presented. The VEMP recordings were obtained using monaural stimulation (i.e., right or left ear). These VEMP responses were recorded simultaneously using a two-channel recording set-up. When the active electrode was located on the SCM muscle that was on the same side as the ear being stimulated, this recording was referred to as the 'ipsilateral test condition.' For example, when the stimulus was presented to the right ear, the ipsilateral recording was obtained with the active electrode located on the right SCM muscle.

In contrast, if the active electrode was located on the SCM muscle opposite to the ear that was being stimulated, the recording was referred to as the 'contralateral test condition.' In the ipsilateral condition, the SCM muscle was always contracted, whereas in the contralateral condition, the SCM was not contracted. The detectability of VEMP responses from these ipsilateral and contralateral recording channels was separately analyzed at each of the supra-threshold stimulus intensities (i.e., 95 and 90 dB nHL).

The VEMP responses were detected more frequently in the ipsilateral versus contralateral channel. This pattern was particularly evident for the 90 dB nHL responses. Specifically, at 95 dB nHL 100% of the VEMP responses were present in the ipsilateral recordings, whereas 97.22% were present in the contralateral recording. When the stimulus intensity was reduced to 90 dB nHL, the overall detectability rate decreased from approximately 98-100% to approximately 78-89%. Secondly, the detection rate was lower for the contralateral versus ipsilateral recordings at this lower stimulus intensity

Table 10. Prescence or absence (detectability) of ipsilateral and contralateral VEMP recordings from the right and left ears of eighteen individual subjects.

Subject #	Ear	95 dB nHL		90 dB nHL	
		Ipsilateral	Contralateral	Ipsilateral	Contralateral
3663	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
2569	Right	✓	✓	✓	✓
	Left	✓	✓	✗	✗
6471	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
1598	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
4216	Right	✓	✓	✓	✓
	Left	✓	✓	✗	✗
5157	Right	✓	✗	✓	✓
	Left	✓	✓	✓	✓
8012	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
5391	Right	✓	✓	✓	✗
	Left	✓	✓	✓	✓
4777	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✗
4519	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
6300	Right	✓	✓	✓	✗
	Left	✓	✓	✓	✓
8388	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
2391	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
7825	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✗
4412	Right	✓	✓	✗	✗
	Left	✓	✓	✗	✗
8850	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
4326	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
5698	Right	✓	✓	✓	✓
	Left	✓	✓	✓	✓
Total		36/36	35/36	32/36	28/36
Percent present (%)		100	97.22	88.89	77.78

Note. ✓ = VEMP response present/detectable. ✗ = VEMP response absent/not detectable.

(77.78% versus 88.89%). There were no instances where the VEMP was judged to be present in the contralateral channel and absent in the ipsilateral channel.

A second pattern was preliminarily investigated was difference in the peak-to-peak amplitude values for the ipsilateral versus contralateral recordings for 95 dB nHL. In Appendix E, a table is shown displaying the peak-to-peak amplitude values of p13-n23 obtained from both the ipsilateral and contralateral recordings when recorded to monaural stimulation of each ear (i.e., right or left ears). The descriptive statistics associated with these amplitude values are also presented. There were obvious differences in the mean amplitude values obtained from the ipsilateral versus contralateral channels. For example, in the right ear, the mean peak-to-peak amplitude value of p13-n23 was 76.64 μV (SD = 56.81) in the ipsilateral channel and 25.12 μV (SD = 16.10) in the contralateral channel.

It should also be noted that at 95 dB nHL, every subject had present VEMP responses when recorded from the ipsilateral channel when recorded from both sides of the neck. In contrast, subject 5157 had an absent response in the contralateral channel when recorded from the right side of the neck. This subject had present responses in both recording channels when VEMPs were recorded from the left side of the neck. All other subjects had present VEMP responses in both recording channels when recorded from both sides of the neck. Therefore, the data from subject 5157 was not included in the comparison of amplitude values from the ipsilateral versus contralateral channel when recorded from the right side of the neck.

A set of one-way ANOVAs was performed to investigate whether a statistically significant difference existed in the peak-to-peak amplitude values in the ipsilateral

versus contralateral channels. These differences were investigated for the recordings from the right and left sides of the neck separately. Results of the ANOVA showed that the peak-to-peak amplitude values recorded from the ipsilateral channel were significantly larger from those recorded from the contralateral channel when recorded from both the right [F (1, 32) = 12.943, (p=0.001)] and left [F (1, 34) = 18.898, (p=0.000)] sides of the neck.

Tonic EMG Level

As previously mentioned in the review of the literature, the tonic EMG level has been reported to have an effect on the peak-to-peak amplitude of the VEMP response. Akin and colleagues reported that VEMP responses have been successfully recorded by contracting the SCM muscles to tonic EMG levels ranging from 30 to 90 μV (e.g. Akin & Murnane, 2001; Akin et al., 2004). During the collection of pilot data for the current research study, the subjects' tonic EMG level during contraction of their SCM muscle was assessed using the Delsys Bagnoli 2 EMG monitoring system. It was discovered that our pilot subjects required much larger tonic EMG levels to successfully record the VEMP response. Given this finding, the investigators in this current study made the decision for each individual subject to establish a target EMG level at the beginning of their test session.

Prior to calculating the RMS value associated with each tonic EMG level, we conducted several post-processing steps within the Delsys EMGworks Signal Analysis software. These steps included: (1) identifying the time frame (in seconds) that corresponded to the actual time during which VEMP recording for each trial took place;

(2) digitally filtering out any 60 Hz electrical interference that may have influenced the recording of EMG activity using a band stop filter; and (3) determining the RMS value of the actual EMG (in µV), which was calculated by the Delsys system for that particular time frame.

Reports of the target EMG level and the RMS (root mean square) value of the actual EMG levels from each subject are displayed in Table 11. Separate EMG level measurements were obtained for the right and left SCM muscle contractions at each of the stimulus intensities. Since two averaged VEMP waveforms were recorded at each of the stimulus intensities (i.e., trial 1 and trial 2), actual tonic EMG levels were calculated separately for each of these trials. For example, for subject 3663, the target EMG level was set to 750 µV. Then, when the subject contracted their right SCM muscle in response to a 90 dB nHL 500 Hz tone burst stimulus delivered to their right ear, their actual EMG levels were 696.26 µV for trial 1 and 692.72 µV for trial 2. Similarly, when a 90 dB nHL stimulus was delivered to the left ear, their actual EMG levels were 703.16 µV for trial 1 and 754.57 for trial 2.

Next, the percent deviation that each individual's actual EMG level differed from their target EMG level was calculated. These data were also presented in Table 11. This was reported as a percentage for each trial as well as for the averaged data from these trials (shown in the right hand side of Table 11). To calculate the percent deviation values, the actual EMG level value was divided by the target EMG level value. This value was then converted into a percentage by multiplying it by 100. Lastly, these percent values were subtracted from 100 to finally obtain the reported percent deviation values. For

example, for subject 3663, the percent deviation for trial 1 at 90 dB nHL was calculated by:

$$\frac{696.26\ \mu V(Actual\ EMG\ level)}{750\ \mu V\ (Target\ EMG\ level)} = 0.9283\% \times 100 = 92.83\%$$

$$100 - 92.83\% = \mathbf{7.17}\%$$

Once the percent deviation scores were calculated for all 18 subjects, this data revealed that all subjects were able to maintain a stable tonic EMG level in association with the SCM muscle contractions during multiple VEMP recordings. These actual EMG levels were all within 10% of the target EMG levels for all 18 subjects. Lastly, there was a huge range of EMG target levels across the 18 subjects with normal vestibular function that were included in the current study. Specifically, these EMG target levels ranged from 300 µV to 1250 µV, which we considered to be much higher than those reported by Akin and colleagues.

Table 11. Tonic EMG level data for each recording side of 18 individual subjects. The following was reported for each recording side of each subect: EMG target level (μV), actual recorded EMG level RMS values (μV) for separate trials and averages of trials at both 95 and 90 dB nHL; and percent deviation (%) of the actual RMS value from the EMG target level for that subject.

Subject #	EMG Target	Intensity	RAW DATA				AVERAGED			
			Right SCM		Left SCM		Right SCM		Left SCM	
			Actual EMG Level (RMS)	% Deviation	Actual EMG Level (RMS)	% Deviation	Averaged EMG Level (RMS)	% Deviation	Averaged EMG Level (RMS)	% Deviation
3663	750	90	696.26	7.17	703.16	6.25	694.49	7.40	728.87	2.82
			692.72	7.64	754.57	0.61				
		95	721.81	3.76	780.09	3.86	707.80	5.70	764.69	1.92
			693.79	7.50	749.30	0.09				
2569	450	90	398.90	4.96	459.85	2.14	449.18	2.93	481.28	3.94
			499.47	9.90	502.72	5.68				
		95	503.89	4.40	420.51	6.55	459.59	6.06	395.85	0.81
			415.29	7.71	371.18	4.70				
6471	1250	90	1241.12	0.71	1250.26	0.02	1235.34	1.17	1220.48	2.36
			1229.55	1.64	1190.69	4.74				
		95	1200.48	3.96	1256.62	0.53	1259.37	0.74	1243.30	0.54
			1318.25	5.18	1229.98	1.60				
1598	300	90	279.21	6.93	323.47	7.25	289.42	3.53	315.61	4.95
			299.62	0.13	307.76	2.52				
		95	289.81	3.40	271.87	9.38	292.90	2.37	289.09	3.64
			295.99	1.34	306.32	2.06				
4216	300	90	284.32	5.23	283.32	5.56	277.76	7.41	280.04	6.65
			271.19	9.60	276.76	7.75				
		95	276.52	7.83	304.93	1.62	280.97	6.34	297.37	0.88
			285.41	4.86	289.81	3.40				
5157	600	90	631.46	4.98	629.73	4.72	615.64	2.54	630.72	4.87
			599.82	0.03	631.71	5.02				
		95	618.10	2.93	606.33	1.04	614.71	2.39	612.11	1.98
			611.32	1.85	617.90	2.90				
8012	550	90	511.13	7.07	573.49	4.10	550.26	0.05	575.71	4.47
			589.39	6.68	577.93	4.83				
		95	540.25	1.77	565.43	2.73	528.01	4.00	538.85	2.03
			515.76	6.23	512.28	6.86				
5391	1000	90	1062.19	5.85	993.42	0.66	1043.49	4.17	1002.44	0.24
			1024.79	2.42	1011.46	1.13				
		95	1050.55	4.81	1008.01	0.79	1047.22	4.51	1007.88	0.78
			1043.88	4.20	1007.74	0.77				

Note. EMG = Electromyographic; RMS = Root mean square; SCM = Sternocleidomastoid.

Table 11. (cont'd)

4777	400	90	438.69	8.82	362.10	9.47	423.87	5.63	389.85	2.54
			409.05	2.21	417.61	4.22				
		95	411.91	2.89	403.47	0.86	409.34	2.28	401.96	0.49
			406.77	1.66	400.46	0.12				
6300	800	90	763.07	4.62	772.20	3.47	768.96	3.88	770.33	3.71
			774.86	3.14	768.45	3.94				
		95	777.40	2.83	760.45	4.94	765.69	4.29	761.86	4.77
			753.97	5.75	763.27	4.59				
4519	300	90	318.86	5.92	312.13	3.89	315.55	4.93	308.65	2.80
			312.24	3.92	305.17	1.69				
		95	311.12	3.58	311.12	3.58	311.82	3.79	311.67	3.75
			312.52	4.01	312.22	3.91				
8388	600	90	590.28	1.62	594.34	0.94	587.69	2.05	597.94	0.34
			585.10	2.48	601.53	0.25				
		95	605.96	0.98	592.85	1.19	600.08	0.01	589.30	1.78
			594.20	0.97	585.75	2.38				
2391	1000	90	960.37	3.96	959.02	4.10	954.88	4.51	991.98	0.80
			949.39	5.06	1024.94	2.43				
		95	969.96	3.00	959.32	4.07	967.45	3.25	950.89	4.91
			964.94	3.51	942.46	5.75				
7825	900	90	915.52	1.69	881.85	2.02	918.17	1.98	889.28	1.19
			920.83	2.26	896.71	0.37				
		95	877.70	2.48	892.92	0.79	878.91	2.34	876.45	2.62
			880.12	2.21	859.99	4.45				
4412	300	90	308.72	2.82	315.19	4.82	305.15	1.69	320.26	6.33
			301.59	0.53	325.34	7.79				
		95	315.10	4.79	311.84	3.80	321.92	6.81	319.04	5.97
			328.74	8.74	326.25	8.05				
8850	600	90	537.98	4.76	582.91	2.85	552.89	5.06	577.18	3.80
			567.80	5.37	571.44	4.76				
		95	552.64	7.89	572.24	4.63	558.50	6.92	589.73	1.71
			564.36	5.94	607.22	1.19				
4326	1000	90	962.33	3.77	978.55	2.14	990.99	0.90	978.91	2.11
			1019.64	1.93	979.27	2.07				
		95	999.68	0.03	930.82	6.92	998.77	0.12	986.37	1.36
			997.86	0.21	1041.92	4.02				
5698	650	90	621.20	4.43	672.61	3.36	608.44	6.39	667.94	2.69
			595.69	8.36	663.28	2.00				
		95	645.67	0.67	673.89	3.55	642.98	1.08	650.39	0.06
			640.30	1.49	626.88	3.56				

CHAPTER 5

DISCUSSION

The purpose of the current study was to establish normative data for the various response indices of the VEMP response, so that this type of diagnostic assessment can be offered as part of the vestibular test battery at the Speech-Language Hearing Center at Towson University. The VEMP response was recorded to monaural stimulation using a 500 Hz tone burst at varying stimulus intensities from both sides of the necks of 18 young adult participants with a negative history of vestibular pathology. The VEMP was recorded using 2 recording channels (i.e., ipsilateral and contralateral).

In order to make more direct comparisons to studies documented in the literature, our stimulus intensities (expressed in dB nHL) were converted to dB SPL. In the IHS system used in the current study, the 500 Hz tone burst stimuli presented at 0 dB nHL were equivalent to 33 dB peak SPL when presented through ER-3A insert earphones. For example, our 95 dB nHL signal is equivalent to a 128 dB peak SPL signal (i.e., 95 + 33 = 128). A comparison of our current normative data to 95 and 90 dB nHL (i.e., 128 and 123 dB peak SPL respectively) 500 Hz tone burst stimuli to previous normative data reported in the literature needs to be addressed with some caution, given that these studies reported the stimulus intensity for the 500 Hz tone burst in dB SPL units rather than the dB nHL units used in the current study.

The organization of the discussion will follow the same sequence as was found in the results section, beginning with a discussion on the latency findings. Additionally, a

discussion will ensue regarding limitations of the current study as well as suggestions for future research in this area.

Absolute Latency Value for Waves p13 and n23

In the current study, the mean latency values of wave p13 (from collapsed data) were 13.39 ms (SD = 1.16) and 13.38 ms (SD = 1.11) for 500 Hz tone burst stimuli presented at 95 dB nHL and 90 dB nHL respectively. These mean latency wave p13 latency values from the current study are consistent with those of Wang and colleagues (2008), who reported mean p13 latency values of 13.9 ms (SD = 1.0) to 500 Hz tone burst stimuli presented at 95 dB nHL to the ears of 14 adults with normal vestibular function.

These mean wave p13 latency values are also relatively consistent with the normative p13 latency values reported by Basta and colleagues (2005). In their study, they recorded p13 latency values to 500 Hz tone burst stimuli using similar recording parameters as those used in the current study. These investigators reported that their mean p13 latency value was 16.2 ms (SD = 2.5) in their 20 to 40 year age group with normal functioning vestibular systems (n = 23). In this study, the 500 Hz tone bursts were presented at 115 dB SPL. No data was provided by the investigators in regards to converting the dB SPL value into a dB nHL value. Thus, a direct comparison of our data to their data is limited.

In the current study, the mean latency values of wave 23 were 19.22 ms (SD = 2.03) and 19.20 ms (SD = 1.96). The mean n23 latency values from the current study were consistent with those reported by Wang and colleagues (2008). These investigators

reported a mean n23 latency value of 20.9 ms (SD = 1.1). The aforementioned recording parameters used in this study were, again, similar to those employed in the current study. The mean n23 latency values in the current study are also relatively consistent with those presented by Basta and colleagues (2005). These investigators reported a mean n23 latency value of 24.0 ms (SD = 2.6) using similar recording parameters to the current study, and when 500 Hz tone bursts were presented at 115 dB SPL to 23 young adults (ages 20-40) with normally functioning vestibular systems.

As would be expected, there were no significant differences in the mean latency of waves p13 and n23 for the right versus left ear at both supra-threshold stimulus intensities (i.e., 95 and 90 dB nHL). Given that there were no significant ear effects on the mean latency values of waves p13 and n23, the data was collapsed (averaged) across ear to investigate the effects of stimulus intensity on the absolute latency values for each of these two VEMP components.

As was stated by the investigators of previous studies (Akin et al., 2003; Lim et al., 1995), the absolute latency values of the two components of the VEMP response to click stimuli do not vary significantly as a function of stimulus intensity. Specifically, Akin and colleagues (2003) investigated the effects of stimulus intensity on latency values measured from responses evoked to click stimuli presented at 90, 95, and 100 dB nHL. Lim and colleagues (2005) made similar comparisons of latency values from responses evoked to click stimuli presented at 85, 90, and 95 dB nHL.

Results of these ANOVAs confirmed that there were no significant effects of stimulus intensity on this collapsed (averaged) data for mean wave p13 latency ($p = 0.98$)

and for mean wave n23 latency ($p = 0.99$). This lack of a significant effect of stimulus intensity on the mean latency of waves p13 and n23 is consistent with the findings in the two aforementioned studies (Akin et al., 2003; Lim et al., 1995). Akin and colleagues reported no significant difference in mean latency of waves p13 and n23 for click stimuli presented at 90, 95, and 100 dB nHL. Similarly, Lim and colleagues reported the mean p13 and n23 latency did not differ significantly for click stimuli presented at 85, 90, and 95 dB nHL.

Given our current data, it is suggested that the normative range of latency values in our subjects for wave p13 should be from 11.07 ms to 15.71 ms at 95 dB nHL and from 11.16 ms to 15.60 ms at 90 dB nHL. The normative range established for the absolute latency of wave n23 at 95 dB nHL was from 15.16 ms to 23.28 ms. At 90 dB nHL, a similar normative range, from 15.28 ms to 23.12 ms, was established. These ranges were calculated based on two standard deviations from the mean latency value.

Peak-to-peak Amplitude Value of Wave p13-n23

The mean peak-to-peak amplitude values of p13-n23 (from collapsed data) were 77.18 µV (SD = 39.80) and 48.17 µV (SD = 23.00) at 95 dB nHL and 90 dB nHL respectively. Akin and colleagues (2003) reported a mean amplitude value for p13-n23 of 112 µV (SD not reported) for 500 Hz tone bursts presented at 120 dB peak SPL. Welgampola and Colebatch (2001a) reported a high mean amplitude value for p13-n23 of 91.4 µV (SD not reported) when evoked to a 500 Hz tone burst presented at 123.5 dB SPL in 12 adult subjects with normally functioning vestibular systems. The mean amplitude values for the current study are similar to the findings from Janky and Shepard

(2009), who reported a mean amplitude value of 66.12 µV when 500 Hz tone bursts were presented at 123 dB peak SPL to the ears of young adult participants with normal vestibular function.

In the current study, peak-to-peak amplitude values of p13-n23 were significantly larger when stimuli were presented at 95 dB nHL versus the p13-n23 amplitude values when the stimuli were presented at 90 dB nHL. The mean amplitude increased from 48.17 µV to 77.18 µV when the stimulus intensity was increased from 90 to 95 dB nHL. Thus, there was no evidence of saturation effects (i.e., a decrease in response amplitude at this higher stimulus intensity) for any of the individual 18 subjects or for the group mean amplitude data. The current investigators only explored across this 5 dB range because the maximum limits to present 500 Hz tone burst stimuli in the IHS system was 95 dB nHL.

It should be noted that the variability in the data was much larger in the amplitude data in comparison with the latency data. The variability in our current data was apparent in both standard deviation values as well as the huge range of the amplitude values that occurred across our subjects. This pattern was true at both stimulus intensities and especially evident in the 95dB nHL responses (as seen in Table 6). In the current study, VEMP recordings from young adults with normal vestibular function showed peak-to-peak amplitude values for p13-n23 ranging from 3.25 µV to 185.02 µV. Welgampola and Colebatch (2001b) reported a peak-to-peak amplitude range of 25 to 297 µV when the p13-n23 response was evoked to click stimuli presented at 100 dB nHL (i.e., 145 dB SPL). The large range of peak-to-peak amplitude values apparent in the current data is

relatively consistent to the findings of this previous study, even though the stimulus type differed in each of the studies.

Standard deviations were large in the current amplitude data, and were 39.80 and 23.00 at 95 dB nHL and 90 dB nHL respectively. The standard deviation values in the current study were consistent with those reported by Janky and Shepard (2009). These involved a standard deviation value of 31.49 in the 20-29 year age group to the 123 dB peak SPL stimuli versus our standard deviation of 39.80 for stimuli presented at a similar stimulus intensity (i.e., 128 dB nHL).

Given the enormous variation in peak-to-peak amplitude values that occurred in this current as well as previous studies, typically clinicians do not rely on a normative range of peak-to-peak amplitude values for p13-n23 to determine normality versus abnormality of the VEMP response. Instead, it has been recommended that clinicians calculate interaural asymmetry ratio values for amplitude for their patients.

Interaural Asymmetry Ratio Value for Amplitude

In the current study, interaural asymmetry ratio values were calculated from the peak-to-peak amplitude values for p13-n23. The mean interaural asymmetry ratio values were 21.01% and 18.21% at 95 and 90 dB nHL respectively. The results of the ANOVA showed no statistically significant difference in the interaural asymmetry value as a function of stimulus intensity. These mean asymmetry ratio values were consistent with those reported in the literature. For example, Young and colleagues (2003) reported a mean interaural asymmetry value of 13% (SD = 10) and Li and fellow investigators (1999) reported a mean value of 10.8% (SD = 27.3). Collectively, our mean interaural

asymmetry values at both supra-threshold stimulus intensities all fall within one standard deviation of the mean value reported by these groups of investigators (Li et al., 1999; Young et al., 2003).

Several groups of investigators reported that in most adult subjects with normal vestibular function, an interaural asymmetry ratio value for amplitude should be ≤ 30% (Brantberg & Fransson, 2001; Halmagyi et al., 1994). If we apply this criteria as the upper limits of normal for this response index, 15 of our 18 subjects (83.3%) would be judged to have normal asymmetry ratios for their 95 dB nHL responses and 14 of our 18 subjects (77.8%) for their 90 dB nHL responses (as seen in Table 7). For those individuals whose asymmetry ratios were greater than 30%, the strength of the SCM muscle contractions on each side of the neck was likely not a contributing factor to these high asymmetry ratio values.

Another interesting pattern noted in our asymmetry ratio data was that for 4 of our subjects, their asymmetry ratios were considerably different at each of the supra-threshold stimulus intensities, resulting in a normal asymmetry ratio for one stimulus intensity and an abnormal asymmetry ratio for the other stimulus intensity. For example, subject 5698 had an asymmetry ratio of 42.64% at 95 dB nHL (which would be judged abnormal using the 30% criteria) versus 4.66% at 90 dB nHL (which would be judged as a normal asymmetry ratio).

VEMP Threshold

VEMP thresholds were determined as the lowest stimulus intensity where a replicable VEMP response could be found. The VEMP thresholds across subjects in the

current study ranged from 75 to 95 dB nHL. Mean VEMP thresholds were 83.61 (SD = 5.09) and 84.72 dB nHL (SD = 6.29) in the right and left ears respectively. Thus, it is clear that there were no obvious difference in VEMP thresholds across ears.

In comparison to the documented literature, our VEMP threshold values are consistent with the reported mean VEMP thresholds. Akin and colleagues (2003) reported that VEMP thresholds to 500 Hz tone bursts ranged from 80 to 100 dB nHL in individuals with normal vestibular function, with a mean VEMP threshold value of 91 dB nHL (SD = 5.2). Similarly, Ochi and colleagues (2001) reported normal VEMP thresholds to 500 Hz tone burst stimuli ranging from 80 to 95 dB nHL, with a mean value of 87.78 dB nHL (SD = 4.54). The mean VEMP thresholds from each ear of each participant in the current study fell within two standard deviations of the mean VEMP threshold in the study presented by Akin and colleagues (2003), and within one standard deviation of the mean VEMP threshold reported by Ochi and colleagues (2001).

Laterality of VEMP Responses

An understanding of the laterality of the VEMP response gives better insight to the anatomical pathway of the vestibulo-collic reflex. As previously discussed, in the current study we investigated the detectability rate (i.e., presence versus absence) of the VEMP response in each channel at both 95 dB nHL and at 90 dB nHL. At 95 dB nHL, responses were present 100% of the time for the ipsilateral channel and 97.2% of the time for the contralateral channel. At the lower stimulus intensity (i.e., 90 dB nHL), the overall detectability rates decreased, such that VEMP responses were judged to be present 88.9% of the time in the ipsilateral channel and 77.8% in the contralateral channel.

This decrease in response detectability in the contralateral versus ipsilateral channel supports the finding from Brantberg and Fransson (2001), Colebatch and colleagues (1994) and Murofushi and colleagues (2004) that the VEMP response is largely dominant in the ipsilateral channel. Colebatch and colleagues (1994) and Brantberg & Fransson (2001) recorded VEMPs unilaterally to click stimuli in a group of individuals with normal vestibular function and found that VEMP waveforms were present in both the ipsilateral and contralateral recording channels for most of their subjects. However, the response obtained from the ipsilateral channel was always larger in amplitude versus the response obtained from the contralateral channel. Although that issue was not directly evaluated in the current study, this pattern was observed in the individual subject data.

Murofushi and his fellow investigators (2004) discussed the laterality of the VEMP response waveform obtained to a 500 Hz tone burst stimulus presented at 95 dB nHL. In this study, the 500 Hz tone bursts were presented monaurally. The subject was instructed to contract the SCM muscles on each side of the neck simultaneously (i.e., bilateral recording condition). These investigators reported that the VEMP response was consistently present (i.e., 100% of the time) in the ipsilateral channel and only 24% in the contralateral channel. The difference in test protocol (i.e. bilateral in the previous study versus unilateral in the current study) between this study and our current study likely partly accounted for these laterality differences.

In an animal study on cats it has been determined through neurophysiological means that the efferent vestibulospinal neurons, which originate from the saccule primarily projected to the motoneurons of the ipsilateral SCM muscle (Kushiro, Zakir,

Ogawa, Sato, & Uchino, 1999). Thus, it would be expected that the amplitude of the VEMP response recorded from the ipsilateral SCM muscle would have a larger amplitude than that recorded from the contralateral channel. The current results as well as the earlier studies in laterality determined that the VEMP response is dominant in the ipsilateral vestibulo-collic pathway.

Choice of Stimulus Type for Recording VEMP Responses

Based on the findings from the current study (using 500 Hz tone burst stimuli) and those from a related study (using click stimuli), it was decided that the 500 Hz tone burst is the optimal stimulus type for recording VEMP responses. The 500 Hz tone burst stimulus type was chosen for several reasons. First, it was found that when these two stimuli were presented at the same presentation level (in dB nHL), a slightly higher SPL value was being delivered into the ear canals of our subjects when 500 Hz tone burst stimuli were presented (versus click stimuli). Thus, this led to somewhat better morphology of the VEMP responses using the 500 Hz tone burst stimulus type.

Also, the detection rate of VEMP responses was higher in both the ipsilateral and contralateral channels at both supra-threshold stimulus intensities. For example, at 95 dB nHL, VEMP responses were present 100% and 97% of the time in the ipsilateral and contralateral channels when 500 Hz tone burst stimuli were used. In contrast, when click stimuli were used, VEMP responses were only present 94% of the time in the ipsilateral channel and 78% of the time in the contralateral channel.

Tonic EMG Level

Perhaps our most unexpected findings in the current study came from our recording of EMG activity from the contracted SCM muscles during VEMP recording. Our original goal with the use of this EMG recording equipment was to keep SCM muscle contractions constant across both sides of the neck and consistent across subjects. We initially chose a target EMG level of 50 to 60 µV, as consistent with the findings from Akin and colleagues (Akin & Murnane, 2001; Akin et al., 2004). Throughout the collection of pilot data, however, we discovered that at this tonic EMG level of 50 to 60 µV we could not successfully recorded a VEMP response in our subjects. Secondly, most of these pilot subjects reached this 50- to 60-µV tonic EMG level when their SCM muscles were in a relaxed state.

Based on our amplitude findings in the pilot data, we made a decision that each subject would establish their target EMG level based on the natural contraction of their SCM muscle. They were asked to maintain this tonic EMG level (± 10%) for the remainder of their VEMP recordings via visual observation. These subjects naturally contracted their SCM muscle by lifting their head from a reclining position and then turning toward the opposite direction (i.e. if the goal was to contract the right SCM muscle the participant would turn their head toward the left). During the first few VEMP recordings a target EMG level was established based on the participant's natural contraction of their SCM muscle. The establishment of the target EMG level was usually completed within two to three trials (usually the first two trials at 90 dB nHL and the first trial at 95 dB nHL if needed).

In the current study, every subject was able to contract both SCM muscles to within 10% of their EMG target level for at least the recordings for two trials each at 95 and 90 dB nHL. Actual tonic EMG level values ranged widely across the 18 subjects (i.e., 271.19 to 1250.26 μV during the recording of the 90 dB nHL responses and 271.87 to 1318.25 μV for the 95 dB nHL responses). This large range of target EMG levels indicates that much higher tonic EMG levels were required when successfully recording VEMP responses in the current study than the range of 30 to 90 μV that was reported by Akin and colleagues (Akin & Murnane, 2001; Akin et al., 2004). Lim and colleagues (1995) reported a mean EMG level of 210 μV (SD = 151), which was twice the value reported by Akin and her colleagues, but was still considerably lower than the overall mean EMG target level (across sides of neck and stimulus intensity) reported for this study (mean = 646.87 μV; SD = 278.10).

Limitations of the Current Study and Future Directions

Our normative findings were generally similar to those reported by previous investigators who explored the VEMP response. Several limitations of the current study were found throughout the process of our data collection. In the development of the test protocol for the current study, we were interested in exploring saturation effects (i.e., decrease in response amplitude) on the VEMP response at the higher stimulus intensities. Throughout the calibration process, it was discovered that 95 dB nHL (i.e., 128 dB peak SPL) was the maximum stimulus intensity that the IHS system would present through ER-3A insert earphones. We did not discover any saturation effects between 90 and 95 dB nHL. Thus, it is possible that the VEMP response does saturate at higher stimulus intensities. Due to the limitations of the equipment used in the current study, no evidence

in regards to saturation effects on the VEMP response could be reported. A future study could investigate whether saturation effects occur in the VEMP response at higher stimulus intensities than those utilized in the current study.

It needs to be stated that these responses were obtained from young adult subjects (ages 20-30 years; mean = 24 years) and these findings cannot be generalized to older individuals. It is widely known that the CNS is negatively affected by age. Janky and Shepard (2009), who investigated age-related changes on the response indices of the VEMP in subjects of varying ages with normal vestibular function, found significant negative correlations of VEMP amplitude with age at a supra-threshold presentation of 500 Hz tone burst stimuli. These investigators also found significantly increased mean VEMP thresholds with increasing age when 500 Hz tone bursts were used as stimuli (Janky & Shepard, 2009). It is also likely that there would be a larger fatigue factor related to SCM muscle contraction occurring in the older population. Future investigations should include establishing normative data using individuals who are older than 30 years of age.

A note should be made regarding the efficacy of monitoring EMG level during VEMP recordings. The operation that allowed us to monitor this EMG activity was rather cumbersome, requiring an additional investigator to monitor this EMG level while the recording the VEMP response was operated in the opposite side of the booth. It was required that a talk-forward/talk-back system configured with the audiometer was set up. This allowed the two investigators to communicate in synchronizing the simultaneous recording of the VEMP response and tonic EMG level.

The EMG recording electrodes (placed on both SCM muscles) were relatively large 'paddle-like' electrodes, which required a great deal of adhesive to remain secured on the SCM muscles. These electrodes frequently fell off during test sessions. In these cases, the investigator who recorded EMG level re-applied and re-secured the EMG electrodes. The investigators in the current study spent a great deal of time troubleshooting the equipment and setting up test protocols to use the Delsys EMGworks software for our purposes. Even though this was a worthwhile endeavor for our research purposes, it is suspected that most clinical audiologists do not have any background in the recording and analysis of EMG activity. Therefore, due to the cumbersome nature of the operation used to record EMG activity, it is realistic to state that most clinical audiologists would not want to adopt these practices in their regular clinical VEMP assessments.

The current electrode montage used for the EMG monitoring using the Delsys system (active electrode placed on the contracted SCM muscle and reference electrode placed on the back of the left hand) was recommended by the engineer who designed the Delsys system. However, the distance between the active and reference electrode sites may have contributed to the large variability seen in the actual tonic EMG levels recorded across subjects. Akin et al. (2004), who also used the Delsys system to monitor EMG activity, placed the reference electrode on the participants' wrists. With this similar electrode montage of Delsys electrodes, they obtained tonic EMG levels within the range of 30 to 90 μV (Akin et al., 2004). It should be investigated in future studies whether different placement of the reference electrode would contribute to the variability in tonic EMG levels across subjects.

Questions lie in regards to whether this tonic EMG level does have an overall influence on the amplitude of the VEMP response. Colebatch and colleagues (1994) reported increases in peak-to-peak amplitude values of p13-n23 with increases in stimulus intensity of click stimuli. Akin and colleagues (2004) reported that within subjects, VEMP amplitudes increased with increases in tonic EMG levels from 30 to 90 μV. However, this issue was not directly addressed in the current study. A further study could investigate the effects on VEMP response amplitude resulting from the use of a tennis ball or visual observation to estimate equal SCM muscle contraction on each side versus formal EMG monitoring.

Summary of Equipment Parameters and Normative Data

Table 12 displays a summary of the equipment parameters (i.e., stimulus and recording parameters) that were used to establish our normative data using the IHS system. Table 13 displays a summary of the normative data on the various response indices of the VEMP response on our 18 subjects with normal vestibular function. These values will be used as the normative criteria when the VEMP assessment is offered in the Speech-Language and Hearing Center at Towson University.

Table 12. Summary of the stimulus and recording parameters used in the IHS system to establish normative data on the various response indices of the VEMP at the Towson University Speech-Language and Hearing Center.

Stimulus Parameters			
Stimulus Type	500 Hz tone burst		
Rate	5 Hz		
Duration	7 ms		
Stimulus Intensity	**Supra-threshold:** 95 and 90 dB nHL (128 and 123 dB peakSPL)	**Threshold Search:** 70-85 dB nHL (103-118 dB peakSPL)	
Transducer	ER-3A Insert Earphones		
Stimulation	Monaural		
Recording Parameters			
Recording channels	2-channel recording (ipsilateral and contralateral)		
Electrode Montage	**Recording from Right SCM:** Active (ipsilateral channel): middle third of contracted right SCM muscle Active (contralateral): middle third of relaxed left SCM muscle Reference: upper sternum Ground: Forehead (Fpz)	**Recording from Left SCM:** Active (ipsilateral channel): middle third of contracted left SCM muscle Active (contralateral channel): middle third of relaxed right SCM muscle Reference: upper sternum Ground: Forehead (Fpz)	
Amplifier Gain Setting	50K		
Bandpass Filter Setting	50-1500 Hz		
Number of Sweeps/Trials	128 sweeps per trial	2 trials	Total of 256 sweeps contributing to each averaged waveform
Analysis Window	64 ms post-stimulus onset		

Table 13. Summary of the normative data on the various response indices of the VEMP for the Towson University Speech-Language and Hearing Center.

	Absolute Latency (ms)				Peak-to-peak amplitude (μV)		Interaural Asymmetry Ratio (%)		VEMP threshold (dB nHL)	
	p13		n23							
	95 dB nHL	90 dB nHL	95 dB nHL	90 dB nHL	95 dB nHL	90 dB nHL	95 dB nHL	90 dB nHL	Right Ear	Left Ear
Mean	13.39	13.38	19.22	19.20	77.18	48.17	21.01	18.21	83.61	84.72
SD	1.16	1.11	2.03	1.96	39.80	23.00	14.33	14.51	5.09	6.29
Range	10.13-17.25	10.75-17.63	15.88-25.88	15.50-23.13	3.25-185.02	13.37-131.46	2.12-53.96	0.12-43.27	75-95	75-95

Note. *VEMP literature (Brantberg & Fransson, 2001; Halmagyi et al., 1994) has suggested a normative upper cutoff criteria of ≤ 30% for the interaural asymmetry ratio response index. It should be noted that three of our subjects had elevated asymmetry ratio values for their 95 dB nHL responses and four of our subjects had elevated asymmetry ratio values for their 90 dB nHL responses based on this criteria.
**None of our subjects with normal vestibular function had VEMP thresholds ≤ 70 dB nHL.

APPENDICES

APPENDIX A – RECRUITMENT 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 **sphill5@students.towson.edu** or **(301) 523-0946**

Dr. Peggy Korczak (Associate Professor) at **pkorczak@towson.edu** or **(410) 704-5903**

APPENDIX B – INFORMED CONSENT FORM

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 normal otologic history. 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 their upper sternum and earphones being inserted into the patient's ears. The patient is then instructed, during each recording, to turn their head to the side to contract the sternocleidomastoid (SCM) muscle. Acoustic stimuli will be presented to the participant's ears during recording, and the response from the contracted SCM muscle will be recorded. The stimulus intensity will be adjusted between recordings. Approximately twenty-four recordings will take place in each participant's test session, and each recording will take approximately 30 seconds. 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 – 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.

_____
_____
_____

4. Do you have difficulties with intolerance to loud sounds?

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

If yes, please describe.

_____
_____
_____

APPENDIX D – IRB APPROVAL

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

TOWSON
UNIVERSITY

Date: Friday, December 16, 2011

NOTICE OF APPROVAL

TO: Lauren McGrath **DEPT:** ASLD

PROJECT TITLE: *Normative Study on Vestibular Evoked Myogenic Potentials (VEMPS)*

SPONSORING AGENCY:

APPROVAL NUMBER: 12-A025

The Institutional Review Board for the Protection of Human Participants has approved the project described above. Approval was based on the descriptive material and procedures you submitted for review. Should any changes be made in your procedures, or if you should encounter any new risks, reactions, injuries, or deaths of persons as participants, you must notify the Board.

A consent form: [ ] is [ ] is not required of each participant

Assent: [ ] is [ ] is not required of each participant

This protocol was first approved on: 16-Dec-2011
This research will be reviewed every year from the date of first approval.

Steven Mogge, Member
Towson University Institutional Review Board
WSP

APPENDIX E – LATERALITY AND AMPLITUDE

Appendix E. Peak-to-peak amplitude values of p13-n23 recorded simultaneously from both the ipsilateral and contralateral channels in the recordings from each side of the neck. Each side of the neck was recorded separately in each of our 18 subjects. Descriptive statistics are presented below the raw amplitude values.

Subject #	Right SCM		Left SCM	
	Ipsilateral	Contralateral	Ipsilateral	Contralateral
3663	24.43	24.00	45.34	20.02
2569	53.24	39.97	30.79	10.12
6471	133.71	30.82	114.99	48.63
1598	44.90	11.48	40.49	6.53
4216	6.79	3.96	3.25	6.25
5157*	11.58*	NR	38.72	18.25
8012	35.23	13.80	53.09	25.65
5391	38.93	10.30	42.37	19.31
4777	34.92	13.90	63.54	9.97
4519	89.89	23.19	49.06	13.61
6300	145.64	39.77	93.34	24.00
8388	76.17	30.55	111.48	22.78
2391	185.02	56.07	103.23	26.71
7825	59.44	59.44	83.76	83.76
4412	12.62	7.36	9.92	4.01
8850	127.62	23.28	108.69	20.88
4326	56.43	22.82	53.88	17.91
5698	177.91	16.33	71.55	23.68
Mean	76.64	25.12	62.08	22.34
SD	56.81	16.10	34.13	18.43
Median	56.43	23.19	53.49	19.67
Range	6.79-185.02	3.96-59.44	3.25-114.99	4.01-83.76
n	17	17	18	18

Note. * indicates that the subject/amplitude value was not used in statistical analysis because the VEMP response was absent in the contralateral channel; NR = no response.

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