# THE EFFECT OF COCHLEAR IMPLANTATION ON THE VESTIBULAR EVOKED MYOGENIC POTENTIAL RESPONSE IN CHILDREN AND ADULT PARTICIPANTS.

by

JAMIE MARIE MOUSHEY BOGLE

B.S., Saint Louis University, 2002

Au.D., University of Colorado at Boulder, 2007

A thesis submitted to the

Faculty of the Graduate School of the

University of Colorado in partial fulfillment of the

requirements for the degree of

Doctor of Philosophy

Department of Speech, Language and Hearing Sciences

2010





ProQuest LLC
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106-1346

This thesis entitled:
The Effect of Cochlear Implantation on the Vestibular Evoked Myogenic Potential Response in Children and Adult Participants
written by Jamie Marie Moushey Bogle
has been approved for the Department of Speech, Language and Hearing Sciences.

_____

Christine Yoshinaga-Itano, committee chair

_____

Phillip Gilley, committee member

Date _____

The final copy of this thesis has been examined by the signatories, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

IRB protocol #0510.8

Moushey Bogle, Jamie Marie (Ph.D., Speech, Language and Hearing Sciences)

The Effect of Cochlear Implantation on the Vestibular Evoked Myogenic Potential Response in Pediatric and Adult Participants.

Thesis directed by Professor Christine Yoshinaga-Itano

Cochlear implantation has become an integral option for both children and adults with severe to profound degrees of sensorineural hearing loss, providing access to the auditory environment. While improvement in access to auditory information is required for appropriate spoken speech understanding in adults and for appropriate speech and language development in children with significant sensorineural hearing loss, the effects of cochlear implantation on the additional organs within the ear have not been fully evaluated. This study evaluated 40 cases (15 male, 25 female) between four and 60 years of age ($M = 22.03$, $SD = 18.10$). The goal of this study was to evaluate the presence of the vestibular evoked myogenic potential (VEMP) response in children and adults with severe to profound sensorineural hearing loss, and to determine the proportion of change to this response following implantation. Additional variables were evaluated to determine possible risk factors for absent VEMP responses both prior to and following cochlear implantation. While the VEMP responses were consistent with adults with normal hearing and vestibular systems prior to implantation, 47% of cases demonstrated a change from present to absent VEMP response following implantation, a significant decrease. No additional variables, with the exception of hearing loss stability prior to implantation, were found to be significant in these comparisons. The results of this study indicate the further need for vestibular evaluation within the

population of cochlear implant candidates. Knowledge of the state of this system may provide additional information about the stability of the inner ear and should be used as an important counseling tool for clinicians of both pediatric and adult cochlear implant candidates. Due to the high proportion of change in the presence of the VEMP response, additional study of this response should be conducted to obtain greater understanding of how the vestibular system is affected by cochlear implantation surgery.

*Keywords:* cochlear implantation, VEMP, vestibular, saccule, deafness

# ACKNOWLEDGEMENTS

I would like to gratefully and sincerely thank Dr. Christine Yoshinaga-Itano and Dr. R. Steven Ackley for their guidance, understanding, and patience during my graduate studies at the University of Colorado at Boulder. Also, I would like to thank my doctoral committee for all of their input and accessibility throughout the duration of this project as well as the staff at the University of Colorado Hospital who so graciously allowed me access to their facilities in order to complete this project.

I would like to thank my classmates, especially Darcy Strong, Becky Nelson, Melissa Garafalo, Denise Nicholson, and Cory Portnuff. Their constant support, mentoring, and friendship over the last seven years are greatly appreciated. I could not hope to be the writer or researcher that I strive to be without their support.

CONTENTS

# TABLES

Table

# FIGURES

Figure

# CHAPTER 1

## INTRODUCTION

The cochlea and vestibular systems are closely related developmentally, and may be exposed to simultaneous insult, leaving individuals with sensorineural hearing loss at increased risk for vestibular anomalies when compared to those with normal hearing thresholds. Furthermore, individuals with severe to profound degrees of hearing loss, as well as those with acquired forms of hearing loss (e.g. hearing loss due to meningitis infection), have demonstrated additional risk for vestibular dysfunction due to the additional trauma inflicted on the entire labyrinth (Arnvig, 1955; Goldstein, Landau, & Kleffner, 1958; Huygen, van Rijn, Cremers, & Theunissen, 1993; Sandberg & Terkilsen, 1965).

Research has found that individuals with severe to profound degrees of sensorineural hearing loss demonstrate abnormal vestibular function; however, the rate of dysfunction varied based on the test protocol used in the study. Generally, clinical protocols have provided evaluation of the horizontal semicircular canal, which has demonstrated the function of one of the five vestibular end organs of the inner ear. Prior to implantation, 30 to 40% of children and adults with severe to profound sensorineural hearing loss have presented with abnormal horizontal semicircular canal function (Buchman, Joy, Hodges, Telischi, & Balkany, 2004; Krause et al., 2009). Additional testing of the vestibular system has focused on the saccule, a linear accelerometer within the inner ear. Studies have suggested that the saccule has demonstrated abnormal function in approximately 30% of children and adults with severe to profound

sensorineural hearing loss (King, 2009; Melvin, Della Santina, Carey, & Migliaccio, 2008; Moushey, Strong, & Ackley, 2010). These tests will be further considered in the review of the literature.

Individuals with severe to profound sensorineural hearing loss have been offered cochlear implantation as a means of acquiring access to auditory function. Studies have suggested that the implantation surgery itself may subsequently injure the vestibular end organs, particularly the saccule, leading to vestibular dysfunction following surgery (Gstoettner et al., 1997; Tien & Linthicum, 2002). In adults, cochlear implantation has been reported to lead to both subjective and objective vestibular dysfunction, with reports of vertigo and imbalance following surgery (Steenerson, Cronin, & Gary, 2001). Studies have shown that 20 to 30% of cochlear implant recipients have demonstrated decreased horizontal semicircular canal function following surgery (Buchman et al., 2004), while saccular testing has demonstrated a change in function in up to 40% of cases (King, 2009; Melvin et al., 2008).

**Statement of Problem**

Currently, minimal attention is given to the status of the vestibular system of individuals receiving cochlear implants. Research has demonstrated the risk for vestibular dysfunction to these individuals due to possible concurrent dysfunction within the inner ear, as well as acquired vestibular loss due to the possible effects of cochlear implantation on the additional structures within the labyrinth (e.g. Buchman et al., 2004; Melvin et al., 2008; Steenerson et al., 2001). The caloric response, which evaluates the horizontal semicircular canal via the vestibulo-ocular reflex, is considered to be the gold

standard of vestibular testing (Desmond, 2004). While the caloric response is diagnostically useful in adults in children as young as four years of age, and provides information about each horizontal semicircular canal individually (Eviatar & Eviatar, 1978; Fife et al., 2000), the effects of cochlear implantation have not been shown to solely impact the function of the horizontal semicircular canal. Prior research has suggested that the saccule is the organ most affected by cochlear implantation (Tien & Linthicum, 2002). The vestibular evoked myogenic potential (VEMP) response is a relatively new addition to the clinical protocol that evaluates the saccule and the inferior branch of cranial nerve VIII (CN VIII). The VEMP response may prove to be a useful addition to the vestibular protocol in order to evaluate individuals for vestibular dysfunction before and after cochlear implantation.

**Purpose of Study**

Because of the risk of vestibular dysfunction for individuals before and after cochlear implantation, this research has proposed to evaluate the VEMP response of the saccule and the inferior branch of CN VIII before and after cochlear implantation in children between four and 17 years of age and in adults between 18 and 60 years of age. This data was collected by completing a review of retrospective data collected between 2007 and 2010 that was available in the Department of Otolaryngology at the University of Colorado Hospital.

**Research Questions**

1. Is the VEMP response present in both ears after cochlear implantation?

2. Is the proportion of the presence of the VEMP response significantly lower after cochlear implantation when compared to the proportion of the presence of the VEMP response before implantation?
3. Is the presence of the VEMP response in cochlear implant candidates significantly lower than the presence of the VEMP response for individuals with normal hearing?
4. Is the presence of the VEMP response in cochlear implant recipients significantly lower than the presence of the VEMP response for individuals with normal hearing?
5. Is there a significant relationship between the presence of the VEMP response and the etiology of hearing loss before cochlear implantation?
6. Is there a significant relationship between the presence of the VEMP response and the caloric response before cochlear implantation?
7. Is there a significant relationship between the presence of the VEMP response and the etiology of hearing loss after cochlear implantation?
8. Is there a significant relationship between the presence of the VEMP response and the caloric response after cochlear implantation?
9. Does the amount of time between cochlear implantation and post-cochlear implantation vestibular testing impact the presence of the VEMP response?

**Importance of Study**

While it has been acknowledged that individuals with severe to profound sensorineural hearing loss are at risk for concurrent vestibular dysfunction, little

attention has been given to the effects of vestibular loss in this population. In adults and older children, vestibular dysfunction has been shown to present following implantation as vertigo or imbalance. The implantation surgery itself also has been suggested to lead to reduction or elimination of vestibular function in the unilaterally implanted ear, leading to asymmetrical function, or to reduction or elimination of vestibular function bilaterally in the case of bilateral cochlear implantation (e.g. Buchman et al., 2004; Melvin et al., 2008; Steenerson et al., 2001).

**Scope of Study**

The scope of this study was to evaluate the vestibular systems of individuals with severe to profound sensorineural hearing loss between four and 60 years of age before and after cochlear implantation. The results of this study may be used to further support the evaluation of the vestibular systems of cochlear implant candidates, the evaluation of change in the presence of the VEMP response following implantation, as well as to determine variables that may influence this change.

**Limitations of Study**

The retrospective nature provided a significant limitation to this study. Data were collected as part of the clinical protocol for all cochlear implant candidates evaluated at the University of Colorado Hospital. The retrospective nature of this study created additional variables that would not necessarily be present in a prospective study. While the test protocol for evaluating the vestibular system would remain constant, additional variables, such as the amount of time between implantation and the follow up

evaluation, were not consistent. Additionally, individuals did not always receive testing due to time constraints at follow up appointments.

The VEMP response is only one of a multitude of vestibular tests that may be used to evaluate the vestibular system. By focusing on the VEMP response, this study was limited to evaluating possible changes within the saccule and the inferior branch of CN VIII.

# CHAPTER 2

## REVIEW OF THE LITERATURE

Forty percent of children and adults with severe to profound degrees of sensorineural hearing loss have demonstrated vestibular dysfunction (Buchman et al., 2004; Krause et al., 2009); however, the rate of dysfunction has been variable based on the vestibular end organ under evaluation. Most clinical protocols have provided evaluation of the horizontal semicircular canal, demonstrating the function of one of the five vestibular end organs within the inner ear. Prior to cochlear implantation, 30 to 40% of individuals with severe to profound sensorineural hearing loss have demonstrated abnormal function of the horizontal semicircular canal (Buchman et al., 2004; Krause et al., 2009). Studies have suggested that the saccule has demonstrated abnormal function in approximately 30% of children and adults before implantation (King, 2009; Melvin et al., 2008; Moushey et al., 2010).

Unfortunately, temporal bone studies have indicated that the saccule has been most at risk for damage during implantation (Tien & Linthicum, 2002), requiring additional testing of the inner ear. Because of the increased risk to the sacculae of individuals receiving cochlear implants, knowledge of the status of this end organ, as well as the inferior branch of CN VIII, is beneficial. Adult cochlear implant recipients have demonstrated both subjective and objective vestibular dysfunction after surgery (Steenerson et al., 2001), with 20 to 30% demonstrating decreased horizontal semicircular canal function (Buchman et al., 2004). Saccular testing has demonstrated a change in function in 30 to 40% of cases (King, 2009; Melvin et al., 2008). The

purpose of this review of the literature is to provide literature relevant to the vestibular systems of individuals with severe to profound sensorineural hearing loss and the impact of cochlear implantation on the VEMP response.

**Evolution of the Vestibular System**

Common evolutionary theory indicates that the inner ear most likely developed to monitor rotational and linear head movements relative to gravity over 450 million years ago, making the vestibular system an ancient sensory system (Gray, 1955; Stevens & Warhofsky, 1965). Through evolution, organs within the inner ear took on an additional function—sound detection. The organs commonly involved in sound detection are the saccule, the lagenar (an otolith organ found in birds and fish), or the basilar or amphibian papillae. Generally, the detection of sound by the vestibular end organs focuses on the saccule (Lysakowski & Goldberg, 2004). Although the cochlea obtained primary auditory function in mammals, animal studies have demonstrated that the saccule has retained some auditory function in addition to its role in the vestibular system (Cazals, Aran, & Erre, 1983; McCue & Guinan, 1995; Moffat & Caprianica, 1976; Wit, Bleeker, & Mulder, 1984; Young, Fernández, & Goldberg, 1977).

**Anatomy/Physiology of the Vestibular System**

The inner ear is located within the temporal bone, containing five end organs that detect head acceleration (three semicircular canals, two otolith organs) and one that detects sound. The cochlear and vestibular systems are closely related, as they are derived from the same organ systems tissues. Embryological studies have demonstrated the similar development of the auditory and vestibular systems. The

membranous labyrinth develops in early gestation from the thickened ectoderm, known as the otic placode, leading to the primitive inner ear (Morsli, Choo, Ryan, Johnson, & Wu, 1998). The primitive inner ear divides into dorsal and ventral areas; the ventral area develops into the saccule and the cochlear duct, while the dorsal area leads to the semicircular canals, the utricle, and the endolymphatic duct (Sadler, 2004; Zemlin, 1998).

**Semicircular canals.** The three semicircular canals are aligned in order to have a pair located within the contralateral inner ear. The horizontal semicircular canals are paired together, while the posterior and the contralateral superior semicircular canals are paired (Della Santina, Potyagaylos, Migliaccio, Minor, & Carey, 2005). The horizontal semicircular canals are aligned approximately 20-degrees from the plane connecting the external auditory canal to the lateral canthus. The posterior and superior semicircular canals are aligned approximately 90-degrees from the plane of the horizontal semicircular canals (Della Santina et al., 2005). Since the semicircular canals are not aligned perfectly to earth horizontal or earth vertical, any head rotation stimulates all of the semicircular canals to some degree (Cremers et al., 1998).

The semicircular canals have an enlargement on one end called the ampulla. Located within the ampulla is the cupula, which fills the ampulla and completely cross-sections the semicircular canal (Hillman & McLaren, 1979). The cupula is not attached to the top of the ampulla, but remains in place due to tugor pressure within the fluid-filled semicircular canal. The cupula maintains the same specific gravity as the endolymph filling the canal, allowing the cupula to be non-responsive to linear accelerations

(Scherer & Watanabe, 2001). Beneath the cupula lies the crista, which contains the sensory hair cells and neural fibers. The hair cells (stereocilia, kinocilia) extend into the cupula and transmit the response of cupular displacement through the afferent neural pathway.

The semicircular canals within the vestibular system are responsible for the detection of angular head acceleration and the transmission of that information to the brainstem. The transmission of information is accomplished by initiating the flow of endolymph within the canal due to head movement (inertial force) (Breuer, 1874; Camis & Creed, 1930). When the motion of the endolymph leads to deformation of the cupula, chemical transduction channels are opened or closed, depending on the direction of the deflection of the stereocilia. Movement of the stereocilia towards the kinocilia leads to excitation of the afferent nerve fibers (Hillman & McLaren, 1979; Lysakowski & Goldberg, 2004; McLaren & Hillman, 1979).

**Otolith organs.** The otolith organs of the saccule and the utricle include similar structures to those found within the semicircular canals. The stereocilia of the otolith organs, however, project into a gelatinous substance, on which lies calcium carbonate crystals called otoconia. The otoconia provides inertial mass, providing the otolith maculae with a specific gravity greater than that of the surrounding endolymph.

As previously stated, the semicircular canals are not responsive to changes in linear acceleration. Due to the differences in inertial mass within the maculae of the otolith organs, these organs are able to respond to changes in linear acceleration. In mammals, the otolith organs are linear accelerometers, maintaining head position in the

presence of linear acceleration (Fernández & Goldberg, 1976; Uchino, 1997). The increased inertia within the otolith organs leads to displacement of the sensory cell base. When displacement occurs, the stereocilia move and activate the afferent nerve fibers (Rabbitt, Boyle, & Highstein, 2004). The difference in specific gravity leads to the responsiveness to linear acceleration. The utricle is excited during horizontal linear accelerations, while the saccule responds to the vertical plane (Lysakowski & Goldberg, 2004). The otolith organs are able to determine the direction of linear acceleration, as the otolith maculae are able to sense acceleration primarily within their specific plane. Movement outside of this plane is not sensed (Fernández & Goldberg, 1976; Goldberg, Desmadryl, Baird, & Fernández, 1990).

**Labyrinthine fluids.** Two types of fluids are present within the inner ear: perilymph and endolymph. Perilymph is rich in sodium with few potassium ions, similar to the composition of cerebrospinal fluid. On the other hand, endolymph is rich in potassium ions and low in sodium. The concentration of sodium and potassium ions varies between species (Ghanem, Breneman, Rabbitt, & Brown, 2008).

The fluids are separated within the inner ear by the membranous labyrinth, allowing for two functions. First, since the fluids are enclosed, they are less sensitive to changes in atmospheric pressure, allowing the semicircular canals to detect angular changes in head movement without susceptibility to changes in atmospheric pressure (Yamauchi, Rabbitt, Boyle, & Highstein, 2002). Secondly, the separation of fluids provides an electrochemical gradient, which is necessary for neural transmission (Rask-

Anderson, Tylstedt, Kinnefors, & Schrott-Fischer, 1997; Salt, 2001; Salt & DeMott, 2000).

Hair cells within the vestibular end organs project into the endolymph-filled portion of the labyrinth. Tight junctions between the hair cells and the supporting cells separate the endolymph from the perilymph. The chemical gradient between the two fluids allows for neural transmission. More specifically, the endolymph gradient is responsible for hair cell displacement, while the perilymph gradient is responsible for neural transmission (Art & Fettiplace, 1984; Art, Crawford, Fettiplace, & Fuchs, 1984; Art, Wu, & Fettiplace, 1995; Fuchs & Evans, 1988; Goodman & Art, 1996a; 1996b; He & Dallos, 1999).

**Sensory hair cells.** There are two types of hair cells within the vestibular end organs: type I and type II. In 1965, Wersäll evaluated the semicircular canals of guinea pigs using an electron microscope, describing two types of hair cells within the cristae. Further research demonstrated that hair cell types were located within different regions of the cristae, and varied in size, spacing and morphology (Lindeman, 1969). In the center of the cristae, hair cells were larger and more widely spaced than hair cells located at the edge (Lysakowski & Goldberg, 1997). Similar observations were found within the otolith maculae (Lindeman, 1969; Werner, 1933). Within the striola, the curved landmark within the otolith organs that defines hair cell orientation, hair cells were found to be larger and more widely spaced than in surrounding areas (Lapeyre, Guillaume, & Cazals, 1992; Lindeman, 1969).

Type I hair cells have a flask shape and one calynx nerve ending, which can synapse with up to four nerve fibers. Type I hair cells are found in mammals, birds, and reptiles, but are not found in fish or amphibians (Lysakowski, 1999; Wersäll & Bagger-Sjöbäck, 1974). Type II hair cells have a cylindrical shape and multiple afferent and efferent nerve endings (Lysakowski, Minor, Fernández, & Goldberg, 1995). Interestingly, both hair cell types demonstrate variations in distribution between species. For example, both type I and type II hair cells are found throughout the cristae and the maculae; however, birds and reptiles demonstrate a more constricted distribution of type I hair cells, which are only found within the cristae and the maculae of the utricle (Brichta & Peterson, 1994; Lysakowski, 1999; Rosenhall, 1970).

Both hair cell types have a resting firing rate between 70 and 100 spikes per second (Goldberg & Fernández, 1971; Lysakowski et al., 1995), although type I and type II hair cells have irregular and regular firing rates, respectively. The irregular firing rate of the type I hair cells is more responsive to large head movements and is important in initiating the vestibulo-ocular reflex (Lysakowski et al., 1995; Minor, Lasker, Backous, & Huller, 1999). On the other hand, the regular firing rate of the type II hair cells is responsible for maintaining the vestibulo-ocular reflex (Shubert & Shepard, 2008).

**Afferent neural pathway.** The vestibular nerve (CN VIII) includes two branches. The superior branch innervates the horizontal and superior semicircular canals and the utricle, while the inferior branch innervates the posterior semicircular canal and the saccule (Naito, Newman, Lee, Beykirch, & Honrubia, 1995). There are between 15,000

and 25,000 neural fibers within the vestibular system of humans (López, Honrubia, & Baloh, 1997; Park, Tang, López, & Ishiyama, 2001; Richter, 1980). From the vestibular end organs, afferent nerve fibers travel together until reaching the pontomedullary junction. The afferent nerve fibers of the superior branch synapse in either the superior or medial vestibular nuclei or within the cerebellum (Brodal & Brodal, 1985; Furuya, Kawano, & Shimazu, 1975; Goldberg, 2000). The afferent nerve fibers of the inferior branch synapse within the medial, lateral, or inferior vestibular nuclei (Naito et al., 1995).

**Blood supply.** Blood supply to the vestibular end organs is through the labyrinthine artery. This artery divides, with one branch supplying blood to the superior and horizontal semicircular canals, the utricle, and a portion of the saccule. The other branch further divides to supply the cochlea, the posterior semicircular canal, and the majority of the saccule. Blood drainage follows a similar pathway. The superior vein drains blood from the superior and horizontal semicircular canals and the utricle, while the inferior vein drains the saccule, the posterior semicircular canal, and the cochlea (Baloh & Honrubia, 1990).

## Vestibular Testing Procedures

Cochlear implantation has been demonstrated to lead to additional risk for vestibular dysfunction due to changes in the inner ear structures (Tien & Linthicum, 2002). Because of this additional risk in individuals who may already present with atypical vestibular function, vestibular testing is a reasonable addition to the cochlear implantation evaluation. In order to evaluate the vestibular system clinically, a test

battery should include testing of the horizontal semicircular canal (e.g. caloric testing) and the saccule (i.e. VEMP testing), as well as documentation of subjective balance function. Additional tests of vestibular function are available, such as off-vertical axis testing and posturography; however, these tests are not always available within the clinical setting and normative data are not available for young children (Phillips & Backous, 2002).

**Semicircular canal testing—caloric testing.** In order to assess the function of the vestibular system, caloric testing measures the vestibulo-ocular reflex, and is currently considered to be the gold standard of vestibular testing (Desmond, 2004). The vestibulo-ocular reflex pathway produces compensatory eye movements, known as nystagmus, in the direction opposite head rotation. The nystagmus allows for stable vision during head movement (Fontana & Porth, 2005). The vestibulo-ocular reflex pathway begins within the horizontal semicircular canal and travels along CN VIII to the vestibular nuclei, located within the brainstem, which is the main integrative system for balance (Fontana & Porth, 2005). These primary afferent fibers synapse within the medial and ventrolateral portions of the vestibular nucleus. From here, the motoneurons within the abducens nucleus synapse within the lateral and medial rectus muscles. Importantly, the superior and posterior semicircular canals have similar connective pathways through the vestibulo-ocular system, but these pathways are typically not measured within a clinical battery (Shubert & Shepard, 2008).

The purpose of caloric testing is to evaluate the horizontal semicircular canal and the superior branch of CN VIII by assessing the status of the vestibulo-ocular reflex

response (Desmond, 2004). In order to conduct caloric testing, participants are inclined 30-degrees from horizontal. This test angle aligns the horizontal semicircular canal with the horizontal plane of gravity. Without caloric stimulation, no vestibular response should be elicited, as the cupula within the endolymph is not affected by gravity. Irrigation of the external auditory canal is completed by introducing a medium, such as water, at a temperature above (44-degrees Celsius) or below (30-degrees Celsius) body temperature (D'Agostino, Melagrana, Ravera, & Taborelli, 1999; Desmond, 2004). As the temperature rises or falls, the density of the endolymph changes and induces convection currents within the endolymph. The convection currents mimic head movement (Fontana & Porth, 2005). The brain infers the change in firing rate of the neurons of the ipsilateral horizontal semicircular canal as movement or turning, and produces a compensatory nystagmic response (Desmond, 2004).

Typically, the caloric response is recorded using infrared video recording of the eyes, which is then evaluated by computer software. The calculated peak nystagmic response is compared between the ears in order to determine the presence of unilateral peripheral vestibular loss. A difference in vestibular function of greater than 20 to 25% between sides, as determined by Jonkees formula, represents vestibular asymmetry clinically (Balatsouras et al., 2007; Chang & Young, 2007; Jonkees, Maas, & Phillipszoon, 1962).

***Pediatric considerations.*** Clinical use of caloric testing has not commonly been included in protocols for infants and young children. However, research protocols have indicated that an appropriate caloric response matures over the first few months of life,

becoming diagnostically useful in children as young as 10-months of age (Eviatar & Eviatar, 1979; Melagrana, D'Agostino, Pasqual, & Taborelli, 1996; Ornitz, Atwell, Walter, Hartmann, & Kaplan, 1979; Snashall, 1983; Staller, Goin, & Hildebrandt, 1986). Researchers and an expert panel of physicians have established the caloric response as appropriate for children older than four years of age, as they are generally able to tolerate the procedure (Eviatar & Eviatar, 1978; Fife et al., 2000).

**Otolith testing.** Clinical testing of the otolith organs has been completed with various tests; however, the vestibular evoked myogenic potential (VEMP) response of the saccule and the inferior branch of CN VIII has generally been the only otolith test consistently completed clinically. While evaluation of the complete vestibular system is important for those at risk for vestibular dysfunction, research has demonstrated the saccule as the vestibular end organ most at risk for damage during cochlear implantation (Tien & Linthicum, 2002). Due to this, further discussion regarding the evaluation of the otolith organs will be limited to the saccule and the VEMP response.

## Vestibular Evoked Myogenic Potential Response

Work completed by Tullio in 1938 laid the foundation for the study of the acoustic sensitivity of the vestibular system. His work focused on observing changes in head and eye movements, as well as postural changes, in response to sound in animal models. Von Békésy (1935) previously excluded the cochlea as the source of the vestibular response to sound, believing that these responses were due to fluid displacement within the otolith organs. Animal models were soon established to record electrical responses, which were first described in the pigeon by de Vries and Bleeker

(1949). Human studies followed, extending the work of Wit, Bleeker, and colleagues. For example, Ribaric, Previc, and Kozina (1984) evaluated the frequency following response (FFR) and the middle latency response (MLR) using low frequency bone conducted stimuli in participants with profound sensorineural hearing loss. Both the FFR and MLR were present in participants with known vestibular function, but absent in those with non-responsive vestibular end organs. These results indicated that the FFR and MLR could be mediated by the vestibular system in individuals with known non-functional cochleae, but not in those with absent vestibular function.

In 1958, short latency responses, originally believed to be cortical, were observed at the inion, the most prominent point projecting from the occipital bone at the base of the skull (Geisler, Frishkopf, & Rosenblith, 1958). Further research found that the response was actually myogenic in origin, arising from the vestibulo-collic reflex, as surface electrodes placed on the sternocleidomastoid (SCM) muscle demonstrated a biphasic, short latency, inhibitory response to loud acoustic stimuli (Bickford, Jacobson, Cody, & Thane, 1964; Cody & Bickford, 1969; Colebatch, Halmagyi, & Skuse, 1994; Lim, Clouston, Sheean, & Yiannikas, 1995; Robertson & Ireland, 1995; Townsend & Cody, 1971). Colebatch and colleagues (1994) proposed that this response, termed vestibular evoked myogenic potential (VEMP), included the saccular afferent neural pathway.

Later studies established the use of the VEMP response to demonstrate the function of the saccule and the inferior branch of CN VIII (Akin, Murnane, & Proffitt, 2003; Al-Abdulhadi, Zeitouni, Al-Sebeih, & Katsarkas, 2002; Chen, Young, & Wu, 2000;

Clarke, Schonfeld, & Helling, 2003; Colebatch et al., 1994; Ferber-Viart, Dubreuil, & Duclaux, 1999; Li, Houlden, & Tomlinson, 1999; McCue & Guinan, 1994a; Ochi, Ohashi, & Nishino, 2001; Wang & Young, 2006; Welgampola & Colebatch, 2001). The following description focuses on the cervical VEMP (c-VEMP) response pathway. Note that additional pathways exist, including measurable responses along the vestibulo-spinal pathway. Additionally, measurements of these responses can be made throughout the vestibulo-spinal tract; however, as a majority of the literature and clinical practice currently focuses on the c-VEMP, the remaining discussion will examine this pathway.

**VEMP pathway.** Prior research has demonstrated that the vestibular system, especially the saccule, is responsive to sound. Furthermore, the saccule serves as the primary or secondary auditory organ in many non-mammalian species (Lewis, Baird, Leverenz, & Koyama, 1982; Lowenstein & Roberts, 1951; Moffat & Caprianica, 1976; Popper & Fay, 1973). Additionally, animal studies have demonstrated responses to sound within the range of human hearing occurring within the vestibular neural pathway (Carey, Hivonen, Huller, & Minor, 2004; Curthoys, Kims, McPhedran, & Camp, 2006; McCue & Guinan, 1997; 1995; 1994b; Mikaelian, 1964; Murofushi & Curthoys, 1997; Murofushi, Curthoys, Topple, Colebatch, & Halmagyi, 1995; Young, Fernández, & Goldberg, 1977).

Stimulation of the vestibular end organs by acoustic stimuli depends on which organ is observed, the mode of stimulation, and the status of the inner ear (Halmagyi, Curthoys, Colebatch, & Aw, 2005). For example, the mode of stimulation may or may not affect the vestibular end organ. Semicircular canal neurons have been shown to be

rarely responsive to air-conducted acoustic stimuli (Carey et al., 2004; Mikaelian, 1964; Murofushi & Curthoys, 1997; Murofushi et al., 1995), while otolith neurons have demonstrated increased firing to similar acoustic stimuli (McCue & Guinan, 1997; 1995; 1994a; 1994b; Murofushi & Curthoys, 1997; Murofushi, Curthoys, & Gilcrest, 1996; Murofushi et al., 1995).

The origin of the VEMP response has been determined to be the saccule (Nong, Ura, Kyuna, Owa, & Noda, 2002), with the VEMP pathway describing the function of the saccule and the inferior branch of CN VIII (Ferber-Viart et al., 1999). The afferent pathway, consisting of approximately 4,000 axons, travels from the saccule via the inferior branch of the vestibular nerve to the lateral vestibular nucleus in lower order animals (Bergström, 1973; Colebatch et al., 1994; Markham, 1989). In primates, studies have suggested that the afferent VEMP pathway travels to the medial vestibular nucleus or to the inferior vestibular nucleus (Carleton & Carpenter, 1983; Ferber-Viart, Duclaux, Colleaux, & Dubreuil, 1997; Robertson & Ireland, 1995; Stein & Carpenter, 1967). At this point, the afferent fibers divide into ascending and descending branches.

The ascending fibers travel to the central area of the vestibular complex (Barmack, Baughman, Errico, & Shojaku, 1993; Büttner-Ennever, 1992; Gerrits, 1990). The vestibular nuclei receiving afferent input from the saccule have descending pathways through the spinal motoneurons. The possible efferent pathway includes the lateral vestibulo-spinal tract and the medial vestibulo-spinal tract, as these are both known efferent pathways through the spinal motoneurons. The vestibulo-spinal cells located within the lateral and medial vestibular nuclei contain a majority of axons that

continue along the descending vestibulo-spinal tract. In any case, the motoneurons of both the lateral and medial vestibulo-spinal tracts project into the SCM muscles (Bickford et al., 1965; Sato, Imagawa, Isu, & Uchino, 1997; Uchino et al., 1997).

The cervical VEMP pathway has been shown to be obliterated following the destruction of the medial vestibulo-spinal tract, with auditory thresholds unaffected (Masaki et al., 2002; Matsuzaki & Murofushi, 2002), and has only been documented at the level of the third cervical vertebrae (Masaki et al., 2002). In lower order animals, however, the saccule has been described as an acoustic receptor (Lowenstein & Roberts, 1951; Saidel & Popper, 1986). Because of the presence of an acoustic response within the saccule of lower order animals, researchers have hypothesized that the mammalian saccule has retained some auditory function in addition to its role in balance (Murofushi, Curthoys, & Topple, 1995).

Normal VEMP responses have been observed in participants with absent or severe malformations of the semicircular canals and/or cochlea. Significantly, caloric responses have been atypical in those with normal VEMP responses, indicating that typical saccular function may exist in cases with absent or atypical inner ear structures (Sheykholeslami & Kaga, 2002). Conversely, obliteration of the vestibular end organs using gentamicin has produced absent VEMP responses with no change in auditory thresholds (Matsuzaki & Murofushi, 2002; Yang & Young, 2005). The lack of VEMP responses following gentamicin exposure suggests that the absent VEMP responses were due to vestibular end organ damage and were not impacted by the state of the cochlea, as gentamicin targets the hair cells of the vestibular system (Minor, 1999).

**Test procedure.**

***Electrode placement.*** The optimal site of electrode placement varies, depending on the research protocol. The middle to upper portion of the SCM muscle body has been used in many studies after finding that the p1-n1 waveform was elicited in all adult participants with typical vestibular function (Akin et al., 2003; Cheng & Murofushi, 2001a; 2001b; Colebatch et al., 1994; Ochi et al., 2001; Sheykholeslami, Murofushi, & Kaga, 2001; Welgampola & Colebatch, 2001). The active electrode is placed on the SCM muscle, with the reference electrode above the sternum (Rauch, 2006). Other electrode placements locate the active electrode just below the clavicle, with the reference electrode on the ipsilateral SCM muscle body (Isaradisaikul et al., 2008). By changing the polarity of the reference and active electrodes, the waveform inverts and produces a negative-positive biphasic response. The ground electrode, regardless of the polarity of the reference and active electrodes, is generally placed on the forehead (Isaradisaikul et al., 2008).

***Participant position.*** Various test positions have been used to elicit the VEMP response. Many studies require that the participants sit upright and turn the head to activate the ipsilateral SCM muscle (Rauch, 2006; Rauch, Zhou, Kujawa, Guinan, & Herrmann, 2004). Another test position required that the participant recline to the caloric test position (30-degrees from horizontal). From here, the participant was asked to lift and turn the head to activate the ipsilateral SCM muscle (Isaradisaikul et al., 2008). When comparing these two positions, a reclined position has been shown more likely to elicit a VEMP response and to produce larger p1-n1 inter-peak amplitudes than

a sitting position; however, there may be more risk from fatigue when requiring the participant to lift and turn the head (Wang & Young, 2006).

***Stimuli.*** VEMP responses have been obtained through a variety of stimuli, including air- and bone-conducted acoustic stimuli, skull taps, and galvanic stimulation. Clinically, VEMP responses have generally been obtained using air-conducted stimuli, with various protocols using this stimulus.

The VEMP response has demonstrated frequency tuning, indicating that the saccule is more responsive at specific frequencies. Research into determining the frequency tuning of this response has found that the optimal air-conducted toneburst stimulus was between 500 and 1k Hz, with the peak response at 700 Hz. The peak frequency response produced waveforms at lower thresholds with larger p1-n1 inter-peak amplitudes, indicating increased sensitivity at this frequency (Welgampola & Colebatch, 2001). While a multitude of stimuli have been used to elicit the VEMP response, the most commonly used air-conducted stimulus used in the literature is 500 Hz.

The frequency tuning of the VEMP response has also been evaluated in children. Zhou and colleagues (2009) compared the VEMP responses of both click and 500 Hz toneburst stimuli in children between two and 16 years of age. This study found that 500 Hz produced a more robust VEMP response and required less stimulus intensity than the click stimulus. Due to this, the stimulus discussion will continue with the protocol parameters using 500 Hz toneburst stimuli.

The rate of stimulus presentation has been shown to affect the VEMP response. P1-n1 inter-peak amplitudes were largest when using a five-Hz rate or slower, and were significantly reduced or absent at rates higher than 10 Hz. Additionally, the selection of five-Hz reduced the amount of time that the participant was required to hold the test position, reducing muscle fatigue when compared to one Hz rate protocols (Murofushi, Matsuzaki, & Takegoshi, 2001; Wu & Murofushi, 1999).

When using 500 Hz toneburst stimuli, a rise/fall time of one millisecond (ms) produced VEMP response; however, bone-conducted stimuli may prove to be another useful stimulus. Bone-conducted responses have been obtained by using a bone oscillator or by skull taps using a tendon hammer. The intensity level required to elicit bone-conducted responses has been found to be less than 50 dB, which may provide a more comfortable stimulus for the participant. The optimal frequency to elicit a bone-conducted VEMP response was demonstrated to be between 200 and 250 Hz, lower than when using air-conducted stimuli (Sheykholeslami, Kermany, & Kaga, 2001; Welgampola, Rosengren, Halmagyi, & Colebatch, 2003). The ipsilateral amplitude was 1.5 times greater than in air-conducted responses and the waveform generally occurred one ms earlier (Welgampola et al., 2003). Unfortunately, bone oscillators available within the United States have not been able to produce sufficient stimulus intensity levels to produce reliable VEMP responses clinically (Welgampola & Colebatch, 2001).

**Results.** Typical VEMP responses have been described by numerous studies, and vary depending on protocol and test parameters (e.g. Ackley, Tamaki, Oliszewski, & Inverso, 2004; Akin et al., 2003; Colebatch et al., 1994; Isaradisaikul et al., 2008).

Figure 1 demonstrates a typical VEMP response. Figure 2 demonstrates an absent VEMP response.

Figure 1

*Typical VEMP Response.*

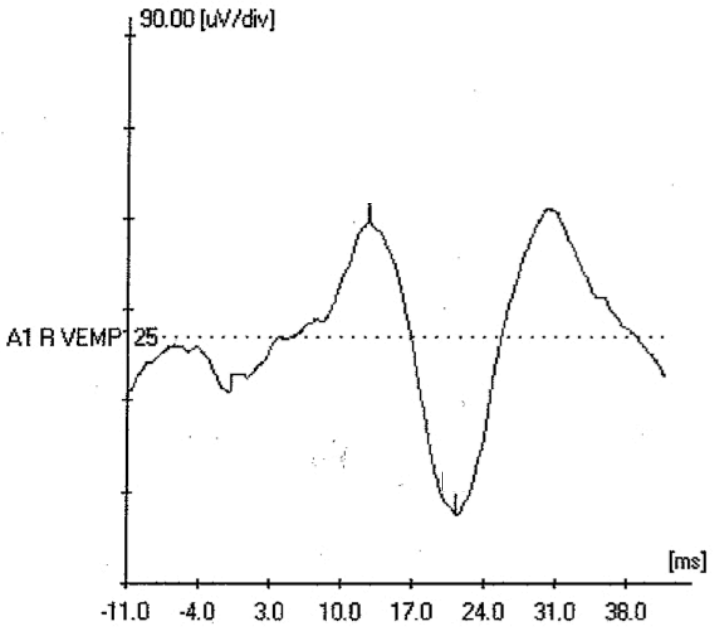
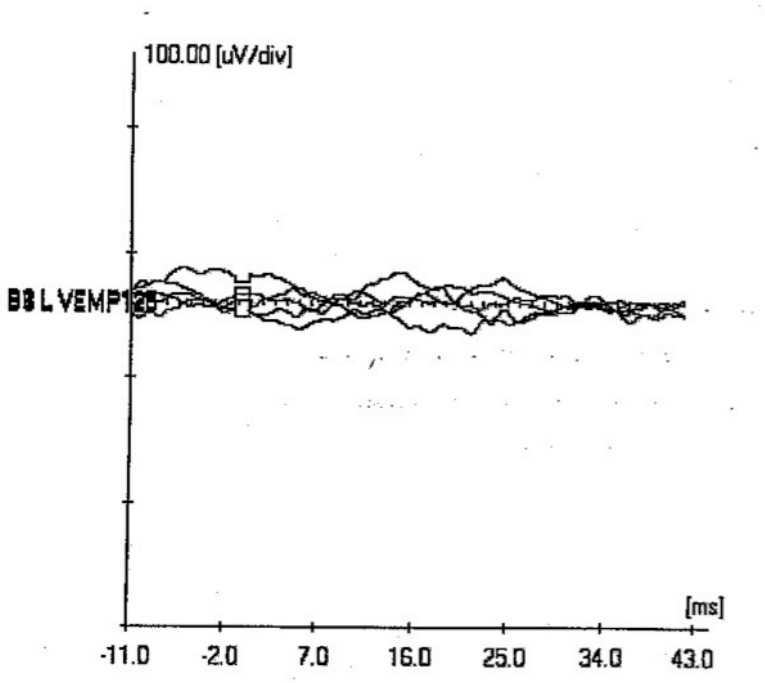


Figure 2

*Absent VEMP Response.*




***Latency.*** In adults, the initial positive peak (p1) occurs at a latency of approximately 13 ms, with the following negative peak (n1) at approximately 23 ms (Akin et al., 2003; Bickford et al., 1964; Colebatch et al., 1994; Ferber-Viart, 1999; Rauch, 2006). Clinically, however, variability in latency has been noted and actual latency values for p1 and n1 have varied based on the protocol. For example, Murofushi and colleagues (2001) found mean p1 and n1 latencies of 11.8 ms (*SD* = 0.86) and 20.8 ms (*SD* = 2.2), respectively. Using these data, the maximum cutoff values for typical latencies in adults would be 13.5 ms and 25.2 ms for p1 and n1. Once latencies exceeded these cutoff values, the response would be considered prolonged and would possibly indicate a lesion along the neural pathway. Using these normative

data may lead to improper diagnosis of prolonged latencies when using a different protocol, demonstrating the importance of clinically developed normative data.

Additionally, adult latency values cannot be used when describing the responses of infants and toddlers due to anatomical and maturational variations. For example, Kelsch and colleagues (2006) reported that, while the average latency response for a group of children between three and 11 years of age were 11.3 ms and 17.3 ms for p1 and n1, respectively, the range of latency values of p1 (8.3 – 14.4 ms) and n1 (14.8 – 21.9 ms) was larger than typically experienced with adult data. Furthermore, this study found that children between three and five years of age demonstrated significantly shorter latencies than experienced in older children and adults.

Latencies have been shown to vary between sides, although the difference has not been significant (Zhou & Cox, 2004). Young and Kuo (2004) evaluated the side differences for the VEMP response when using binaural stimulation (500 Hz, 95 dB nHL), finding no significant differences. When using air-conducted stimuli, the inter-aural differences for p1 and n1 latencies were 1.6 ms (*SD* = 1.6) and 1.8 ms (*SD* = 1.5), respectively (Basta, Todt, & Ernst, 2005). Asymmetrical responses have been noted in the literature, involving p1 more often than n1; however, these asymmetries have been insignificant. The following formula may be used to analyze latency symmetry: [(right – left)/(right + left)]. Normative values for latency symmetry are 0.45 (0.20 – 1.13) for p1 and 0.8 (0.45 – 1.39) for n1 (Brantberg & Fransson, 2001). Unlike some auditory evoked potential responses, latency was demonstrated to not vary significantly with decreased intensity levels. While most studies have reported appropriate latencies at

the maximum stimulus intensity level, reporting them at threshold has not produced significant differences (Akin, Murnane, & Proffitt, 2003).

Additionally, latency values have not been shown to vary between individuals with sensorineural hearing loss and those with normal hearing. Ackley et al (2004) reported on the VEMP responses of 15 adults with sensorineural hearing loss, comparing their VEMP responses to the results of 15 adults with normal hearing. These results indicated that the VEMP response to 500 Hz toneburst stimuli (95 dB nHL) were not significantly different between these two groups. P1 latencies averaged 15.01 ms (*SD* = 2.32) and 15.08 ms (*SD* = 1.85), with n1 latency averages of 23.16 ms (*SD* = 2.25) and 23.95 ms (*SD* = 2.45) for participants with sensorineural hearing loss and normal hearing thresholds, respectively. These findings indicated that no significant differences in the latencies of p1 and n1 were expected due to the status of the cochlea.

***Threshold.*** Initial VEMP response studies were conducted with binaural stimulation of 120 dB SPL. These studies reported that as the intensity level of the stimulus decreased, the amplitude, and therefore the threshold of the response, decreased. The VEMP response disappeared by 90 – 100 dB SPL (Bickford et al., 1964; Geisler et al., 1958). More recently, studies have found inter-subject thresholds to vary between 75 and 90 dB SPL (Colebatch et al., 1994; Rauch, 2004). While children are also expected to demonstrate variations in threshold, pediatric studies have not evaluated this response characteristic. Instead, the waveform characteristics have been reported at a specific intensity level (e.g. 95 dB nHL) (Chang & Young, 2007; Kelsch et al., 2006).

The actual threshold of the response is not significant unless the threshold is noted at levels significantly below expected values and the individual is suspected of presenting with a third window disorder, such as superior semicircular canal dehiscence. This condition may present with thresholds 20 dB lower than expected (Streubel, Cremer, Carey, Weg, & Minor, 2001). Recently, Chen and colleagues (2009) reviewed computed tomography (CT) scans of children over three years of age with diagnosed hearing loss, finding 18 of the 131 presenting with dehiscence of the superior or posterior semicircular canal. While this diagnosis may prove to be important in children, the use of the VEMP response has not been used to successfully diagnose semicircular canal dehiscence in children. At this point, when evaluating the VEMP response in individuals with sensorineural hearing loss and no complaints associated with superior semicircular canal dehiscence, it is not clinically relevant to evaluate threshold.

***Amplitude.*** The inter-peak amplitude of the VEMP response varies, depending on factors such as the stimulus intensity level and the level of SCM muscle contraction. For example, as the stimulus intensity level increases, the p1-n1 inter-peak amplitude increases (Rauch, 2004). Unfortunately, the p1-n1 inter-peak amplitude of the VEMP response has demonstrated large inter-subject variability in both adult and pediatric participants. A study conducted by Wu, Young, and Murofushi (1999) found the average p1-n1 inter-peak amplitude for responses obtained using 500 Hz toneburst stimuli (2 ms rise/fall, 2 ms plateau, 5 Hz rate) to average 54.6 mV (*SD* = 28.9). On the other hand, Isaradisaikul and colleagues (2008) reported the average p1-n1 inter-peak

amplitude to be 160.71 mV (*SD* = 101.11) when using a similar protocol (1 ms rise/fall, 2 ms plateau, 5 Hz rate). The variability in the p1-n1 inter-peak amplitudes reported between these two studies has been attributed to the test position used to obtain these results. Wu et al (1999) conducted this evaluation using a head turn while sitting, while Isaradisaikul et al (2008) required that the participant lift and turn the head from a recumbent position, increasing the contraction of the SCM muscle.

When evaluating the symmetry of the response, the following formula has been used: [(right – left)/(right + left)]. The mean amplitude ratio value was reported as 0.21 (0.08 – 0.36) for adults. Amplitude ratios outside of this range have been considered asymmetrical (Brantberg & Fransson, 2001; Young, Wu, & Wu, 2001). In cases without monitoring of SCM muscle contraction, such as obtained when evaluating children, amplitude ratios greater than 0.5 have been considered asymmetrical (Tribukait et al., 2004). Note that amplitude ratios can only be calculated when responses at the same stimulus intensity levels have been recorded for both sides.

Individuals with significant sensorineural hearing loss have presented with decreased p1-n1 inter-peak amplitudes, possibly due to inner ear anomalies or dysfunction concurrent with hearing loss. Ackley et al (2004) reported differences in the p1-n1 inter-peak amplitude between participants with sensorineural hearing loss and those with normal hearing thresholds. While not statistically significant, the average p1-n1 inter-peak amplitude for those with sensorineural hearing loss (*M* = 65.44 mV, *SD* = 51.04) trended lower than the results obtained for those with normal hearing thresholds (*M* = 91.07 mV, *SD* = 30.29). In addition, the variability of this response in the group

with sensorineural hearing loss suggested that additional factors, such as etiology of hearing loss, might influence the VEMP response.

**Pediatric considerations.** The "soft" signs of vestibular dysfunction in children are not evaluated when using traditional vestibular test protocols, as the posterior semicircular canal and the otolith organs, which are essential for appropriate gross motor skill development, are not evaluated. Studies have examined the use of the VEMP response in determining the vestibular status of children of various ages in order to provide additional information about the vestibular system.

While obtaining the VEMP response typically requires active participant involvement, the VEMP response has been documented as present and robust in infants within one week following birth, with children between 2- and 5-days of life demonstrating VEMP responses in 40% of cases (Chen et al., 2007). Young, Chen, Hsieh, and Wan (2009) also evaluated newborns between 2- and 5-days of life, as well as between 6- and 13-days of life. The results found that, although none of the infants were found to have a response at day two, nearly all (92%) demonstrated reliable responses by day five. No significant changes in latency were noted between the groups, aside from infants at day three, who demonstrated prolonged latencies when compared to infants at day five ($p < .05$).

Jin, Nakamura, Shinjo, and Kaga (2006) evaluated preschool-aged children ($M$ = 3.8 years, $SD$ = 1.4) with normal hearing and balance function. Average p1 and n1 latencies for stimuli presented at 95 dB nHL were defined as 10.5 ms ($SD$ = 0.5) and 16.1 ms ($SD$ = 1.3), respectively, with p1-n1 inter-peak amplitude reported to average

181.0 mV (*SD* = 90.0). Kelsch and colleagues (2006) used the VEMP response to evaluate 30 children with normal hearing ranging in age from three to 11 years of age. The average p1 and n1 latencies were 11.3 ms (8.3 – 14.4 ms) and 17.3 ms (14.8 – 21.9 ms), respectively, with the average p1-n1 inter-peak amplitude average of 122.2 mV (20.9 – 351.6 mV). Additionally, Chang and Young (2007) evaluated another set of children with normal hearing and vestibular function, ranging from five to 15 years of age. Within this study, all children presented with VEMP responses (500 Hz, 95 dB nHL, 1 ms rise/fall, 2 ms plateau) with p1 and n1 latencies of 11.9 ms (*SD* = 0.8) and 18.0 ms (*SD* = 1.5), respectively. The p1-n1 inter-peak amplitude averaged 118 mV (*SD* = 52). Note the differences in the range of latencies between these studies. It appears as though latency values shift with age, particularly before five years of age; however, the small number of children within each of these studies as well as differences in protocol may have lead to the variability of these results.

The results of these studies have indicated that children ranging in age from a few days of life through adolescence have VEMP responses similar to those found in adults, although decreased latencies may be evident in children less than five years of age. Overall, these studies found that children older than five years of age demonstrated an absence of developmental effects of the VEMP response (Bath, Harris, & Yardley, 1998; Colebatch et al., 1994; Ochi et al., 2001; Welgampola & Colebatch, 2001; Wu & Murofushi, 1999; Wu et al., 1999).

Children with sensorineural hearing loss may present with anomalies of additional inner ear structures, including the otolith organs. Zhou and colleagues (2009)

evaluated 23 children between two and 16 years of age with bilateral sensorineural hearing loss. Etiology was reported in 13 children (*GJB2* mutation: $n = 7$, congenital CMV: $n = 3$, bacterial meningitis: $n = 1$, Cogan syndrome: $n = 1$, auditory neuropathy spectrum disorder: $n = 1$). Within this study, 21 of the 23 children demonstrated abnormal VEMP findings. These children were more likely to present with higher threshold levels or lower p1-n1 inter-peak amplitudes when compared to control data. Additionally, these children were also at risk for demonstrating absent VEMP responses.

When compared to control data, VEMP responses obtained for children with sensorineural hearing loss differed from responses obtained from children with normal hearing thresholds for p1-n1 inter-peak amplitude, but not for p1 or n1 latency. Results from this study by Zhou and colleagues (2009) are summarized in Table 1.

Table 1

*Comparison of VEMP Responses for Typical and Hearing Loss Children.*

| VEMP Parameter | Normal Hearing *M* (*SD*) | Hearing Loss *M* (*SD*) |
|---|---|---|
| Threshold (dB nHL)*** | 74.45 (4.86) | 82.74 (13.59) |
| Amplitude (mV)** | 77.40 (53.36) | 58.21 (53.71) |
| P1 Latency (ms) | 14.65 (0.72) | 15.14 (1.71) |
| N1 Latency (ms) | 21.54 (1.01) | 22.40 (3.40) |

*Notes:* Zhou et al (2009); $^{**}p < .01$; $^{***}p < .001$

The authors hypothesized that this difference in response indicated abnormal saccular function, as the threshold and p1-n1 inter-peak amplitude indicated decreased function for those with sensorineural hearing loss. On the other hand, the inferior branch of the vestibular nerve appeared to function similarly between groups, as no latency differences were noted. This study confirms previous findings regarding the differences between individuals with hearing loss and those with normal hearing as reported by Ackley et al (2004).

**Vestibular Function in Cochlear Implant Candidates**

In adults and older children, cochlear implantation has been reported to lead to both subjective and objective vestibular dysfunction, with reports of vertigo and imbalance following surgery (Steenerson et al., 2001). Little follow up information is available for young children; however, the majority of studies presented below provided information regarding adults and older children who were able to verbalize changes in vestibular function.

**Surgical effects of cochlear implantation.** Anatomically, the saccule is the closest vestibular end organ to the basal turn of the cochlea, the location that research has previously demonstrated as the area most at risk for damage during implantation (Gstoettner et al., 1997; Tien & Linthicum, 2002). Adult temporal bone studies have suggested that the saccule may be at risk for damage or collapse in over half of cases following implantation. Damage found within the saccule has included vestibulofibrosis and saccular membrane distortion, which may have lead to possible vestibular dysfunction following implantation (Tien & Linthicum, 2002; Todt, Basta, & Ernst, 2008).

In general, electrode arrays are inserted into the cochlea to the point of first resistance. Gstoettner and colleagues (1997) evaluated adult temporal bones in order to describe cochlear trauma found in those with cochlear implants. In nine of the 11 temporal bones, trauma was noted in multiple structures, including the basilar membrane, spiral ligament, osseous spiral lamina, organ of Corti, and Reissner's membrane. Within these 11 temporal bones, seven showed minimal damage, with two demonstrating severe trauma, such as membrane rupture or electrode displacement.

One of the main indications for inner ear trauma is pressure during insertion, as contact pressure is created between the electrode array and tissues within the cochlea. Generally, this trauma has been documented at the base of the cochlea, as the electrode array directs towards the modiolus. Contact with the outer cochlear wall at this perpendicular angle may force the electrode array upward, tearing the basilar membrane (Rebscher et al., 1999). While all electrode array insertions pose a risk for tearing the basilar membrane, most basilar membrane trauma has been documented at approximately 10-millimeters (mm) from the round window, correlating with an angle of approximately 175-degrees. Additional areas of pressure during implantation have been described between 180-degrees and 270-degrees and 405-degrees and 450-degrees (Verbist et al., 2009).

Tien and Linthicum (2002) evaluated 11 pairs of adult human temporal bones, each with one cochlear implant. Eight of the 11 pairs demonstrated damage either ipsilateral to the cochlear implant or bilaterally. The authors described these differences as possibly attributed to the trauma of electrode insertion or other surgically induced

condition. Seven of the temporal bone pairs presented with atypical vestibular end organs bilaterally; however, more damage was noted on the side ipsilateral to the cochlear implant in five cases. The location of damage within the vestibular system was noted to be mostly within the saccule ($n = 7$), with a smaller proportion demonstrating damage within the utricle ($n = 3$) and semicircular canals ($n = 2$). The authors reported that the majority (75%) of those with damage to the basal turn of the cochlea also demonstrated damage to the vestibular end organs, usually isolated to the saccule.

Histopathologically, this study found saccular membrane distortion, as well as fibrosis (calcification, ossification) and reactive neuromas within the saccule. Overall, the incidence of damage to the vestibular system in this study was 54.5%, which was hypothesized to be due to prior damage to the vestibular end organs and surgically induced effects following insertion, infection, interruption of inner ear fluid homeostasis, or vascular changes. Importantly, 25% of these cases demonstrated damage in the implanted ear only, indicating that surgical intervention may have been the cause of the noted damage to the vestibular structures and that concurrent vestibular anomalies may have lead to increased damage due to the implantation surgery. The authors evaluated clinical examination results when available, finding that the histopathological changes noted post-mortem did not correspond to clinical presentation. In this study, that while 54.5% demonstrated noted changes to the structures within the vestibular system, only 33% reported difficulty with balance following surgery, suggesting that damage to the inner ear does not always correlate with reported of vestibular dysfunction.

After combining the information provided by Tien and Linthicum (2002) and Gstoettner and colleagues (1997) studies, it is unclear if the minimal damage noted in the majority of cochleae following implantation correlated with damage to the vestibular end organs. Because studies evaluating the effects of electrode array insertion have been conducted on cadavers and through modeling of the inner ear, the exact cause of inner ear trauma has not been determined definitively. The effect of insertion trauma, however, has been assumed since the front end of the electrode array becomes deformed following contact with the wall of the cochlea (Chen et al., 2003), and the side ipsilateral to the cochlear implant has presented with increased damage to inner ear structures post-mortem (Tien & Linthicum, 2002). Additionally, maintaining the electrode array within the scala tympani may lead to reduction in vestibular trauma as well, not only due to reduced trauma to the inner ear structures, but to possible changes in the endolymphatic system as well (Tien & Linthicum, 2002).

Long-term dizziness has been found in those with dysfunction of the saccule, which may possibly be attributed to the trauma of electrode insertion (Basta, Todt, Goepel, & Ernst, 2008). On the other hand, Enticott and colleagues (2006) reported some interesting results evaluating the placement of the electrode array within the cochlea. All participants in this study were implanted with the Nucleus 24 multichannel internal implant. While the surgeon completing the surgery was not found to be a significant predictor for vestibular dysfunction, the actual placement of the electrode (typical placement versus tight or loose placement within the cochlea) was significant. Those with a typical electrode placement were 3 times less likely to demonstrate

subjective vestibular complaints than those with other electrode placements. Although no differences have been noted between internal device manufacturers, surgical technique has been suggested to influence vestibular function following implantation. For instance, testing of the horizontal semicircular canal and the saccule, along with subjective reports of vestibular dysfunction following surgery have suggested that the use of a round window approach in place of an anteroposterior approach may more likely preserve the functionality of the vestibular end organs (Todt et al., 2008).

**Subjective vestibular function.** Subjectively, up to 74% of adult cochlear implant recipients have experienced symptoms of vestibular dysfunction over time (Enticott et al., 2006; Steenerson et al., 2001); however, the duration of dizziness, as well as its onset, has varied and does not always correlate with clinical measures (Bonucci, Filho, Mariotto, Amantini, & Alvarega, 2008). For example, Buchman and colleagues (2004) evaluated subjective dizziness, finding that although reported dizziness increased at one month following implantation, this change was not significant for the overall group. Interestingly, those with reported dizziness before implantation tended to report increased dizziness following surgery.

Additional work on this topic by Ito (1998) provided data regarding the timing of the dizziness following implantation. Of 55 adult cochlear implant recipients, 58% reported dizziness during the first two weeks following implantation, with a small proportion (8%) with dizziness that did not begin until at least one month following surgery, indicating a possible delay in subjective symptoms. Jacot and colleagues (2009) reported on a series of 89 pediatric participants ranging in age from seven

months to 16 years. During the first 24 to 48 hours following implantation, 27% (*n* = 24) demonstrated vestibular symptoms including dizziness and vomiting. Another study, provided by Kubo et al (2001), evaluated a number of adult cochlear implant recipients (*n* = 94) and found that approximately half (*n* = 46) reported dizziness following implantation. The duration of dizziness within this group was approximately one month following surgery and was characterized by transient or positional nystagmus, or by continual lightheadedness or unsteadiness. The authors hypothesized that these recipients possibly presented with perilymphatic fistula, although a diagnosis of benign paroxysmal positional vertigo (BPPV) could also have been possible in these cases. Unfortunately, this diagnosis was not evaluated. Thirty-four percent (*n* = 15) demonstrated delayed onset of dizziness. These adults reported rotary vertigo with abrupt onset, beginning between one and three months following implantation in 28% and greater than 12 months in 44%. The authors reported characteristics similar to those found in Meniere's disease, as these participants also reported tinnitus and changes in hearing sensitivity during these periods of time.

Filipo et al (2006) further supported this work by evaluating 21 adult participants ranging in age from 18 to 79 years. Following implantation, 21.4% (*n* = 3) experienced rotary vertigo lasting a few days, while 42.8% (*n* = 6) reported unsteadiness. Interestingly, 21.4% (*n* = 3) presented with documented BPPV and were treated with positioning maneuvers. Additional evaluation of the presence of BPPV in participants following cochlear implantation found that BPPV may present in less than 10% of adult

cochlear implant recipients within the first year following surgery; however, this condition has not been reported in children (Zanetti, Campovecchi, Belzanelli, & Pasini, 2007).

**Semicircular canal function.** Historically, the horizontal semicircular canal has been the most widely evaluated vestibular end organ. Correlations between semicircular canal function and degree of hearing loss have been demonstrated in children with congenital hearing loss, indicating that individuals with pure tone averages of greater than 98 dB only demonstrate caloric function in 20% of cases. For pure tone averages less than 90 dB, caloric responses have been demonstrated in 80% of individuals (Sandberg & Terkildsen, 1965). Cochlear implantation has been shown to disrupt the function of the horizontal semicircular canal, as evaluated by caloric or sinusoidal harmonic acceleration (rotary chair) testing, in up to half of cases (Brey et al., 1994; Enticott et al., 2006; Huygen et al., 1995; Krause et al., 2009; Vibert et al., 2001). The reasons for this change in vestibular function include surgically induced changes to the labyrinth (e.g. trauma, deafferentation, vestibulopathy, inflammation), as well as electrical stimulation of the vestibular system by the cochlear implant (Buchman et al., 2004).

Buchman and colleagues (2004) evaluated 86 pediatric (2 – 16 years) and adult (18 – 87 years) cochlear implant candidates. Before implantation, no difference was found in the caloric response between sides (ipsilateral: $M$ = 31-degrees/second, $SD$ = 21-degrees/second; contralateral: $M$ = 29-degrees/second, $SD$ = 25-degrees/second). Twenty-three percent of the sides selected for implantation and 32% of the contralateral sides demonstrated hyporeflexia or areflexia before surgery, indicating poor or absent

horizontal semicircular canal function. Seven participants demonstrated unilateral horizontal semicircular canal function; five of those were implanted on the side with documented vestibular function.

At four months following implantation, 29% ($n$ = 8) demonstrated a loss of caloric function of at least 21-degrees/second; however, no significant change was noted in the group overall in the caloric response of either ear until two years post-implantation. At this point, the implanted sides demonstrated significantly decreased caloric responses ($p$ = .048). The authors concluded that the risk of vestibular loss, as evaluated by horizontal semicircular canal function, was approximately 20% for adults and 10% for children. The difference in the rate of change was contributed to variations in etiology of inner ear dysfunction.

Another study conducted by Szirmai and colleagues (2001) evaluated the effect of cochlear implantation on the horizontal semicircular canals of 60 children and adults aged five to 59 years. Before implantation, caloric testing found that 25% ($n$ = 15) demonstrated typical responses, with the remaining presenting with bilateral areflexia (46.7%), unilateral areflexia (15%), and directional preponderance (13.3%). Following implantation, caloric testing was repeated, finding changes in the caloric response in half of these participants. It must be pointed out that air-caloric irrigation was conducted for these evaluations, which may have increased the prevalence of areflexia and variability noted within this group.

Recently, Krause and colleagues (2009) evaluated 49 adults in order to compare pre- and post-implantation function of the vestibular system. Before implantation ($n$ =

45), 53% (*n* = 24) demonstrated typical responses for sinusoidal harmonic acceleration testing, with 40% (*n* = 18) demonstrating asymmetrical responses and four percent with bilateral areflexia. Caloric testing found similar responses bilaterally before implantation (ipsilateral: *M* = 24.4-degrees/second; contralateral: *M* = 25.4-degrees/second).

Following implantation (*n* = 42), 29% (*n* = 12) demonstrated typical function to sinusoidal harmonic acceleration testing, while 38% (*n* = 16) had significant directional preponderance to the stimulation. Caloric testing demonstrated a mean response of 14.5-degrees/second (*SD* = 11.6) for the implanted side and 20.4-degrees/second (*SD* = 15.0) for the contralateral side. For the implanted side, 36% (*n* = 15) presented with typical responses, with the remaining participants demonstrating decreased (21%) or absent (36%) responses. Thirty-two percent (*n* = 12) demonstrated a change from typical to reduced or absent function following implantation. Although one-third of participants demonstrated a significant decrease in function following implantation, no significant correlation was found between vestibular function and age, gender, implant manufacturer, or surgeon. The authors hypothesized that vestibular dysfunction was due to traumatic insertion of the electrode array, perilymph loss, endolymphatic hydrops, or electrical stimulation of the vestibular system.

***Pediatric considerations.*** Researchers have also evaluated children to determine the risk for decreased horizontal semicircular canal function following implantation. Shinjo, Jin, and Kaga (2007) evaluated 20 children between three and eight years of age who qualified for cochlear implantation based on degree of hearing loss (pure tone average $\geq$ 87.5 dB HL). This study found that 15% (*n* = 3) presented

with typical vestibular function, while 50% (*n* = 10) demonstrated absent caloric responses. Jacot and colleagues (2009) also evaluated vestibular function in 224 pediatric cochlear implant recipients ranging in age from seven months to 16 years. Within this group, half presented with vestibular dysfunction as described by canal and otolith function. Following implantation (*n* = 71), 60% (*n* = 43) had no change in horizontal semicircular canal function, while 9.8% (*n* = 7) demonstrated areflexia, 16.9% (*n* = 12) demonstrated hyporeflexia, and seven percent (*n* = 5) demonstrated hyperreflexia. The reported caloric dysfunction of 30% within this group was consistent with adult findings indicating that approximately one-third of adults demonstrate decreased caloric responses following implantation (Enticott et al., 2006). A group of these children (*n* = 27) was re-evaluated between three months and seven years post-implantation. The majority (63%) demonstrated stable responses over this time period; however, 18.8% improved responses and 11% decreased. No correlations between vestibular function and degree of hearing loss were noted. The results of this study indicated that the risk of vestibular dysfunction following cochlear implantation was approximately 10%, similar to results found in adults (Buchman et al., 2004). Furthermore, the authors hypothesized that the high-risk period for developing vestibular dysfunction following cochlear implantation was three months. Also, a percentage of children (18.8%) demonstrated improved responses over time, indicating that vestibular dysfunction may be transient in some cases.

Caloric function has long been utilized to evaluate the vestibular function in individuals at risk; however, the high variability reported in individuals following cochlear

implantation may be due to multiple variables. First, variability in the study sample may lead to various vestibular outcomes depending on the composition of the sample. This is difficult to evaluate due to the large proportion of individuals with unknown etiology of hearing loss. Second, while caloric testing is considered to be the gold standard for vestibular evaluation, not all studies utilize the same vestibular test protocol, adding to confusing outcomes. Also, the effects of vestibular compensation may lead to various results. Compensation may occur soon following implantation and may proceed at various rates over time. Finally, there have been some reports of interaction with the contralateral ear that may influence changes in the vestibular response ipsilateral to the cochlear implant (Filipo et al., 2006).

**Otolith function.** Because temporal bone studies have described the saccule as the vestibular end organ most at risk for injury following cochlear implantation (Tien & Linthicum, 2002), evaluation of the saccular response should provide more insight into changes in vestibular function due to surgery. Melvin and colleagues (2008) evaluated the VEMP responses in 36 ears of adult participants (23 – 69 years of age) before and after cochlear implantation. Nineteen ears were tested using the VEMP response, finding 37% ($n = 7$) absent prior to implantation. Following implantation, 31% ($n = 5$) demonstrated a change in VEMP response to either absent or increased threshold. Additionally, Jacot and colleagues (2009) reported that otolith function was impaired in 45% of pediatric participants prior to cochlear implantation. Follow up testing found that otolith function was changed in 55% of cases ($n = 39$ of 71). Unfortunately, this study

did not differentiate between the otolith test battery, and included changes in both the VEMP and the off vertical axis rotation (OVAR) test protocols in its results.

King (2009) evaluated participants with cochlear implants in order to determine differences in the VEMP response before implantation, as well as six weeks and six months post-implantation. This group consisted of children and adults ranging in age from 12 to 86 years. Before implantation ($n$ = 74 ears), 35% ($n$ = 26) demonstrated absent VEMP responses, while 2.7% ($n$ = 2) were asymmetric in p1-n1 inter-peak amplitude. At six weeks post-implantation, 17 previously typical sides were re-evaluated to determine the rate of change in the status of the VEMP response. At this time, 35% ($n$ = 6) had no response and 5.9% ($n$ = 1) exhibited increased thresholds. Sixteen sides that demonstrated typical VEMP responses before implantation were re-evaluated at six months, finding that 43.75% ($n$ = 7) had no VEMP response and 6.25% ($n$ = 1) showed decreased p1-n1 inter-peak amplitude.

Interestingly, one side that demonstrated an absent VEMP response at six weeks proved to have a present response at six months, although this response had reduced p1-n1 inter-peak amplitude when compared to the non-implanted side. Furthermore, one side that demonstrated reduced p1-n1 inter-peak amplitude at six weeks post-implantation demonstrated amplitudes within the typical range at the six-month evaluation. No differences were noted between surgical techniques (round window versus anterior-inferior cochleostomy) or between internal device manufacturers. Although EMG monitoring of the SCM muscle was not conducted for these comparisons, it is interesting to note the possibility of improvement over time,

suggesting that the VEMP response post-implantation may be reduced due to transient alteration of the labyrinth.

While the behavioral effect of otolith dysfunction has not been fully evaluated for individuals with severe to profound sensorineural hearing loss, recent data presented by Fujimoto and colleagues (2010) demonstrated different clinical presentations for individuals following unilateral vestibular loss. Within this group of 108 participants, six percent ($n$ = 2) of 33 participants with absent VEMP responses were unable to successfully complete a task requiring them to stand on a foam rubber with the eyes closed. A further six percent ($n$ = 2) of 31 participants who presented with atypical caloric function and typical VEMP responses were unable to complete this task. Interestingly, 20% ($n$ = 9) of 44 participants with both atypical caloric and VEMP responses demonstrated an inability to complete this task, suggesting a decrease in behavioral function when both organ systems were documented with diminished function. However, while those with absent caloric responses demonstrated abnormal function on all postural tasks, those with absent VEMP responses appeared to be less affected, demonstrating the difference in the effect of specific end organ loss on cases of unilateral vestibular loss.

***Pediatric considerations.*** Appropriate functionality of the otolith organs has been demonstrated as a requirement for appropriate gross motor development in young children. For example, the postural stability required to stand on one foot has been described as an otolith-mediated ability (Worchel & Dallenbach, 1950). Shall (2009) evaluated 30 pediatric participants with severe to profound sensorineural hearing loss

with the VEMP response and the Movement Assessment Battery for Children (Movement ABC), a test of manual dexterity and balance. Nineteen participants had at least one cochlear implant, with five of those bilaterally implanted. VEMP responses were present bilaterally in only four participants, who also demonstrated typical function using the Movement ABC. The authors reported that those participants with VEMP responses on one side tended to demonstrate better behavioral scores than those with absent VEMP responses bilaterally; however, those with VEMP responses unilaterally or bilaterally absent demonstrated significantly difference Movement ABC scores than children with normal hearing. The effect of even unilaterally absent VEMP responses appears to correlate with significantly poorer behavioral vestibular function.

Importantly, this study utilized bone-conducted VEMP responses while holding a bone oscillator to the ipsilateral mastoid process. Lack of appropriate placement and pressure may have lead to decreased VEMP responses, particularly considering that 21.21% ($n$ = 7) children demonstrated unilateral VEMP responses and 66.67% ($n$ = 22) were absent bilaterally. Also of importance was the presence of concurrent cochlear implantation in over half of these participants ($n$ = 19), with bilateral implantation in 73.68% ($n$ = 14). Adult studies have indicated that cochlear implantation may eliminate the VEMP response following surgery, further complicating these results. Interestingly though, evaluation of the current status of the VEMP response regardless of the effect of implantation in relation to behavioral measures of balance function is an important research question that has not yet been evaluated.

# CHAPTER 3

# METHOD

## Participants

Forty total cases were available for analysis. Twenty-two participants between four and 15 years of age were evaluated prior to implantation. Four participants demonstrated absent VEMP responses bilaterally and were not scheduled for post-implantation vestibular testing. Of the remaining 18 pediatric participants, five received post-implantation vestibular testing. Sixteen participants between 20 and 60 years of age were evaluation prior to implantation. Participants were excluded if their age was greater than 60 years of age due to the possibility of decreased VEMP response rate in this age group (Su, Huang, Young, & Cheng, 2004) and if their age was less than four years of age due to variations in test protocol. Two adult participants were bilaterally implanted, one simultaneously and one sequentially, and are included as separate cases. One participant demonstrated bilaterally absent VEMP responses at pre-implantation vestibular testing and was not scheduled for post-implantation vestibular testing. Of the remaining 15 adult participants, 11 completed post-implantation vestibular testing.

## Instrument

**Overview/description.** The vestibular evoked myogenic potential (VEMP) response has been demonstrated to be a reliable measure to evaluate the state of the saccule and the inferior branch of CN VIII. As the risk of surgical insult to the vestibular system has focused on the saccule (Tien & Linthicum, 2002), the VEMP response

provides valuable information regarding the status of this pathway before and after cochlear implantation.

**Control data.** Control data were collected in a previous VEMP reliability study, which evaluated 20 adult participants ($M$ = 31.8 years, $SD$ = 6.2) with normal hearing and vestibular function (Isaradisaikul et al., 2008). All participants demonstrated typical middle ear function (Jerger, 1970) on the day of testing and had the ability to maintain appropriate SCM muscle contraction.

**Test protocol.** This test protocol followed a previously established protocol for this clinical site (Isaradisaikul et al., 2008). Air-conducted 500 Hz toneburst stimuli (5 Hz rate, 1 ms rise/fall, 2 ms plateau) were presented monaurally through ER3A insert earphones (Etymotic Research, Elk Grove, IL, USA). The EMG signal was amplified and filtered from 10 to 1500 Hz using the Blackman gating function. VEMP responses were obtained using a Biologic Explorer or a Biologic Navigator evoked potential system (Bio-Logic Systems Corporation, Mundelein, IL, USA). Disposable, pre-gelled snap electrodes (Viasys Healthcare Inc., Madison, WI, USA) were used. Reference electrodes were placed on the upper third of the SCM muscle body. The active electrode was placed on the top of the sternum, just below the clavicle, with the ground electrode on the forehead. Participants were placed in a recumbent position, 30-degrees from horizontal.

***Reliability.*** Studies have found reliably present VEMP responses in nearly all participants with typical vestibular function (Cody & Bickford, 1969; Colebatch et al., 1994; Isaradisaikul et al., 2008; Robertson & Ireland, 1995). Because of the influence of

additional variables, such as tonic muscle contraction, on the presence of these responses, reliability has been evaluated. The EMG level of SCM muscle contraction has been demonstrated as vital to producing reliable VEMP responses; higher EMG levels reduce the variability of p1-n1 inter-peak amplitude (Akin et al., 2004). Although EMG level has been demonstrated as important for obtaining reliable results for specific parameters, monitoring this level has not been of clinical relevance when evaluating the presence of the VEMP response alone (Isaradisaikul et al., 2008).

**Procedure**

**Description of data collection.** Data were collected retrospectively for all available participants who were evaluated for cochlear implantation between 2007 and 2010. Both pediatric and adult cochlear implant candidates received vestibular testing, as appropriate by age, before and after cochlear implantation as described by clinical protocol. This retrospective data was collected by chart review.

**Location of data collection.** All data was collected in the Department of Otolaryngology at the University of Colorado Hospital.

**Test procedure.** Pediatric and adult cochlear implantation candidates were evaluated for vestibular dysfunction in order to determine the status of the vestibular system before surgery. Participants were evaluated as part of the cochlear implantation candidacy protocol at the University of Colorado Hospital. The vestibular protocol for cochlear implant candidates included monothermal (warm) caloric screening and VEMP response testing before and after cochlear implantation.

Before vestibular testing, tympanometry was conducted to ensure that the middle ear space was functioning within typical limits and that no perforations were noted in the tympanic membrane (Jerger,1970). Additionally, the middle ear was screened to ensure that no conductive component contributed to atypical VEMP responses. Participants were placed in a recumbent position, 30-degrees from horizontal. Monothermal (warm) caloric irrigations were conducted bilaterally according to clinical protocol.

Participants were maintained in the test position above (30-degrees inclined from horizontal). The skin was prepped with Nuprep gel (Weaver and Company, Aurora, CO, USA) and disposable, pre-gelled snap electrodes were placed onto the skin. The reference electrode was located on the upper third of the SCM muscle body, with the active electrode on the sternum, just below the clavicle. The ground electrode was placed on the forehead.

Air-conducted 500 Hz toneburst stimuli (5 Hz rate, 1 ms rise/fall, 2 ms plateau) were presented monaurally through ER3A insert earphones (Etymotic Research, Elk Grove, IL, USA). Approximately 100 stimuli were presented for each trial, with at least two trials presented at the maximum intensity level. The initial presentation level was 115 dB SPL, which was the maximum output level for the Biologic Explorer evoked potential unit. The Biologic Navigator evoked potential unit has a maximum output level of 125 dB SPL for this test protocol. The participants were instructed to relax the neck between trials to ensure that fatigue did not influence the response. While threshold of

the VEMP response was determined for many of these participants, this study focused only on the presence of the VEMP response at high stimulus intensity levels.

Following cochlear implantation, participants were re-evaluated at least two months post-implantation with the test battery repeated as described above. The sound processor was removed from the head during VEMP testing to reduce electrical interference.

**Data recording.** All data collected retrospectively was transferred to a datasheet, per accepted protocol, and then added to the SPSS (v13.0) database (SPSS Inc., Chicago, IL, USA) for analysis. Once data was added to the database, the datasheets were destroyed.

**Informed consent/COMIRB.** Authorization for this retrospective chart review was obtained through the Colorado Multiple Institute Review Board (COMIRB) (#05-1110). This protocol allowed for waived informed consent due to the lack of risk provided by the retrospective nature of this analysis. The University of Colorado at Boulder Institute Review Board authorized COMIRB as the entity of record, approved under protocol #0510.8.

## Data Analysis

**Question 1: Is the VEMP response present in both ears after cochlear implantation?** The null hypothesis for this question was that the participants with present VEMP responses bilaterally would retain these present VEMP responses bilaterally following cochlear implantation.

***Participants.*** This question evaluated 10 adult participants with available pre- and post-cochlear implantation data. Participants were required to have bilaterally present VEMP responses and no concurrent cochlear implant at the time of the pre-implantation evaluation. No pediatric participants were available for this analysis.

***Data analysis.*** All participants were required to have present VEMP responses bilaterally before cochlear implantation in order to be included in this analysis. Data was categorized by ear (implant, non-implant) in order to complete this analysis. VEMP responses were categorized as present or absent. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the VEMP response by ear was calculated. No statistical analyses were conducted due to the small number of participants for this question.

**Question 2: Is the proportion of the presence of the VEMP response significantly lower after cochlear implantation when compared to the proportion of the presence of the VEMP response before implantation?** The null hypothesis for this question was that the proportion of presence of the VEMP response after cochlear implantation would not be significantly lower than the proportion of presence of the VEMP response before cochlear implantation.

***Participants.*** This question evaluated 15 participants with available pre- and post-cochlear implantation data. Five pediatric participants and 10 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17.

***Data analysis.*** All participants with pre- and post-cochlear implantation vestibular testing were evaluated. Only the implanted ear was included in this analysis, with VEMP responses characterized as present or absent. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the VEMP response for the implanted ear was calculated. The significance was tested using McNemar's change test, $\alpha = .05$.

**Question 3: Is the presence of the VEMP response in cochlear implant candidates significantly lower than the presence of the VEMP response for individuals with normal hearing?** The null hypothesis for this question was that there would be no significant difference between the proportion of present VEMP responses between the normal hearing group and the cochlear implant group before implantation.

***Participants.*** This question evaluated 40 cochlear implant cases (22 pediatric, 18 adult), as well as 20 adult participants with normal hearing who were evaluated during a previous study (Isaradisaikul et al., 2008).

***Data analysis.*** All participants with pre-cochlear implantation vestibular testing were evaluated. Only the ear selected for implantation was included in this analysis due to the high proportion of participants with pre-existing cochlear implants contralaterally. VEMP responses were characterized as present or absent. This data was compared with the right side data available for typically hearing adults (Isaradisaikul et al., 2008). Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the VEMP response was calculated for each group. The significance was tested using Fisher's exact test, $\alpha = .05$.

**Question 4: Is the presence of the VEMP response in cochlear implant recipients significantly lower than the presence of the VEMP response for individuals with normal hearing?** The null hypothesis for this question was that the proportion of present VEMP responses would not be significantly different between the normal hearing group and the cochlear implant group.

***Participants.*** This question evaluated 15 participants with available post-cochlear implantation data. Five pediatric participants and 10 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17. These data were compared to 20 adults with normal hearing from a previous study (Isaradisaikul et al., 2008).

***Data analysis.*** All participants with post-cochlear implantation vestibular testing were evaluated. Only the implanted ear was included for this analysis. VEMP responses were characterized as present or absent. These data were compared to the right side data available for adults with normal hearing (Isaradisaikul et al., 2008). Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the VEMP response was calculated. The significance was tested using Fisher's exact test, $\alpha$ = .05.

**Question 5: Is there a significant relationship between the presence of the VEMP response and the etiology of hearing loss before cochlear implantation?** The null hypothesis for this question was that there would be no significant relationship

between the presence of the VEMP response and the etiology of hearing loss before cochlear implantation.

***Participants.*** This question evaluated 22 pediatric and 18 adult cases. Four cases were excluded from this analysis due to lack of information regarding the etiology of hearing loss, reducing the total number of cases for analysis to 36.

***Data analysis.*** All participants with pre-cochlear implantation VEMP responses were evaluated. Because of the multiple etiologies and the high percentage of unknown etiology within this group, hearing loss was categorized as congenital or acquired/progressive and compared to the presence of the VEMP response before implantation. Additionally, this question analyzed the data based on inner ear anomalies as evaluated by computed tomography (CT) scan, with imaging coded as typical or atypical per physician report. Data was analyzed using SPSS (v13.0). The relative frequency of various etiologies was calculated; however, significant was calculated by analyzing the etiology of hearing loss as congenital or acquired/progressive. Another comparison analyzed the significance of inner ear anomalies, described as typical or atypical. Data were analyzed by age category of the participant as well. The significance was tested using Fisher's exact test, $\alpha = .05$.

**Question 6: Is there a significant relationship between the presence of the VEMP response and the caloric response before cochlear implantation?** The null hypothesis for this question was that there would be no significant relationship between the presence of the VEMP response and the presence of the typical caloric response before implantation.

***Participants.*** This question evaluated 11 pediatric and 18 adult cochlear implant cases with available VEMP and caloric data.

***Data analysis.*** All participants with pre-cochlear implantation VEMP and caloric responses were evaluated. VEMP data was categorized as present or absent, while caloric testing results were categorized as typical or atypical as described by clinical protocol. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the typical caloric response as compared to the presence of the VEMP response was calculated. The significance was tested using Fisher's exact test, $\alpha$ = .05.

**Question 7: Is there a significant relationship between the presence of the VEMP response and the etiology of hearing loss after cochlear implantation?**

The null hypothesis for this question was that there would be no significant relationship between the presence of the VEMP response and the etiology of hearing loss after cochlear implantation.

***Participants.*** This question evaluated 15 participants with available post-cochlear implantation data. Five pediatric participants and 10 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17.

***Data analysis.*** All participants with post-cochlear implantation VEMP responses were evaluated. Because of the multiple etiologies and the high percentage of unknown etiology within this group, the data was also categorized into congenital or acquired/progressive hearing loss. Data was analyzed using SPSS (v13.0). The

relative frequency of various etiologies was calculated; however, significance was calculated by analyzing the etiology of hearing loss as congenital or acquired/progressive. The significance was tested using Fisher's exact test, $\alpha = .05$.

**Question 8: Is there a significant relationship between the presence of the VEMP response and the caloric response after cochlear implantation?** The null hypothesis for this question was that there would be no significant relationship between the presence of the VEMP response and the presence of the typical caloric response after cochlear implantation.

***Participants.*** This question evaluated 11 participants with available post-cochlear implantation data. Two pediatric participants and 9 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 13.

***Data analysis.*** All participants with post-cochlear implantation VEMP responses and caloric responses were evaluated. VEMP data was categorized as present or absent, while caloric testing results were categorized as typical or atypical as described by clinical protocol. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the typical caloric response as compared to the presence of the VEMP response was calculated. The significance of this comparison was calculated using Fisher's exact test, $\alpha = .05$.

**Question 9: Does the amount of time between cochlear implantation and post-cochlear implantation vestibular testing impact the presence of the VEMP response?** The null hypothesis for this question was that there would be no difference

between the rate of presence of the VEMP response in cases evaluated at two months post-implantation and those evaluated later than two months post-implantation.

***Participants.*** This question evaluated 15 participants with available post-cochlear implantation data. Five pediatric participants and 10 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17.

***Data analysis.*** All participants with post-cochlear implantation VEMP responses were evaluated. Participants were categorized into two groups, depending on the duration post-implantation that follow up vestibular testing was completed. Clinical protocol indicated that follow up vestibular testing was to be completed at the two-month post-cochlear implantation appointment; however, participants were often tested later. Therefore, participants were categorized as evaluated at two months or evaluated at later than two months. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the VEMP response for the implanted ear for each time category was calculated. The significance was tested using Fisher's exact test, $\alpha = .05$.

# CHAPTER 4

# RESULTS

## Participants

Participants in this study included children between four and 15 years of age and adults between 20 and 60 years of age. All participants presented with severe to profound sensorineural hearing loss bilaterally and were evaluated for cochlear implant candidacy in the Department of Otolaryngology at the University of Colorado Hospital. All cochlear implant candidates were scheduled for evaluation of vestibular function, as indicated by clinical protocol. Unfortunately, additional factors, including test equipment availability, time available for follow up testing at subsequent sound processor programming appointments, and fatigue of the participant often hindered post-implantation vestibular testing.

Thirty-eight total participants (40 total cases) were available for analysis. Twenty-two participants between four and 15 years of age were evaluated prior to implantation. Four participants demonstrated absent VEMP responses bilaterally and were not scheduled for post-implantation vestibular testing. Of the remaining 18 pediatric participants, 27.78% ($n = 5$) received post-implantation vestibular testing. Sixteen participants between 20 and 60 years of age were evaluated prior to implantation. Two adult participants were bilaterally implanted, one simultaneously and one sequentially, and were included as separate cases, leading to 18 total adult cases. One participant demonstrated bilaterally absent VEMP responses at pre-implantation vestibular testing and was not schedule for post-implantation vestibular testing. Of the

remaining 15 adult participants, 73.33% (*n* = 11) completed post-implantation vestibular testing.

**Demographic characteristics.**

***Age.*** Participants in this study included children between four and 15 years of age (*M* = 7.73) and adults between 20 and 60 years of age (*M* = 39.05). Twenty-two pediatric participants were available for analysis; eighteen adult cases were evaluated.

***Gender.*** Cases in this study included 15 males and 25 females. For the pediatric group, 31.8% (*n* = 7) were male and 68.2% (*n* = 15) were female. The adult cases were more evenly distributed, with 44.44% (*n* = 8) male and 55.56% (*n* = 10) female. Note that two females were included twice in the adult group, leading to more female cases. No significant difference was found in the total group when evaluating the effect of gender on the pre-implantation VEMP response (Fisher's exact test, *p* = .081), as well as in the adult group (Fisher's exact test, *p* = .556) or the pediatric group (Fisher's exact test, *p* = .187). Based on this comparison, gender was collapsed and not considered in further analyses.

***Internal device.*** Three internal devices were implanted within these participants (*n* = 33), manufactured by two companies. Overall, Device A was used in 54.55% (*n* = 18) with Device B in 27.27% (*n* = 9). The remaining 18.18% (*n* = 6) of participants were implanted with Device C. The pediatric cases (*n* = 17) were implanted with Device A or Device B, each with 41.12% (*n* = 7) of the sample. The remaining 17.65% (*n* = 3) were implanted with Device C. Statistical analyses found no significant difference between the use of the three internal devices and the presence of the VEMP response following

implantation (Pearson's Chi-Squared, $p = .128$). Based on this, internal device was not considered in further analyses. Table 2 describes the internal devices used in these cases.

Table 2

*Internal Devices Used in Cochlear Implantation.*

| Internal Device | Total ($n = 33$) | Pediatric ($n = 17$) | Adult ($n = 16$) |
|---|---|---|---|
| Device A | 54.55% ($n = 18$) | 41.12% ($n = 7$) | 68.75% ($n = 11$) |
| Device B | 27.27% ($n = 9$) | 41.12% ($n = 7$) | 12.5% ($n = 2$) |
| Device C | 18.18% ($n = 6$) | 17.65% ($n = 3$) | 18.75% ($n = 3$) |

***Concurrent cochlear implantation.*** A large proportion of these participants were implanted previously and were under evaluation for a second cochlear implant. Overall ($N = 40$), 45% ($n = 18$) were implanted unilaterally at the time of pre-implantation vestibular testing, leading to exclusion of data from analyses. Interestingly, 68.18% ($n = 15$) of pediatric cases were under evaluation for the contralateral cochlear implant, while only 16.67% ($n = 3$) of adults were previously implanted. A significant difference was found between the proportion of previous cochlear implantation between the age categories (Fisher's exact test, $p = .001$). Table 3 demonstrates the proportion of cases that were implanted concurrent to the pre-implantation vestibular evaluation.

Table 3

*Cases Demonstrating Previous Cochlear Implantation.*

| Previous Implant | Total (*N* = 40) | Pediatric (*n* = 22) | Adult (*n* = 18) |
|---|---|---|---|
| Yes | 45% (*n* = 18) | 68.18% (*n* = 15) | 16.67% (*n* = 3) |
| No | 55% (*n* = 22) | 31.82% (*n* = 7) | 83.33% (*n* = 15) |

***Evoked potential unit.*** Two evoked potential units were utilized in order to collect data for these analyses. Overall (*N* = 40), 67.5% (*n* = 27) were evaluated using the Biologic Navigator evoked potential system, with the remaining 32.5% (*n* = 13) evaluated with the Biologic Explorer device. Sixty-eight percent (*n* = 15) of the pediatric cases (*n* = 22) were evaluated with the Biologic Navigator. The remaining seven cases were evaluated with the Biologic Explorer. The adult cases (*n* = 18) were evaluated with the Biologic Navigator in 66.67% (*n* = 12) of cases and with the Biologic Explorer in 33.33% (*n* = 6). Four cases without concurrent cochlear implantation presented with absent VEMP responses at the pre-implantation evaluation. Of these cases, two were evaluated with the Biologic Explorer, and two with the Biologic Navigator. No significant difference was found when comparing the results of pre-implantation VEMP response testing and the evoked potential unit used for the total group (Fisher's exact test, $p$ = .184). Additionally, no significant difference was found for the adult group (Fisher's exact test, $p$ = .682) or the pediatric group (Fisher's exact test, $p$ = .077). Post-implantation results were similar, finding no significant difference when comparing the

results of post-implantation VEMP response testing and the evoked potential unit for the total group (Fisher's exact test, $p = .453$) or the adult group (Fisher's exact test, $p = .682$). The pediatric group was not evaluated for this comparison due to the small number of post-implantation VEMP results ($n = 5$). Based on these results, VEMP responses obtained with both the Biologic Navigator and the Biologic Explorer were collapsed for further analyses. Table 4 describes the equipment used to evaluate the pre-implantation cases. Table 5 describes the equipment used to evaluate the post-implantation cases.

Table 4

*Evoked Potential Equipment Utilized for Pre-Implantation Evaluation.*

| Equipment | Total ($N = 40$) | Pediatric ($n = 22$) | Adult ($n = 18$) |
|---|---|---|---|
| Navigator | 67.5% ($n = 27$) | 68.18% ($n = 15$) | 66.67% ($n = 12$) |
| Present VEMP | 92.6% ($n = 25$) | 93.3% ($n = 14$) | 91.7% ($n = 11$) |
| Absent VEMP | 7.4% ($n = 2$) | 6.7% ($n = 1$) | 8.3% ($n = 1$) |
| Explorer | 32.5% ($n = 13$) | 31.82% ($n = 7$) | 33.33% ($n = 6$) |
| Present VEMP | 76.9% ($n = 10$) | 57.1% ($n = 4$) | 100.0% ($n = 6$) |
| Absent VEMP | 23.1% ($n = 3$) | 42.9% ($n = 3$) | -- |

*Note:* -- indicates no data available

Table 5

*Evoked Potential Equipment Utilized for Post-Implantation Evaluation.*

| Equipment | Total (*n* = 17) | Pediatric (*n* = 5) | Adult (*n* = 12) |
|---|---|---|---|
| Navigator | 82.4% (*n* = 14) | 80.0% (*n* = 4) | 83.3% (*n* = 10) |
| Present VEMP | 57.1% (*n* = 8) | 100.0% (*n* = 4) | 40.0% (*n* = 4) |
| Absent VEMP | 42.9% (*n* = 6) | -- | 60.0% (*n* = 6) |
| Explorer | 17.6% (*n* = 3) | 20.0% (*n* = 1) | 16.7% (*n* = 2) |
| Present VEMP | 33.33% (*n* = 1) | -- | 50.0% (*n* = 1) |
| Absent VEMP | 66.67% (*n* = 2) | 100.0% (*n* = 1) | 50.0% (*n* = 1) |

*Note:* -- indicates no data available

**Question 1: Is the VEMP response present in both ears after cochlear implantation?**

**Participants.** This question evaluated 10 adult participants (*M* = 38.7 years of age, *SD* = 14.18) with available pre- and post-cochlear implantation VEMP response testing. Participants were required to have bilaterally present VEMP responses and no concurrent cochlear implantation at the time of the pre-implantation evaluation. No pediatric participants were available with pre- and post-cochlear implantation data that did not also have concurrent cochlear implantation and were therefore excluded.

**Data analysis.** All participants were required to have present VEMP responses prior to cochlear implantation in order to be included in this analysis. Data was categorized by ear (implanted, non-implanted) in order to complete this analysis. Also

note that one adult participant was included twice in this analysis, as this participant was bilaterally implanted simultaneously. Data was analyzed using SPSS (v13.0).

***Adult group.*** Ten total adult cases were analyzed for this question. For those 10 cases with present VEMP responses prior to surgery, five demonstrated present VEMP responses on the implanted side following surgery. The contralateral side ($n = 9$) demonstrated present VEMP responses in all cases, except for the simultaneous implantation case. This participant demonstrated one present VEMP response and one absent VEMP response following surgery.

**Question 2: Is the proportion of the presence of the VEMP response significantly lower after cochlear implantation when compared to the proportion of the presence of the VEMP response before implantation?**

**Participants.** This question evaluated 17 cases ($M = 29.35$ years of age, $SD = 18.03$) with available pre- and post-cochlear implantation data. Five pediatric and 12 adult cases were analyzed. Note that 10 adult participants were used for this analysis, but as two of these participants were bilaterally implanted, both sides were analyzed separately.

**Data analysis.** All cases were required to have present VEMP responses in the ear to be implanted at the pre-implantation evaluation. Only the ear to be implanted was included in this analysis, with VEMP responses characterized as present or absent. Data was analyzed using SPSS (v13.0).

***Total group.*** All of these participants presented with present VEMP responses prior to cochlear implantation. Following surgery, VEMP responses were present in

52.9% (*n* = 9) and absent in 47.1% (*n* = 8). A significant difference between the pre- and post-cochlear implant conditions when evaluating the presence of the VEMP response, *p* = .008.

***Adult group.*** Twelve adult cases (*M* = 37.83 years of age, *SD* = 14.20) were evaluated for this comparison. Following surgery, VEMP responses were present in 41.7% (*n* = 5) and absent in 58.3% (*n* = 7). A significant difference was noted between the pre- and post-cochlear implantation conditions when evaluating the presence of the VEMP response, *p* = .016.

***Pediatric group.*** Five pediatric cases (*M* = 9.0 years of age, *SD* = 3.54) were evaluated for this comparison. Following surgery, VEMP responses were present in 80% (*n* = 4) and absent in 20% (*n* = 1). Due to the small number of participants in this category, no statistical analysis was conducted. Table 6 provides the results for this question for all participants.

Table 6

*Comparison of VEMP Response for Implanted Side Pre- and Post-Implantation.*

| Implant Condition | Total (*n* = 17) | Adult (*n* = 12) | Pediatric (*n* = 5) |
|---|---|---|---|
| Pre-Implantation | | | |
| Present VEMP | 100.0% (*n* = 17) | 100.0% (*n* = 12) | 100.0% (*n* = 5) |
| Absent VEMP | -- | -- | -- |
| Post-Implantation | | | |
| Present VEMP | 52.9%** (*n* = 9) | 41.7%* (*n* = 5) | 80.0% (*n* = 4) |
| Absent VEMP | 47.1% (*n* = 8) | 58.3% (*n* = 7) | 20.0% (*n* = 1) |

*Note: *p* < .05, **p* < .01; -- indicates no data available

**Question 3: Is the presence of the VEMP response in cochlear implant candidates significantly lower than the presence of the VEMP response for individuals with normal hearing?**

**Participants.** Thirty-eight cochlear implant candidates (22 children, 16 adults) were available for analysis. Two adult participants were implanted bilaterally and evaluated on each side, bringing the total number of cases to 40. This question evaluated 40 total cochlear implant cases, as well as 20 adult participants with normal hearing and vestibular function who were evaluated during a previous study (Isaradisaikul et al., 2008).

**Data analysis.** Prior to cochlear implantation, five participants demonstrated absent VEMP responses bilaterally. One of these participants with bilaterally absent

VEMP responses at the pre-implantation evaluation also presented with concurrent cochlear implantation. Eighteen participants presented with a cochlear implant unilaterally, and were under evaluation for contralateral implantation; however, only the ear to be implanted was evaluated for this analysis.

***Total group.*** Of the sides to be implanted, 12.5% ($n$ = 5) of the VEMP responses were absent prior to implantation. The remaining 87.5% ($n$ = 35) were present at this evaluation. When compared to a group of adults with normal hearing and vestibular function, a non-significant difference was noted between these two groups, $p$ = .159.

***Adult group.*** Of the 18 sides to be implanted, 5.56% ($n$ = 1) of the VEMP responses were absent prior to implantation, with the remaining 94.44% ($n$ = 17) present at this evaluation. When compared to a group of adults with normal hearing and vestibular function, a non-significant difference was noted between these two groups, $p$ = .474.

***Pediatric group.*** Of the 22 sides to be implanted, 18.18% ($n$ = 4) of the VEMP responses were absent prior to implantation, with the remaining 81.82% ($n$ = 18) present at this evaluation. When compared to a group of adults with normal hearing and vestibular function, a non-significant difference was noted, $p$ = .109. Table 7 provides the results of these comparisons.

Table 7

*Comparison of VEMP Response between Hearing and Hearing Loss Categories.*

| Age Category | *n* | Present VEMP Pre-CI |
|---|---|---|
| Total | 40 | 87.5% (*n* = 35) |
| Adult | 18 | 94.44% (*n* = 17) |
| Pediatric | 22 | 81.82% (*n* = 18) |

**Question 4: Is the presence of the VEMP response in cochlear implant recipients significantly lower than the presence of the VEMP response for individuals with normal hearing?**

**Participants.** This question evaluated 15 participants with available post-cochlear implantation data. Five pediatric participants and 10 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17. These data were compared to 20 adults with normal hearing from a previous study (Isaradisaikul et al., 2008).

**Data analysis.** Following cochlear implantation, one participant demonstrated absent VEMP responses bilaterally. Note that this participant was bilaterally implanted, and that the previously implanted side was noted to be absent at the pre-implantation evaluation. Only the ear that was implanted was included in this analysis.

***Total group.*** Of these 17 cases, 52.94% ($n = 9$) demonstrated present VEMP responses, with 47.06% ($n = 8$) demonstrating absent VEMP responses ipsilateral to the cochlear implant. When compared to a group of adults with normal hearing and vestibular function, a significant difference was noted between these groups, $p = .001$.

***Adult group.*** Following cochlear implantation, 12 adult cases were available. Within this group, 41.67% ($n = 5$) presented with VEMP responses concurrent to the cochlear implant, with 58.33% ($n = 7$) demonstrated absent responses. When compared to a group of adults with normal hearing and vestibular function, a significant difference was noted between these two groups, $p < .001$.

***Pediatric group.*** Following cochlear implantation, five children were available for vestibular testing. Of the five sides to be evaluated, 80% ($n = 4$) of the VEMP responses were present after implantation. The remaining 20% ($n = 1$) was absent following surgery. Due to the small number of cases within this age group, no statistically analysis was completed. Table 8 provides the results from these comparisons.

Table 8

*Comparison of VEMP Responses Post-Implantation to Control Group.*

| Age Category | *n* | Present VEMP Post- CI |
|---|---|---|
| Total | 17 | 52.9%** (*n* = 9) |
| Adult | 12 | 41.67%*** (*n* = 5) |
| Pediatric | 5 | 80.0% (*n* = 4) |

*Note: **p* < .01, ****p* < .001

**Question 5: Is there a significant relationship between the presence of the VEMP response and the etiology of hearing loss before cochlear implantation?**

**Participants.** This question evaluated 22 pediatric and 18 adult cases. Four cases were excluded from this analysis due to lack of information regarding the etiology of hearing loss, reducing the total number of cases for analysis to 36.

Within this group, 16 cases demonstrated acquired/progressive forms of hearing loss, 20 were described as congenital hearing loss, and four did not provide information. Within the acquired/progressive hearing loss category, nine cases described unknown etiology for hearing loss, with two cases of hyperbilirubenemia, two cases of large vestibular aqueduct syndrome (LVAS), and one case each of congenital cytomegalovirus (CMV) infection, meningitis infection, and Meniere's disease. Five pediatric participants reported acquired/progressive hearing loss. The etiology was described as congenital CMV infection (*n* = 1), LVAS (*n* = 1), and unknown etiology (*n* = 3). The remaining 11 adult cases were described with etiologies including

hyperbilirubinemia (*n* = 2), LVAS (*n* = 1), meningitis infection (*n* = 1), Meniere's disease (*n* = 1), and unknown etiology (*n* = 6).

Within the congenital hearing loss category (*n* = 20), 10 cases described unknown etiology for hearing loss, with nine cases of hearing loss attributed to genetic causes (*GJB2*-related hearing loss, Waardenburg syndrome, family history of hearing loss), and one case of maternal German measles. Fourteen pediatric cases reported congenital hearing loss. The etiology was described as due to genetic causes (*GJB2*-related hearing loss, Waardenburg syndrome, family history of hearing loss) in the majority of cases (*n* = 8) with the remaining cases described as unknown (*n* = 6). The six adult cases with congenital hearing loss were described with etiologies including genetic causes (unknown syndrome, family history of hearing loss) (*n* = 2), maternal German measles (*n* = 1), and unknown etiology (*n* = 3). Table 9 describes the etiology of hearing loss for these cases.

Table 9

*Etiology of Hearing Loss.*

| Etiology | Total (*n* = 36) | Adult (*n* = 17) | Pediatric (*n* = 19) |
|---|---|---|---|
| Unknown | 58.3% (*n* = 21) | 58.8% (*n* = 10) | 57.9% (*n* = 11) |
| Congenital CMV | 2.8% (*n* = 1) | -- | 5.3% (*n* = 1) |
| LVAS | 5.6% (*n* = 2) | 5.9% (*n* = 1) | 5.3% (*n* = 1) |
| Meningitis | 2.8% (*n* = 1) | 5.9% (*n* = 1) | -- |
| German Measles | 2.8% (*n* = 1) | 5.9% (*n* = 1) | -- |
| Hyperbilirubinemia | 5.6% (*n* = 2) | 11.8% (*n* = 2) | -- |
| Meniere's Disease | 2.8% (*n* = 1) | 5.9% (*n* = 1) | -- |
| Waardenburg | 2.8% (*n* = 1) | -- | 5.3% (*n* = 1) |
| *GJB2* | 5.6% (*n* = 2) | -- | 10.5% (*n* = 2) |
| Family History | 11.1% (*n* = 4) | 5.9% (*n* = 1) | 15.8% (*n* = 3) |

*Note:* -- indicates no data available

Additionally, information regarding the morphology of the inner ear was available for 24 cases, with three cases demonstrating abnormal inner ear morphology as described by CT scan. These participants were diagnosed with LVAS (*n* = 2) and superior semicircular canal dehiscence (*n* = 1). Ten pediatric cases had documented inner ear information, with one demonstrating abnormal imaging due to LVAS. Fourteen adult cases had documented inner ear information available, with two demonstrating

abnormal inner ear morphology due to LVAS and superior semicircular canal dehiscence. Table 10 describes the inner ear morphology within these groups.

Table 10

*Inner Ear Morphology.*

| Inner Ear Morphology | Total (*n* = 24) | Adult (*n* = 14) | Pediatric (*n* = 10) |
|---|---|---|---|
| Atypical | 12.5% (*n* = 3) | 14.28% (*n* = 2) | 10.0% (*n* = 1) |
| Typical | 87.5% (*n* = 21) | 85.71% (*n* = 12) | 90.0% (*n* = 9) |

**Data analysis.** Prior to cochlear implantation, four cases demonstrated absent VEMP responses bilaterally. Analyses were conducted by evaluating the stability of hearing loss (congenital, acquired/progressive) and the presence of inner ear anomaly (typical, atypical) in comparison to the presence of the VEMP response prior to implantation. The VEMP response was categorized as present or absent.

***Total group.*** Prior to implantation, four cases demonstrated absent VEMP responses bilaterally. Within this group, all four cases were described as having acquired/progressive hearing loss. The etiology of hearing loss was unknown in three cases, and attributed to congenital CMV in one case. The cases presenting with bilaterally present VEMP responses prior to implantation were categorized as having acquired/progressive hearing loss in 41.17% (*n* = 7), with diagnosis of unknown etiology (*n* = 3), Meniere's disease (*n* = 1), meningitis infection (*n* = 1), hyperbilirubinemia (*n* = 2), and LVAS (*n* = 1). When the presence of the VEMP response was compared to

hearing loss stability (congenital, acquired/progressive), a significant difference was noted between the hearing loss stability categories, $p = .031$.

When evaluating the presence of inner ear anomalies, as noted on CT scan, 87.5% ($n = 21$) were noted as typical images and 12.5% ($n = 3$) were described as atypical. In the three cases of atypical inner ear morphology, present VEMP responses were noted for the ear of interest. One case demonstrated unilaterally absent VEMP responses, possibly due to concurrent cochlear implantation. When the presence of the VEMP response was compared to inner ear anomaly, a non-significant difference was noted between these two categories, $p = .100$.

***Adult group.*** Prior to cochlear implantation, 7.14% ($n = 1$) demonstrated absent VEMP responses bilaterally, while 92.86% ($n = 13$) presented with present VEMP responses bilaterally. Within this group, the one case with absent VEMP responses bilaterally was described as having acquired/progressive hearing loss. The cases with bilaterally present VEMP responses prior to implantation were described as having acquired/progressive hearing loss in 53.85% ($n = 7$), with diagnoses of Meniere's disease, meningitis infection, hyperbilirubinemia, and LVAS each presenting in one case. The remaining three cases demonstrating acquired/progressive hearing loss had unknown etiology of hearing loss. In the 38.46% ($n = 5$) presenting with stable hearing loss, one case was attributed to family history of hearing loss and one to maternal German measles. The remaining three cases were not diagnosed with an etiology of hearing loss. One case did not have data available for this characteristic. When the presence of the VEMP response was compared with the stability of hearing loss (stable

versus acquired/progressive), a non-significant difference was noted between these groups, $p = 1.000$.

When evaluating the presence of the VEMP response by the presence of typical inner ear imaging, 14 adult cases were available for analysis. Two cases presented with atypical CT imaging, both with present VEMP responses. A non-significant difference was noted, $p = 1.000$.

***Pediatric group.*** Prior to cochlear implantation, 19 pediatric cases were available for analysis. Within this group, 15.79% ($n = 3$) demonstrated absent VEMP responses bilaterally; all three cases with bilaterally absent VEMP responses were described as having acquired/progressive sensorineural hearing loss. The etiology of hearing loss was unknown in two cases, and attributable to congenital CMV infection in one case. The cases presenting with bilaterally present VEMP responses prior to implantation were described as having congenital hearing loss. One case was attributed to family history of hearing loss and two cases were unknown. When evaluating the presence of the VEMP response by the stability of hearing loss, a significant difference was noted, $p = .01$.

Ten pediatric cases were evaluated to compare the presence of the VEMP response to the status of inner ear morphology as described by CT imaging. Within this group, one case demonstrated atypical CT imaging; however, VEMP responses were present in the ear to be implanted in this case. When evaluating this comparison, a non-significant difference was noted, $p = 1.000$. Table 11 provides the comparison

between the VEMP response and hearing loss stability. Table 12 provides the results for the comparison between the VEMP response and inner ear anomaly.

Table 11

*Comparison of VEMP Response Pre-Implantation to Hearing Loss Stability.*

| Age Category | Present VEMP Pre-CI | Absent VEMP Pre-CI |
|---|---|---|
| Total* (*n* = 36) | | |
| Congenital | 55.56% (*n* = 20) | -- |
| Acquired/Progressive | 33.33% (*n* = 12) | 11.11% (*n* = 4) |
| Adult (*n* = 17) | | |
| Congenital | 35.29% (*n* = 6) | -- |
| Acquired/Progressive | 58.82% (*n* = 10) | 5.88% (*n* = 1) |
| Pediatric* (*n* = 19) | | |
| Congenital | 73.68% (*n* = 14) | -- |
| Acquired/Progressive | 10.53% (*n* = 2) | 15.79% (*n* = 3) |

*Note:* $*p < .05$, -- indicates no data available

Table 12

*Comparison of VEMP Response Pre-Implantation to Inner Ear Imaging.*

| Age Category | Present VEMP Pre-CI | Absent VEMP Pre-CI |
|---|---|---|
| Total (*n* = 24) | | |
| Typical | 75.0% (*n* = 18) | 12.5% (*n* = 3) |
| Atypical | 12.5% (*n* = 3) | -- |
| Adult (*n* = 14) | | |
| Typical | 78.57% (*n* = 11) | 7.14% (*n* = 1) |
| Atypical | 14.28% (*n* = 2) | -- |
| Pediatric (*n* = 10) | | |
| Typical | 70.0% (*n* = 7) | 20.0% (*n* = 2) |
| Atypical | 10.0% (*n* = 1) | -- |

*Note:* -- indicates no data available

**Question 6: Is there a significant relationship between the presence of the VEMP response and the caloric response before cochlear implantation?**

**Participants.** This question evaluated 11 pediatric and 18 adult cochlear implant cases with available VEMP and caloric data. Prior to cochlear implantation, four cases demonstrated absent VEMP responses bilaterally. One of the cases with bilaterally absent VEMP responses at the pre-implantation evaluation also presented with concurrent cochlear implantation. Eighteen cases presented with a cochlear implant

unilaterally and were under evaluation for contralateral implantation. Cases with concurrent cochlear implantation were excluded from this analysis.

**Data analysis.** All participants with pre-cochlear implantation VEMP and caloric responses were evaluated. VEMP data was categorized as present or absent, while caloric testing results were categorized as typical or atypical as described by clinical protocol. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the typical caloric response as compared to the presence of the VEMP response was calculated.

***Total group.*** After excluding cases with concurrent cochlear implants and those who were not evaluated using both VEMP and caloric testing, 17 cases were available for analysis. Two cases presented with absent VEMP responses bilaterally, with typical caloric responses. Of the remaining 15 cases with present VEMP responses bilaterally, 66.67% (n = 10) demonstrated typical caloric responses. The remaining five cases demonstrated atypical caloric responses, with asymmetrical responses ranging from 23 to 56%. Additionally, one case presented with 33% directional preponderance and one case with bilateral areflexia. When the presence of the VEMP response was compared to the caloric response, a non-significant difference was noted, $p = 1.000$.

***Adult group.*** Prior to cochlear implantation, 15 cases were available for analysis. Only one case demonstrated typical caloric responses with absent VEMP responses, leaving 14 cases available with present VEMP responses. Within this group, 64.28% ($n = 9$) demonstrated typical caloric and VEMP responses. On the other hand, 35.71% ($n = 5$) demonstrated atypical caloric responses, with atypical symmetry

ranging from 23% to absent response, and present VEMP responses. No case with an atypical caloric response presented with absent VEMP responses. When the presence of the VEMP response was compared to the caloric response, a non-significant difference was noted, $p = 1.000$.

***Pediatric group.*** Prior to cochlear implantation, two cases were available for analysis after excluding those with concurrent cochlear implant at the time of testing. While statistical analyses could not be conducted on such a small group, both cases presented with typical caloric responses, while VEMP responses were only present in one case. Table 13 provides the results from these comparisons.

Table 13

*Comparison of VEMP Response Pre-Implantation to Caloric Response.*

| Age Category | Present VEMP Pre-CI | Absent VEMP Pre-CI |
|---|---|---|
| Total (*n* = 17) | | |
| Caloric Typical | 58.82% (*n* = 10) | 11.76% (*n* = 2) |
| Caloric Atypical | 29.41% (*n* = 5) | -- |
| Adult (*n* = 15) | | |
| Caloric Typical | 60.0% (*n* = 9) | 6.67% (*n* = 1) |
| Caloric Atypical | 33.33% (*n* = 5) | -- |
| Pediatric (*n* = 2) | | |
| Caloric Typical | 50.0% (*n* = 1) | 50% (*n* = 1) |
| Caloric Atypical | -- | -- |

*Note:* -- indicates no data available

**Question 7: Is there a significant relationship between the presence of the VEMP response and the etiology of hearing loss after cochlear implantation?**

**Participants.** This question evaluated 15 participants with available post-cochlear implantation data. Five pediatric participants and 10 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17.

**Data analysis.** Analyses were conducted by evaluating the stability of hearing loss (congenital, acquired/progressive) and inner ear morphology (typical, atypical) as

described by CT imaging and were compared to the presence of the VEMP response. The VEMP response was categorized as present or absent for this comparison.

***Total group.*** The 17 cases available were described with congenital hearing loss in 52.94% ($n$ = 9) and with acquired/progressive hearing loss in 47.06% ($n$ = 8). Of these cases, 52.94% ($n$ = 9) demonstrated present VEMP responses ipsilateral to the cochlear implant, with six cases of congenital hearing loss and three cases of acquired/progressive hearing loss. The remaining 47.06% ($n$ = 8) demonstrated absent VEMP responses ipsilateral to the cochlear implant, with four cases each of congenital and acquired/progressive hearing loss. When comparing the presence of the VEMP response in cochlear implant recipients based on hearing loss stability (congenital, acquired/progressive), a non-significant difference was noted, $p$ = .637.

Thirteen cases provided VEMP results and inner ear imaging information. Only one case demonstrated atypical inner ear imaging as described by CT scan, with absent VEMP responses on the implanted side. When comparing the presence of the VEMP response in cochlear implant recipients to inner ear imaging, a non-significant difference was noted, $p$ = 1.000.

***Adult group.*** Following cochlear implantation, 12 adult cases were available for analysis. When comparing the presence of the VEMP response based on the acquisition of hearing loss (congenital, acquired/progressive), a non-significant difference was noted, $p$ = 1.000.

Nine adult cases had available CT imaging results. One case demonstrated atypical CT imaging and absent VEMP responses on the implanted side. When

comparing the presence of the VEMP response to inner ear imaging results, a non-significant difference was noted, $p = 1.000$.

***Pediatric group.*** Following implantation, five pediatric cases were available for analysis. Due to the small number of cases within this age group, no statistical analyses were completed. Within this age group, four cases presented with congenital hearing loss and one with acquired/progressive. Those with congenital hearing loss demonstrated present VEMP responses post-implantation in three cases, and with absent VEMP responses in one case. The single case of acquired hearing loss in this group demonstrated present VEMP responses post-implantation.

When comparing the presence of the VEMP response to the results of CT imaging studies, all cases presented with typical inner ear imaging. Table 14 provides the results from the comparisons between VEMP results and hearing loss stability. Table 15 provides the results from the comparisons between VEMP results and inner ear imaging studies.

Table 14

*Comparison of VEMP Response Post-Implantation to Hearing Loss Stability.*

| Age Category | Present VEMP Post-CI | Absent VEMP Post-CI |
|---|---|---|
| Total (*n* = 17) | | |
| Congenital | 29.41% (*n* = 5) | 23.53% (*n* = 4) |
| Acquired/Progressive | 23.53% (*n* = 4) | 23.53% (*n* = 4) |
| Adult (*n* = 12) | | |
| Congenital | 16.67% (*n* = 2) | 25.0% (*n* = 3) |
| Acquired/Progressive | 25.0% (*n* = 3) | 33.33% (*n* = 4) |
| Pediatric (*n* = 5) | | |
| Congenital | 60.0% (*n* = 3) | 20.0% (*n* = 1) |
| Acquired/Progressive | 20.0% (*n* = 1) | -- |

*Note:* -- indicates no data available

Table 15

*Comparison of VEMP Response Post-Implantation to Inner Ear Imaging.*

| Age Category | Present VEMP Post-CI | Absent VEMP Post-CI |
|---|---|---|
| Total (*n* = 13) | | |
| Typical | 46.15% (*n* = 6) | 46.15% (*n* = 6) |
| Atypical | -- | 7.69% (*n* = 1) |
| Adult (*n* = 9) | | |
| Typical | 33.33% (*n* = 3) | 55.56% (*n* = 5) |
| Atypical | -- | 11.11% (*n* = 1) |
| Pediatric (*n* = 4) | | |
| Typical | 75.0% (*n* = 3) | 25.0% (*n* = 1) |
| Atypical | -- | -- |

*Note:* -- indicates no data available

**Question 8: Is there a significant relationship between the presence of the VEMP response and the caloric response after cochlear implantation?**

**Participants.** This question evaluated 11 participants with available post-cochlear implantation data. Two pediatric participants and 9 adult participants were analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 13.

**Data analysis.** All participants with post-cochlear implantation VEMP and caloric responses were evaluated. VEMP data was categorized as present or absent, while

caloric testing results were categorized as typical or atypical as described by clinical protocol. Data was analyzed using SPSS (v13.0). The relative frequency of the presence of the typical caloric response as compared to the presence of the VEMP response was calculated.

***Total group.*** Thirteen cases were available for analysis. Of the seven cases demonstrating present VEMP responses following cochlear implantation, 57.14% ($n = 4$) demonstrated typical caloric responses. The remaining 42.86% ($n = 3$) demonstrated atypical caloric responses with asymmetry ranging from 30 – 97%. On the other hand, of the six cases that demonstrated absent VEMP responses post-implantation, half demonstrated typical caloric function. The remaining cases presented with atypical caloric symmetry ranging from 30 – 50%. When the presence of the VEMP response was compared to the presence of typical caloric response following implantation, a non-significant difference was noted, $p = 1.000$.

***Adult group.*** Eleven adult cases were available for analysis. Five cases demonstrated present VEMP responses. Of these five, three demonstrated typical caloric responses. The six cases with absent VEMP responses demonstrated typical caloric responses in half of these cases. When the presence of the VEMP response was compared to the caloric response following implantation, a non-significant difference was noted, $p = 1.000$.

***Pediatric group.*** Only two pediatric cases were evaluated with caloric testing at the post-implantation evaluation. While both cases presented with present VEMP responses on the side of implantation, one case presented with atypical caloric results.

Due to the small number of cases within this age group, no statistical analyses were completed. Table 16 provides the results for these comparisons.

Table 16

*Comparison of VEMP Response Post-Implantation to Caloric Response.*

| Age Category | Present VEMP Post-CI | Absent VEMP Post-CI |
|---|---|---|
| Total (*n* = 13) | | |
| Caloric Typical | 30.77% (*n* = 4) | 23.08% (*n* = 3) |
| Caloric Atypical | 23.08% (*n* = 3) | 23.08% (*n* = 3) |
| Adult (*n* = 11) | | |
| Caloric Typical | 27.27% (*n* = 3) | 27.27% (*n* = 3) |
| Caloric Atypical | 18.18% (*n* = 2) | 27.27% (*n* = 3) |
| Pediatric (*n* = 2) | | |
| Caloric Typical | 50.0% (*n* = 1) | 50% (*n* = 1) |
| Caloric Atypical | -- | -- |

*Note:* -- indicates no data available

**Question 9: Does the amount of time between cochlear implantation and post-cochlear implantation vestibular testing impact the presence of the VEMP response?**

**Participants.** This question evaluated 15 participants with available post-cochlear implantation data. Five pediatric participants and 10 adult participants were

analyzed. Two adult participants were included twice, as they were implanted bilaterally, bringing to total number of cases for analysis to 17.

**Data analysis.** Analyses were conducted by evaluating the time duration between implantation and post-implantation vestibular testing and the presence of the VEMP response. Time was divided into two categories: two months post-implantation, greater than two months post-implantation. The VEMP response was categorized as present or absent for this comparison.

***Total group.*** Of these 17 cases, 52.94% (*n* = 9) demonstrated present VEMP responses, with 47.06% (*n* = 8) were absent ipsilateral to the cochlear implant. Of those who received vestibular testing at two-months post-implantation, 44.44% (*n* = 4) demonstrated present VEMP responses, while 55.56% (*n* = 5) were absent. Cases evaluated later than two months post-implantation (*n* = 8) demonstrated present VEMP responses in 62.5% (*n* = 5) and absent responses in 37.5% (*n* = 3). When comparing the presence of the VEMP response in cochlear implant recipients to the time that post-implantation vestibular testing was completed, a non-significant difference was noted, *p* = .637.

***Adult group.*** Following cochlear implantation, 12 adult cases were available for analysis, with 58.33% (*n* = 7) evaluated at two month post-implantation. The time duration between post-implantation for those who did not receive vestibular testing at the two month appointment (*n* = 5) ranged between six and 21 months. Of those receiving vestibular evaluation at two months post-implantation, 42.86% (*n* = 3) demonstrated present VEMP responses, with 57.14% (*n* = 4) were absent. Of those

evaluated later than two months, two demonstrated present VEMP responses and three were absent. When comparing the presence of the VEMP response based on the time duration between implantation and follow up testing, a non-significant difference was noted, $p = 1.000$.

***Pediatric group.*** Five pediatric cases were available for this analysis. Two were evaluated at two months post-implantation, while the remaining three were evaluated later, ranging between four and 27 months post-implantation. Of the two evaluated at two months post-implantation, one demonstrated present VEMP responses while one was absent. Those evaluated after two months post-implantation ($n = 3$) all demonstrated present VEMP responses. Due to the small number of cases within this age group, no statistical analyses were completed. Table 17 provides the results from these comparisons.

Table 17

*Comparison of VEMP Response Post-Implantation to Time of Post-Evaluation.*

| Age Category | Present VEMP Post-CI | Absent VEMP Post-CI |
|---|---|---|
| Total (*n* = 17) | | |
| 2 months (*n* = 9) | 23.53% (*n* = 4) | 29.41% (*n* = 5) |
| >2 months (*n* = 8) | 29.41% (*n* = 5) | 17.65% (*n* = 3) |
| Adult (*n* = 12) | | |
| 2 months (*n* = 7) | 25.0% (*n* = 3) | 33.33% (*n* = 4) |
| >2 months (*n* = 3) | 16.67% (*n* = 2) | 25.0%(*n* = 3) |

*Note:* -- indicates no data available

# CHAPTER 5

# DISCUSSION

Cochlear implantation has become an integral option for both children and adults with severe to profound sensorineural hearing loss, providing access to the auditory environment. While improvement in access to auditory information is required for appropriate spoken speech understanding in adults and for appropriate speech and language development in children, the effects of cochlear implantation on the additional organs of the inner ear have not been fully evaluated. Significantly, research has demonstrated that cochlear implantation may lead to anatomical changes to additional inner ear structures, particularly the saccule (Tien & Linthicum, 2002), and may correlate with trauma found along the basal turn of the cochlea (Gstoetter et al., 1997).

Cochlear implantation has been associated with subjective and objective vestibular dysfunction; however, the site of lesion varies between studies. Filipo and colleagues (2006) reported that the variability noted between within studies of vestibular dysfunction must be understood with regards to the variability of the study sample evaluated and the test protocol used to evaluate the sample. For example, the study provided by Filipo and colleagues demonstrated improvement in the caloric response over the three-month observation period. Based on these results, caloric function improved to pre-surgical response levels in many cases after reduction to absent function over the first few weeks following implantation.

While the caloric response has been evaluated in numerous studies, inconclusive results following implantation have been reported (e.g. Brey et al., 1995; Huygen et al.,

1995). The addition of the VEMP response test to the vestibular test protocol may provide additional information that could determine how cochlear implantation affects the saccule, leading to better understanding of the vestibular dysfunction experienced by many cochlear implant recipients. Findings from previous studies have indicated that change in saccule response ranges from 30 to 40% following implantation (Jacot et al., 2009; King et al., 2009; Melvin et al., 2008).

**Major Findings of Study**

This study found that recipients of cochlear implantation demonstrate significantly different proportions of the presence of the VEMP response following implantation, but not before implantation, when compared to individuals with normal hearing and vestibular function. Before cochlear implantation, 40 cases were analyzed in order to compare the VEMP response in implantation candidates to those with normal hearing and vestibular function. The 12.5% of absent VEMP responses prior to implantation was found to be non-significant; however, it was expected that individuals with severe to profound sensorineural hearing loss would demonstrate a lower proportion of VEMP responses when compared to those with typical auditory and vestibular function. Based on the literature, atypical VEMP responses have been noted in 30 to 45% of children and adult candidates for cochlear implantation (Jacot et al., 2009; King et al., 2009; Melvin et al., 2008; Moushey et al., 2010). While this proportion is noticeably lower than reported in the literature, note that the literature typically includes both absent and atypical VEMP responses in the analyses. Because this study only evaluated the actual

absence of the VEMP response, it was expected that the change rate found within this study was lower than previously reported.

Seventeen total cases were available for comparison following cochlear implantation and were compared to the same individuals with normal hearing and vestibular function. Significantly, nearly half (n = 8) of the cochlear implant recipients demonstrated absent VEMP responses at the post-implantation evaluation. When compared to the group with normal hearing and vestibular function, they demonstrated significantly fewer VEMP responses. Note that previous literature has established the presence of the VEMP response in nearly all participants with typical vestibular function (e.g. Cody & Bickford, 1969; Colebatch et al., 1994; Isaradisaikul et al., 2008; Robertson & Ireland, 1995).

Specifically, this study found that cochlear implantation leads to significant change in the proportion of present VEMP responses in cases less than 60 years of age. Within this study, 17 cases (5 pediatric, 12 adult) were analyzed in order to determine the change of the VEMP response for the implanted side. Following implantation, 47% (*n* = 8) demonstrated absent VEMP responses on the side of implantation, a significant change in the proportion of present VEMP responses within this group. When evaluating adult cases (*n* = 12), 58% (*n* = 7) demonstrated absent responses.

Previous literature has also reported a significant change following cochlear implantation. Melvin et al (2008) reported a change of 31% (*n* = 5) from typical responses to either absent or atypical VEMP responses. Similarly, King (2009) reported

on 17 cases, finding a change of approximately 40% from typical responses to either absent or atypical VEMP responses at six weeks post-implantation. While the results of this study were slightly higher than those previously reported in the literature, they do provide additional support for the change that occurs to the VEMP response that occurs within the adult population. Unfortunately, a sufficient number of pediatric cases were not available to describe this group statistically, but based on available adult data, it is reasonable to assume that pediatric cases may also be at substantial risk for vestibular dysfunction due to implantation. Within the pediatric category in this study, 15 of the 22 participants presented with concurrent cochlear implantation. While this ear was not evaluated for this study as the vestibular function prior to implantation was unknown, nine of these 15 (60%) demonstrated absent VEMP responses ipsilateral to the initial implant. Of these, only one demonstrated bilaterally absent VEMP responses. Because pre-implantation status of these ears cannot be definitively known, it does suggest that this change in the presence of the VEMP response is also evident within the pediatric population as well.

Interestingly, the only variable found to influence the presence of the VEMP response prior to implantation was the stability of hearing loss (congenital, acquired/progressive). Absent VEMP responses were noted to have acquired/progressive hearing loss in this group, however, more specific comments regarding the relationship of etiology to the presence of the VEMP response cannot be determined at this time. For the total comparison group, 16 cases of acquired/progressive hearing loss were noted, presenting with present VEMP

responses in 12 cases. While only four cases of absent VEMP responses were noted prior to implantation, all four cases were described with acquired/progressive hearing loss. Unfortunately, only one case reported a known etiology of acquired/progressive hearing loss (congenital CMV infection). Due to the small number of cases and the little information available regarding the actual etiology of hearing loss, it is unclear how this variable contributes to predicting the presence of the VEMP response in individuals with severe to profound sensorineural hearing loss. The literature has suggested that individuals with acquired/progressive forms of hearing loss may be at increased risk for vestibular dysfunction as well due to concurrent trauma to the inner ear structures (Arnvig, 1955; Goldstein et al., 1958; Huygen et al., 1993).

While considering the effect of hearing loss stability on the VEMP response, analyses evaluating the effect of this characteristic following implantation was non-significant. Overall, 40% ($n = 7$) of cases with acquired/progressive sensorineural hearing loss demonstrated present VEMP responses following implantation. While diagnoses were provided for over half of these cases (Meniere's disease, meningitis infection, hyperbilirubinemia, LVAS), the remaining three cases were not diagnosed with an etiology. The small number of cases leads to difficulty in making conclusions regarding the stability of hearing loss and the presence of the VEMP response. However, of those with known etiology of acquired/progressive sensorineural hearing loss, many etiologies have been associated with changes in VEMP presentation. For example, Meniere's disease was noted in one adult case. The literature has reported that the VEMP response may be absent in up to half of cases with this diagnosis (de

Waele, Ba Huy, Diard, Freyss, & Vidal, 1999) and may vary based on the progression of the disease process (Young, Huang, & Cheng, 2003). Meningitis was provided as the cause of sensorineural hearing loss in one case that demonstrated present VEMP responses. The literature has indicated that the VEMP response may be present in the majority of children diagnosed with meningitis (Cushing, Papsin, Rutka, James, Blaser, & Gordon, 2009); however, variability due to the type and duration of meningitis infection may lead to various outcomes.

**Non-significant variables.** Interestingly, additional variables were found to be non-significant for these analyses. Caloric function, inner ear imaging, and hearing loss stability (post-implantation) were not found to influence the presence of the VEMP response within this study.

***Caloric function.*** While the horizontal semicircular canal has been the most widely evaluated vestibular end organ, variability in its function has been demonstrated in those with concurrent sensorineural hearing loss. This study found that approximately one-third of cases with severe to profound sensorineural hearing loss demonstrated atypical horizontal semicircular canal function as measured by caloric testing. Bilateral areflexia was noted in only one case (6.67%). Results were consistent with previous literature regarding reduction in function, but lower than previously reported literature regarding absent function which has been shown in up to 30% of cases (e.g. Buchman et al., 2002).

Furthermore, changes in horizontal semicircular canal function also have been demonstrated within this population. Again, the variability in presentation has not been

consistent, leading to less than conclusive descriptions regarding the effect of cochlear implantation on the horizontal semicircular canal. This study found that 46% of cases demonstrated atypical caloric function following implantation, consistent with reported literature, which ranges from 29 to 32% (Buchman et al., 2002; Krause et al., 2009). Because evaluations were not conducted at the same evaluation period, it may be that the variability noted was due to various time courses for compensation (Filipo et al., 2006).

***Caloric and VEMP response comparison.*** Caloric and VEMP responses have often been compared to demonstrate a broader functionality of the vestibular system. This study found that 33% of adult cases demonstrated atypical caloric function with present VEMP responses. In the two adult cases that demonstrated absent VEMP responses bilaterally, both demonstrated typical caloric responses; no adult case was noted with atypical responses to both caloric and VEMP responses. Prior research has demonstrated that the auditory, caloric, and VEMP pathways are different, describing the functionality of separate end organs and neural pathways. For example, in Wu and Young's (2002) evaluation of sudden profound unilateral sensorineural hearing loss, no correlations were found between the presence of profound hearing loss and the presence of the VEMP or caloric response. In children, atypical caloric responses have been seen to accompany atypical VEMP responses; however, these results have also been described as relatively independent of each other. Additionally, the VEMP response has been less likely to present as atypical than the caloric response (Tribukait et al., 2004).

***Inner ear anomalies.*** Within this series of cases, two inner ear anomalies were noted on CT imaging studies: superior semicircular canal dehiscence and large vestibular aqueduct syndrome. Superior semicircular canal dehiscence has not been typically described as correlating with severe to profound sensorineural hearing loss. One study has evaluated the incidence of this disorder in children with severe to profound sensorineural hearing loss, finding that 13.7% ($n$ = 18 of 131) presented with this condition, but with no other clinical characteristics. Based on this study, it is unlikely that this case's VEMP responses were significantly affected by the noted inner ear anomaly. Additionally, as this study only evaluated the presence of the response, it may be that subtle indications of superior semicircular canal dehiscence (e.g. increased p1-n1 inter-peak amplitude, decreased threshold) may be noted.

On the other hand, LVAS has been described with varying vestibular presentations. Two cases of LVAS were present within this dataset, one child and one adult. The pediatric case demonstrated unilaterally absent VEMP responses for the right side, possible due to concurrent cochlear implantation on that side. Vestibular complaints were not noted for this case. The adult case demonstrated bilaterally present VEMP responses. Also note that this case was experiencing active progressive of hearing and vestibular loss at the evaluation prior to implantation. Following surgery, this case demonstrated absent VEMP responses on the side of implantation. This case reported slight vertigo following implantation lasting for a few days. At the time of follow up vestibular testing, this case reported no active vestibular symptoms. Although LVAS has been described as the most common inner ear anomaly found in sensorineural

hearing loss, vestibular complaints and results of vestibular testing have been variable. Abnormal vestibular evaluation results, including caloric responses, may occur in all individuals with LVAS diagnosis, even in those without vestibular symptoms (Emmett, 1985). Few studies have evaluated the vestibular responses in those with LVAS, and little information is available regarding the VEMP response in these individuals. Sheykholeslami, Schmerber, Kermany, and Kaga (2004) provided a series of three case studies diagnosed with LVAS. The authors reported that the VEMP responses were abnormally asymmetric for threshold and amplitude in all cases. More specifically, the authors reported that two of the three cases demonstrated atypically low thresholds and larger p1-n1 inter-peak amplitudes than expected, leading the authors to hypothesize that LVAS leads to a hypersensitivity to sound. Unfortunately, the small sample size available for VEMP responses in cases diagnosed with LVAS has not allowed for conclusive evaluation.

***Duration to post-implantation testing.*** Previous research evaluating the vestibular function in individuals following cochlear implantation has suggested that the time between implantation and post-implantation vestibular testing significantly impacts the outcomes of these studies. For example, King (2009) presented one case that demonstrated an absent VEMP response at six weeks post-implantation that proved to have a present response at six months. Although this is one case, it suggests that temporary alterations within the inner ear may impact the results of VEMP testing. This study evaluated the presence of the VEMP response at two months and at greater than two months following implantation. Unfortunately, the number of available cases did not

allow for more concise evaluation of the time duration following implantation on the results of VEMP testing.

**Explanation of Results**

The change in the VEMP response following cochlear implantation is an effect that has been suggested in the literature. While the structures within the inner ear have been shown to receive significant damage during the implantation process (Gstoetter et al., 1997; Tien & Linthicum, 2002), the actual observable effects of this change in response have not been sufficiently demonstrated in order to suggest change in the implantation protocol or surgical technique. This study provides further support for the observed change in response, as these cases demonstrated a change in the presence of the VEMP response from 100% ($n$ = 17) prior to implantation to 53% ($n$ = 9) following surgery. This study provides further support for previous studies, which found atypical VEMP responses in 30 to 40% of cases following implantation (King, 2009; Melvin et al., 2008). Because of the increased number of individuals diagnosed with severe to profound sensorineural hearing loss that are under evaluation for cochlear implantation, the number of individuals who may be at risk for vestibular dysfunction will also increase. The internal cochlear implant does not vary based on age; individuals of all ages may be at risk for subsequent vestibular dysfunction.

**Alternative Explanations**

The VEMP response relies on various factors that may have lead to absent VEMP responses within these cases. The VEMP response cannot be elicited in the presence of an atypical middle ear system or with insufficient SCM muscle contraction.

**Effect of cochlear implant on middle ear conductance.** The VEMP response has been shown to be highly dependent on typical middle ear conductance, with previous research finding that a conductive component of less than 10 dB may eliminate the VEMP response when using air-conducted stimuli (Bath, Harris, McEwan, & Yardley, 1999). The addition of the cochlear implant device within the middle ear space may lead to a dampening of the intensity of the VEMP stimulus. If the addition of the cochlear implant leads to dampening of the acoustic signal, it may be that the absent VEMP responses may be due to insufficient intensity levels. It seems unlikely that these results are due to changes in middle ear conductance, however, as tympanic testing was required to be within the typical range in order to complete the vestibular test battery, but it must be noted that a small change in the conductive properties of the middle ear space may have contributed to the changes noted in the presence of the VEMP response.

**Tonic muscle monitoring.** The monitoring of muscle tonicity has been described as important for evaluating the p1-n1 inter-peak amplitude of the VEMP response, which in turn may influence the presence of the VEMP response. Studies have demonstrated the reliability of the VEMP response without monitoring to ensure that the SCM muscle was sufficiently contracted (Ackley et al., 2003; Isaradisaikul et al., 2008); however, interpretation of the VEMP response may be more difficult. The EMG level of SCM muscle contraction has been demonstrated as vital to reducing the variability of p1-n1 inter-peak amplitude (Akin et al., 2004). Although EMG level has been demonstrated as important for obtaining reliable results for specific parameters,

monitoring this level has not produced differences clinically when evaluating the presence of this response alone (Isaradisaikul et al., 2008). For this study, however, monitoring was not deemed necessary as only the presence of the response was evaluated. Future research could evaluate the effect of cochlear implantation on specific parameters, such as p1-n1 inter-peak amplitude, which may require monitoring to ensure accurate responses.

**Limitations of Study**

The retrospective nature of this study provides a significant limitation. Although a clinical protocol was in place in order to evaluate these cases, deviation from this protocol often occurred and many possible cases were not evaluated, particularly at the post-implantation vestibular evaluation. At this point, a small number of cases were available for analyses, providing a limitation to this study. In many of the pediatric comparisons, not enough cases were available to provide statistical analyses, particularly following implantation.

The results of this study were expected based on clinical experience, as well as reports of anatomical changes found in previous literature (Tien & Linthicum, 2002); however, the lack of significance of additional variables was unexpected. Unfortunately, the high percentage of unknown information limited the available data points for many of these analyses. Although no additional variables were found to impact the presence of the VEMP response, significant variables may not have been evaluated. Discovering the etiology of sensorineural hearing loss in individuals with severe to profound sensorineural hearing loss may provide significant understanding into the risk factors for

vestibular dysfunction both before and after cochlear implantation, and unfortunately, the majority of cases may be attributed to hearing loss related to genetic factors that were not diagnosed.

Additionally, the test procedures themselves were possibly a limitation to this study. It is known that vestibular function may be present even in the presence of nearly complete sensorineural hearing loss. In theory, the cochlea requires significant interplay between receptor cells to provide appropriate auditory function. The vestibular system differs in that a smaller range of responses is required in order to respond appropriately to typical sensory input. Therefore, a significant number of vestibular cells could be lost before the end organ loses its ability to respond to stimuli within the typical range (Tribukait et al., 2004). It is possible that the available test methods do not provide an accurate representation of hair cell loss within the vestibular end organs.

**Suggestions for Further Research**

This study could be further enhanced by further research into this population as there are numerous facets remaining to be explored. By examining additional research questions, such as the etiology of hearing loss, the surgical technique, and the effect of cochlear implantation in pediatric cases, a better understanding of the effects of cochlear implantation on the vestibular system may be established.

**Etiology of hearing loss.** Information regarding the etiology of sensorineural hearing loss would provide variables that would possibly lead to better understanding of the increased delicacy of the vestibular system under specific conditions. The majority of cases presented with unknown etiology; however, the majority of these individuals

were not evaluated for genetic causes of hearing loss. While knowledge of the etiology of hearing loss may not lead to changes in clinical procedures, it would provide greater understanding of the clinical presentation of various genotypes. For instance, participants with *GJB2*-related hearing loss may be at increased risk for vestibular dysfunction following implantation due to concurrent pathology within the saccule (Qu et al., 2007). Recent data has demonstrated that while the VEMP response may be present at rates consistent with those with normal hearing at 500 Hz, a lower frequency (250 Hz) stimulus may demonstrate variability between these groups (Marin, 2010).

**Surgical technique.** Another interesting prospective study would include the actual effect of various surgical techniques on the presence of the VEMP response. This would require active participation from cochlear implant manufacturers and surgeons in order to develop a technique that would aid in preserving vestibular structures. At this point, many surgeons are exploring the use of hearing preservation surgical techniques in order to reduce damage to cochlear structures (Cohen, 1997). Maintaining residual hearing following implantation may indicate a reduction in damage to the structures of the inner ear, which could also indicate less damage to the vestibular structures. Tien and Linthicum (2002) reported that 75% of the temporal bones evaluated with saccular damage also demonstrated damage to the basal turn of the cochlea. Based on this, reduction in trauma to the cochlea may not only lead to improved residual hearing, but also to maintained function of the saccule. Perhaps this surgical technique would also lead to less change in the VEMP response. While it has been suggested that electrode placement may influence reports of subjective vestibular

dysfunction (Enticott et al., 2006), this difference should be further investigated to evaluate any possible changes to the surgical technique that could increase appropriate placement without compromising auditory outcomes.

**Pediatric considerations.** With children now currently under evaluation for cochlear implantation as young as six months of age at some facilities (Jacot et al., 2009), evaluation of the vestibular system prior to implantation may significantly reduce the effects of changes in vestibular dysfunction. Within this young population, limited clinical testing can be conducted. The VEMP response can be evaluated, providing information regarding the saccule and inferior branch of CN VIII; however, the vestibulo-ocular reflex cannot be evaluated due to poor tolerance of the test procedure. Within any age group, the head thrust test can evaluate the vestibulo-ocular reflex by requiring the participant to maintain fixed vision on a target (i.e. the nose of the examiner). The presence of vestibular loss requires a corrective saccade to maintain the gaze appropriately (Schubert & Shepard, 2008).

While children demonstrate a remarkable ability to compensate for inappropriate vestibular function, atypical congenital or early onset vestibular dysfunction may lead to significant delays in gross motor developmental milestones as the vestibular systems of children do not take on adult postural strategies until approximately six years of age (Jacot et al., 2009; Shumway-Cook & Woollacott, 1985). Additional testing of considerably younger children, as well as infants, may lead to additional knowledge about the effects of this surgery on the vestibular system, as well as the effects of unilateral or bilateral vestibular function changes within this population.

It is within this youngest population that the most significant findings related to the effects of cochlear implantation of the vestibular system may come into view, as these children may be more affected by damage within the vestibular system than children and adults who have already established developmental milestones that require appropriate vestibular function. Research has suggested that especially young children (below 12 months of age), may be more at risk for vestibular dysfunction following the loss of otolith function because of the increased reliance on these structures during postural development (Shall, 2009).

In studies evaluating vestibular dysfunction in cochlear implantation candidates and recipients, the risk of complete vestibular areflexia in children has been demonstrated as approximately 10%. This is misleading, however, as the implantation surgery may only affect one specific vestibular end organ and not the entire vestibular system. Furthermore, the decrease in vestibular function is unpredictable, with no significant predictors noted in the literature (Jacot et al., 2009).

**Impact of vestibular dysfunction following bilateral cochlear implantation.** The impact of sequential or simultaneous bilateral cochlear implantation on the vestibular system should also be evaluated in order to determine developmental effects on the gross motor development of young children, as well as to determine the risk of vestibular loss in the elderly population. Both of these populations provide an interesting discussion regarding acute bilateral vestibular dysfunction. Adults with bilateral vestibular dysfunction may demonstrate postural instability, particularly in situations with reduced visual or proprioceptual information, but rarely do adults

demonstrate loss of postural control (Shinjo et al., 2007). Based on this study, individuals are at separate risk for dysfunction for each side that is implanted. For instance, the participant in this series who was simultaneously implanted demonstrated unilateral VEMP responses following implantation. The impact of unilateral or bilateral reduction of the VEMP response on the balance system in either children or adults has not been fully described, but could pose a safety concern in situations with reduced visual or proprioceptual input.

The development of the vestibular system in children undergoes significant maturation and integration throughout the first years of life, allowing children to obtain more adult-like postural strategies by six years of age (Shumway-Cook & Woollacott, 1985). Interestingly, Shall (2009) evaluated the correlation between the VEMP response and motor development. While this study presented with significant limitations regarding the information provided by the VEMP results, as previously described in the review of the literature, the comparison attempted is worthy and highly important. Future studies require not only objective clinical measures, but also actual behavioral measures to provide important information on changes in vestibular function. In this group of implanted children, especially those with early implantation or those bilaterally implanted, little is understood regarding the actual effect of implantation on gross motor development. It may prove to be that children are able to compensate appropriately, and specific characteristics may put the child more at risk for motor delay. For example, Horn, Pisoni, Sanders, and Miyamoto (2005) evaluated a group of children with cochlear implants, and found that those with acquired hearing loss demonstrated lower motor

scores than children with congenital hearing loss. Based on this research and the VEMP results found in this study, stability of hearing loss may prove to be a significant predictor for motor developmental outcomes in children with early implantation.

**Conclusion**

Cochlear implantation is currently a commonly used method for providing auditory input to individuals with severe to profound sensorineural hearing loss. While this has been highly beneficial to providing auditory information, the results of this study have demonstrated that the VEMP response is likely to change following implantation. In these cases, 53% ($n = 9$) demonstrated a change from present to absent VEMP responses following cochlear implantation. Additional variables, including caloric function, surgeon, and inner ear morphology were not found to influence the VEMP response prior to or following cochlear implantation. While this study evaluated only a small number of cases ($N = 40$ total, 17 post-implantation), the results were consistent with previously reported cases of the effect of cochlear implantation on the VEMP response, further adding to this small literature base describing the effect of cochlear implantation on the VEMP response. Furthermore, the results of this study are of clinical importance as more individuals are evaluated for cochlear implantation, including those with less severe degrees of sensorineural hearing loss, as well as in infants and young children who may not have developed appropriate gross motor skills prior to implantation. The results of vestibular testing before cochlear implantation may also provide additional information regarding the status of the inner ear, possibly aiding in determining the appropriate ear to implant. At this point, vestibular testing appears to

be a reasonable addition to the cochlear implantation test battery and can assist the clinician in providing appropriate counseling of candidates and their families regarding the possible effects of cochlear implantation on the vestibular system.

## References

Ackley, R.S., Tamaki, C. Oliszewski, C., & Inverso, D. (2004). Vestibular evoked myogenic potentials in deaf and hard of hearing subjects. *Insights in Practice: Clinical Topics in Otoneurology: GN Otometrics.*

Akin, F.W., Murnane, O.D., Panus, P.C., Caruthers, S.C., Wilkinson, A.E., & Proffitt, T.M. (2004). The influence of voluntary tonic EMG level on the vestibular evoked myogenic potential. *Journal of Rehabilitation Research & Development, 41,* 473-480.

Al-Abdulhadi, K., Zeitouni, A.G., Al-Sebeih, K., & Katsarkas, A. (2002). Evaluation of vestibular evoked myogenic potentials. *Journal of Otolaryngology, 31*(2), 93-96.

Arnvig, J. (1955). Vestibular function in deafness and severe hardness of hearing. *Acta Oto-Laryngologica, 45*(4), 283-288.

Art, J.J., Crawford, A.C., Fettiplace, R., & Fuchs, P.A. (1985). Efferent modulation of hair cell tuning in the cochlea of the turtle. *Journal of Physiology, 360,* 397-421.

Art, J.J., & Fettiplace, R. (1984). Efferent desensitization of auditory nerve fibre responses in the cochlea of the turtle *Pseudemys scripta elegans. Journal of Physiology, 356,* 507-523.

Art, J.J., Wu, Y.C., & Fettiplace, R. (1995). The calcium-activated potassium channels of turtle hair cells. *Journal of General Physiology, 105,* 49-72.

Balatsouras, D.G., Kaberos, A., Assimakopoulos, D., Katomichelakis, M., Economou, N.C., & Korres, S.G. (2007). Etiology of vertigo in children. *International Journal of Pediatric Otorhinolaryngology, 71,* 487-494.

Baloh, R.W., & Honrubia, V. (1990). *Clinical neurophysiology of the vestibular system.* Oxford, UK: Oxford University Press.

Barmack, N.H., Baughman, R.W., Errico, P., & Shojaku, H. (1993). Vestibular primary afferent projections to the cerebellum of the rabbit. *Journal of Comparative Neurology, 327*(4), 521-534.

Basta, D., Todt, I., & Ernst, A. (2005). Normative values for P1/N1-latencies of vestibular evoked myogenic potentials induced by air- or bone-conducted tone bursts. *Clinical Neurophysiology, 116*(9), 2216-2219.

Basta, D., Todt, I., Goepel, F., & Ernst, A. (2008). Loss of saccular function after cochlear implantation: The diagnostic impact of intracochlear electrically elicited vestibular evoked myogenic potentials. *Audiology & Neurotology, 13*(3), 187-192.

Bath, A.P., Harris, N., McEwan, J., & Yardley, M.P. (1999). Effect of conductive hearing loss on the vestibulo-collic reflex. *Clinical Otolaryngology & Allied Sciences, 24*(3), 181-183.

Bath, A.P., Harris, N., & Yardley, M.P. (1998). The vestibulo-collic reflex. *Clinical Otolaryngology, 23,* 462-466.

Bergström, B. (1973). Morphology of the vestibular nerve. II. The number of myelinated vestibular nerve fibers in man at various ages. *Acta Oto-Laryngologica, 76,* 173-179.

Bickford, R.G., Jacobson, J.L., Cody, D., & Thane, R. (1964). Nature of averaged evoked potentials to sound and other stimuli in man. *Annals of the New York Academy of Sciences, 112*(1), 204-218.

Bonucci, A.S., Filho, O.A.C., Mariotto, L.D.F., Amantini, R.C.B., & Alvarenga, K.F. (2008). Vestibular function in cochlear implant users. *Revista Brasileria de Otorrinolaringologia, 74*(2), 273-278.

Brantberg, K., & Fransson, P.-A. (2001). Symmetry measures of vestibular evoked myogenic potentials using objective detection criteria. *Scandinavian Audiology, 30,* 189-196.

Breuer, J. (1874). Über die funktion der Bogengänges des Ohrlabrinthes. *Wiener medizinisches Jahrbuch, 4,* 72-124.

Brey, R.H., Facer, G.H., Trine, M.B., Lynn, S.G., Peterson, A.M., & Suman, V. (1994). Vestibular changes associated with implantation of a multiple channel cochlear prosthesis. *Otology & Neurotology, 16*(4), 424-430.

Brichta, A.M., & Peterson, E.H. (1994). Functional architecture of vestibular primary afferents from the posterior semicircular canal of a turtle *Pseudemys (Trachemys) scripta elegans. Journal of Comparative Neurology, 344*(4), 481-507.

Brodal, A., & Brodal, P. (1985). Observations on the secondary vestibulocerebellar projections in the macaque monkey. *Experimental Brain Research, 58*(1), 62-74.

Buchman, C.A., Joy, J., Hodges, A., Telischi, F.F., & Balkany, T.J. (2004). Vestibular effects of cochlear implantation. *Laryngoscope, 114*(Suppl 103), 1-22.

Büttner-Ennever, J.A. (1992). Patterns of connectivity in the vestibular nuclei. *Annals of the New York Academy of Sciences, 656,* 363-378.

Camis, M., & Creed, R.S. (1930). *The physiology of the vestibular apparatus.* Oxford, UK: Clarendon Press.

Carey, J.P., Hirvonen, T.P., Huller, T.E., & Minor, L.B. (2004). Acoustic responses of vestibular afferents in a model of superior canal dehiscence. *Journal of Otology & Neurotology, 25,* 345-352.

Carleton, S.C., & Carpenter, M.B. (1983). Afferent and efferent connections of the medial inferior and lateral vestibular nuclei in the cat and monkey. *Brain Research, 278,* 29-51.

Cazals, Y., Aran, J.M., & Erre, J.P. (1983). Intensity difference thresholds assessed with eighth nerve and auditory cortex potentials: Compared values from cochlear and vascular responses. *Hearing Research, 10,* 263-268.

Chang, C., & Young, Y. (2007). Caloric and vestibular evoked myogenic potential tests in evaluating children with benign paroxysmal vertigo. *International Journal of Pediatric Otorhinolaryngology, 71*(3), 495-499.

Chen, C.-N., Wang, S.-J., Wang, C.-T., Hsieh, W.-S., & Young, Y.-H. (2007). Vestibular evoked myogenic potentials in newborns. *Audiology & Neurotology, 12,* 59-63.

Chen, C.W., Young, Y.H., & Wu, C.H. (2000). Vesitbular neuritis: Three-dimensional videonystagmography and vestibular evoked potential results. *Acta Oto-Laryngologica, 120,* 845-848.

Chen, E.Y., Paladin, A., Phillips, G., Raske, M., Vega, L., Peterson, D., & Sie, K.C.Y. (2009). Semicircular canal dehiscence in the pediatric population. *International Journal of Pediatric Otorhinolaryngology, 73,* 321-327.

Cheng, P.W., & Murofushi, T. (2001a). The effects of plateau time of vestibular-evoked myogenic potentials triggered by tone bursts. *Acta Oto-Laryngologica, 121,* 935-938.

Cheng, P.W., & Murofushi, T. (2001b). The effect of rise/fall time on vestibular-evoked myogenic potentials triggered by short tone bursts. *Acta Oto-Laryngologica, 121,* 696-699.

Clarke, A.H., Schonfeld, U., & Helling, K. (2003). Unilateral examination of utricle and saccular function. *Journal of Vestibular Research, 13,* 215-225.

Cody, D.T.R., & Bickford, R.G. (1969). Averaged evoked myogenic responses in normal man. *Laryngoscope, 79,* 400-446.

Cohen, N.L. (1997). Cochlear implant soft surgery: Fact or fantasy. *Otolaryngology-Head & Neck Surgery, 117*(3), 214-216.

Colebatch, J.G., Halmagyi, G.M., & Skuse, N.F. (1994). Myogenic potentials generated by a click-evoked vestibulocollic reflex. *Journal of Neurology, Neurosurgery & Psychiatry, 57,* 190-197.

Cremers, C.W.R.J., Admiraal, R.J.C., Huygen, P.L.M., Bolder, C., Everett, L.A., Joosten, F.B.M., Green, E.D., …Otten, B.J. (1998). Progressive hearing loss, hypoplasia of the cochlea and widened vestibular aqueducts are common features in Pendred's syndrome. *International Journal of Pediatric Otorhinolaryngology, 45*(2), 113-123.

Curthoys, I.S., Kim, J., McPhedran, S.K., & Camp, A.J. (2006). Bone conduction vibration selectively activates irregular primary otolithic vestibular neurons in the guinea pig. *Experimental Brain Research, 175*(2), 256-267.

Cushing, S.L., Papsin, B.C., Rutka, J.A., James, A.L., Blaser, S.L., & Gordon, K.A. (2009). Vestibular end-organ and balance deficits after meningitis and cochlear implantation in children correlate poorly with functional outcome. *Otology & Neurotology, 30*(4), 488-495.

D'Agostino, R., Melagrana, A., Ravera, B., & Taborelli, G. (1999). Compared study of optokinetic and caloric nystagmus in children with unilateral hyporeflexia and other vestibular disorders. *International Journal of Pediatric Otorhinolaryngology, 50,* 163-167.

de Vries, H.I., & Bleeker, J.D.J.W. (1949). The microphonic activity of the labyrinth of the pigeon: Part II: The response of the cristae in the semicircular canals. *Acta Oto-Laryngologica, 37*(4), 298-306.

de Waele, C., Ba Huy, P.T., Diard, J.-P., Freyss, G., & Vidal, P.-P. (1999). Saccular dysfunction in Meniere's disease. *American Journal of Otology, 20*(2), 233-237.

Della Santina, C.C., Potyagaylos, V., Miliaccio, A.A., Minor, L.B., & Carey, J.P. (2005). Orientation of human semicircular canals measured by three-dimensional multiplanar CT resolution. *Journal of the Association for Research in Otolaryngology, 6*(3), 191-206.

Desmond, A.L. (2004). *Vestibular evaluation: Evaluation and treatment.* New York, NY: Thieme.

Enticott, J.C., Tari, S., Koh, S.M., Dowell, R.C., & O'Leary, S.J. (2006). Cochlear implant and vestibular function. *Otology & Neurotology, 27,* 824-830.

Eviatar, L., & Eviatar, A. (1979). The normal nystagmic response of infants to caloric and perrotary stimulation. *Laryngoscope, 89,* 1036-1044.

Eviatar, L., & Eviatar, A. (1978). Neurovestibular examinations of infants and children. *Advances in Oto-Rhino-Laryngology, 23,* 169-191.

Ferber-Viart, C., Dubreuil, C., & Duclaux, C. (1997). Myogenic vestibular evoked potentials in normal subjects: Comparison between responses obtained on sternomastoid and trapezius muscles. *Acta Oto-Laryngologica (Stockholm), 117,* 472-481.

Fernández, C., & Goldberg, J.M. (1976). Physiology of peripheral neurons innervating otolith organs of the squirrel monkey. III. Response dynamics. *Journal of Neurophysiology, 39,* 996-1008.

Fife, T.D., Tusa, R.J., Furman, J.M., Zee, S.D., Frohman, E., Baloh, R.W., …Eviatar, L. (2000). Assessment: Vestibular testing techniques in adults and children: Report of the theraputics and technology subcommittee of the American Academy of Neurology. *Neurology, 55,* 1431-1444.

Filipo, R., Patrizi, M., La Gamma, R., D'Elia, C., La Rosa, G., & Barbara, M. (2006). Vestibular impairment and cochlear implantation. *Acta Oto-Laryngologica, 126,* 1266-1274.

Fontana, S., & Porth, C. (2005). Disorders of hearing and vestibular function. In C. Porth (Ed.), *Pathophysiology: Concepts of altered health states.* Philadelphia, PA: Lippincott, Williams & Wilkins.

Fuchs, P.A., & Evans, M.G. (1988). Voltage oscillations and ionic conductances in hair cells isolated from the alligator cochlea. *Journal of Comparative Physiology Part A: Neuroethology, Sensory, Neural and Behavioral Physiology, 164*(2), 151-163.

Fujimoto, C., Murofushi, T., Chihara, Y., Ushio, M., Yamaguchi, T., Yamasoba, T., & Iwasaki, S. (2010). Effects of unilateral dysfunction of the vestibular nerve system on postural stability. *Clinical Neurophysiology, 121*(8), 1279-1284.

Furuya, N., Kawano, K., & Shimazu, H. (1975). Functional organization of vestibulofastigial projection in the horizontal semicircular canal system in the cat. *Experimental Brain Research, 24*(1), 75-87.

Geisler, C.D., Frishkopf, L.S., & Rosenblith, W.A. (1958). Extracranial responses to acoustic clicks in man. *Science, 128*(3333), 1210-1211.

Gerrits, N.M. (1990). Vestibular nuclear complex. In G. Paxnos (Ed.), *The human nervous system.* San Diego, CA: Academic Press.

Ghanem, T.A., Breneman, K.D., Rabbitt, R.D., & Brown, H.M. (2008). Ionic composition of endolymph and perilymph in the inner ear of oyster toadfish, *Opsanus tau. The Biological Bulletin, 214,* 83-90.

Goldberg, J.M. (2000). Afferent diversity and the organization of central vestibular pathways. *Experimental Brain Research, 130*(3), 277-297.

Goldberg, J.M., Desmadryl, G., Baird, R.A., & Fernández, C. (1990). The vestibular nerve of the chinchilla. V. Relation between afferent discharge properties and peripheral innervation patterns in the utricular macula. *Journal of Neurophysiology, 63,* 791-804.

Goldberg, J.M., & Fernández, C. (1971). Physiology of peripheral neurons innervating semicircular canals of the squirrel monkey. I. Resting discharge and response to constant angular accelerations. *Journal of Neurophysiology, 34,* 635-660.

Goodman, M.B., & Art, J.J. (1996a). Variations in the ensemble of potassium currents underlying resonance in turtle hair cells. *Journal of Physiology, 497*(Pt 2), 395-442.

Goodman, M.B., & Art, J.J. (1996b). Positive feedback by a potassium-selective inward rectifier enhances tuning in vertebrate hair cells. *Biophysical Journal, 71*(1), 430-442.

Gstoettner, W., Plenk, Jr., H., Franz, P., Hamzavi, J., Baumgartner, W., Czerny, C., & Ehrenberger, K. (1997). Cochlear implant deep electrode insertion: Extent of insertional trauma. *Acta Oto-Laryngologica, 117*(2), 274-277.

Gray, O. (1955). A brief survey of the phylogenesis of the labyrinth. *Journal of Laryngology & Otology, 69,* 151-179.

Halmagyi, G.M., Curthoys, I.S., Colebatch, J.G., & Aw, S.T. (2006). Vestibular responses to sound. *Annals of the New York Academy of Sciences, 1039,* 54-67.

He, D.Z.Z., & Dallos, P. (1999). Somatic stiffness of cochlear outer hair cells is voltage-dependent. *Proceedings of the National Academy of Sciences in the United States of America, 96*(14), 8223-8228.

Hillman, D.E., & McLaren, J.W. (1979). Displacement configuration of semicircular canal cupulae. *Neuroscience, 4*(12), 1989-2000.

Huygen, P.L., Hinderink, J.B., Broek, P., Van Den Borne, S., Van Den Brokx, J.P.L., Mens, L.H.M., & Admiraal, R.J.C. (1995). The risk of vestibular function loss after intracochlear implantation. *Acta Oto-Laryngologica, 115*(1), 270-273.

Isaradisaikul, S., Strong, D.A., Moushey, J.M., Gabbard, S.A., Ackley, R.S., & Jenkins, H.A. (2008). Reliability of vestibular evoked myogenic potentials in healthy subjects. *Otology & Neurotology, 29,* 542-544.

Ito, J. (1998). Influence of multichannel cochlear implant on vestibular function. *Otolaryngology-Head & Neck Surgery, 118*(6), 900-902.

Jacot, E., Van Den Abbeele, T., Debre, H.R., & Weiner-Vacher, S.R. (2009). Vestibular impairments pre- and post-cochlear implant in children. *International Journal of Pediatric Otorhinolaryngology, 73,* 209-217.

Jerger, J. (1970). Clinical experience with impedance audiometry. *Archives of Otolaryngology-Head & Neck Surgery, 92*(4), 311-324.

Jin, Y., Nakamura, M., Shinjo, Y., & Kaga, K. (2006). Vestibular-evoked myogenic potentials in cochlear implant children. *Acta Oto-Laryngologica, 126*(2), 164-169.

Jonkees, L.B., Maas, J.P., & Phillipszoon, A.J. (1962). Clinical nystagmography: A detailed study of electronystagmography. *Practica Oto-Rhino-Laryngologica, 24,* 25-29.

Kelsch, T.A., Schaefer, L.A., & Esquivel, C.R. (2006). Vestibular evoked myogenic potentials in young children: Test parameters and normative data. *Laryngoscope, 116,* 895-900.

King, S. (2009). *Changes in saccular function after cochlear implantation.* Session presented at the annual meeting of the American Academy of Audiology, Dallas, TX.

Krause, E., Louza, J.P.R., Hempel, J.-M., Wechtenbruch, J., Rader, T., & Gürkov, R. (2009). Effect of cochlear implantation on horizontal semicircular canal function. *European Archives of Oto-Rhino-Laryngology, 266,* 811-827.

Kubo, T., Yamamoto, K.-i., Iwaki, T., Doi, K., & Tamura, M. (2001). Different forms of dizziness occurring after cochlear implant. *European Archives of Oto-Rhino-Laryngology, 258,* 9-12.

Lapeyre, P.N.M., Guilaume, A., & Cazals, Y. (1992). Differences in hair cell bundles associated with type I and type II vestibular hair cells of the guinea pig saccule. *Acta Oto-Laryngologica, 112,* 635-642.

Lewis, E.R., Baird, R.A., Leverenz, E.L., & Koyama, H. (1982). Inner ear: Dye injection reveals peripheral organ of specific sensitivities. *Science, 215*(4540), 1641-1643.

Li, M.W., Houlden, D., & Tomlinson, R.D. (1999). Click evoked EMG responses in sternocleidomastoid muscles: Characteristics in normal subjects. *Journal of Vestibular Research, 9,* 327-334.

Lim, C.L., Clouston, P., Sheean, G., & Yiannikas, C. (1995). The influence of voluntary EMG activity and click intensity on the vestibular click evoked myogenic potential. *Muscle & Nerve, 18,* 1210-1213.

Lindeman, H.H. (1969). Regional differences in sensitivity of the vestibular sensory epithelia to ototoxic antibodies. *Acta Oto-Laryngologica, 67*(2-6), 177-189.

López, I., Honrubia, V., & Baloh, R.H. (1997). Aging and the human vestibular nucleus. *Journal of Vestibular Research, 7*(1), 77-85.

Lowenstein, A., & Roberts, T.D.M. (1951). The localization and analysis of the responses to vibration from the isolated elasmobranches labyrinth. *Journal of Physiology (London), 114,* 471-489.

Lysakowski, A. (1999). Development of synaptic innervation in the rodent utricle. *Annals of the New York Academy of Sciences, 87,* 422-425.

Lysakowski, A., & Goldberg, J.M. (2004). Morphophysiology of the vestibular sensory periphery. In S.M. Highstein, R.R. Fay, & A.N. Popper (Eds.), *The vestibular system.* Berlin, Germany: Springer-Vertiag.

Lysakowski, A., & Goldberg, J.M. (1997). A regional ultra-structure analysis of the cellular and synaptic architecture in the chinchilla cristae ampullares. *Journal of Comparative Neurology, 389,* 419-443.

Lysakowski, A., Minor, L.B., Fernández, C., & Goldberg, J.M. (1995). Physiological identification of morphologically distinct afferent classes innervating the cristae ampullares of the squirrel monkey. *Journal of Neurophysiology, 73,* 1270-1281.

Marin, R. (2010). *An electrophysiological examination of saccular function in participants with GJB2 deafness.* (Unpublished doctoral dissertation). Gallaudet University, Washington DC.

Markham, C.H. (1989). Anatomy and physiology of otolith-controlled ocular counterrolling. *Acta Oto-Laryngologica, 108*(Suppl 468), 263-266.

Masaki, Y., Ogasawra, K., Yoshikawa, H., Watanabe, M., Furukawa, T., Ando, I., & Ichikawa, G. (2002). Cervical reflex induced by click stimuli in cats. *Acta Oto-Laryngologica, 122,* 607-612.

Matsuzaki, M., & Murofushi, T. (2002). Click-evoked potentials on the neck of the guinea pig. *Hearing Research, 165,* 152-155.

McCue, M.P., & Guinan, Jr., J.J. (1997). Sound-evoked activity in primary afferent neurons of a mammalian vestibular system. *Otology & Neurotology, 18*(3), 355-360.

McCue, M.P., & Guinan, Jr., J.J. (1995). Spontaneous activity and frequency selectivity of acoustically responsive vestibular afferents in the cat. *Journal of Neurophysiology, 74*(4), 1563-1572.

McCue, M.P., & Guinan, Jr., J.J. (1994a). Acoustically responsive fibers in the vestibular nerve of the cat. *Journal of Neuroscience, 14,* 6058-6070.

McCue, M.P., & Guinan, Jr., J.J. (1994b). Influence of efferent stimulation on acoustically responsive vestibular afferents in the cat. *Journal of Neuroscience, 14,* 6071-6083.

McLaren, J.W., & Hillman, D.E. (1979). Displacement of the semicircular canal cupula during sinusoidal rotation. *Neuroscience, 4*(12), 2001-2008.

Melagrana, A., D'Agostino, R., Pasqual, G., & Taborelli, G. (1996). Study of labyrinthine function in children using the caloric test: Our results. *International Journal of Pediatric Otorhinolaryngology, 37,* 1-8.

Melvin, T.-A.N., Della Santina, C.C., Carey, J.P., & Migliaccio, A.A. (2008). The effects of cochlear implantation on vestibular function. *Otology & Neurotology, 30,* 87-94.

Mikaelian, D. (1964). Vestibular response to sound: Single unit recording from the vestibular nerve in fenestrated deaf mice (Df/Df). *Acta Oto-Laryngologica, 58,* 409-422.

Minor, L.B. (1999). Intratympanic gentamicin for control of vertigo in Meniere's disease: Vestibular signs that specify completion of therapy. *American Journal of Otology, 20*(2), 209-219.

Minor, L.B., Lasker, D.M., Backous, D.D., & Huller, T.E. (1999). Horizontal vestibuloocular reflex evoked by high-acceleration rotations in the squirrel monkey. I. Normal responses. *Journal of Neurophysiology, 82*(3), 1254-1270.

Moffat, A.J.M., & Caprianica, R.R. (1976). Auditory sensitivity of the saccule in the American toad (*Bufo americanus*). *Journal of Comparative Physiology A: Neuroethology, Sensory, Neural & Behavioral Physiology, 105*(1), 1-8.

Morsli, H., Choo, D., Ryan, A., Johnson, R., & Wu, D.K. (1998). Development of the mouse inner ear and origin of its sensory organs. *Journal of Neuroscience, 18*(9), 3327-3335.

Murofushi, T., & Curthoys, I.S. (1997). Physiological and anatomical study of click-sensitive afferents in the guinea pig. *Acta Oto-Laryngologica, 117*(1), 66-72.

Murofushi, T., Curthoys, I.S., & Gilcrest, D.P. (1996). Response to guinea pig vestibular nucleus neurons to clicks. *Experimental Brain Research, 111*(1), 149-152.

Murofushi, T., Curthoys, I.S., Topple, A.N., Colebatch, J.G., & Halmagyi, G.M. (1995). Responses of guinea pig primary vestibular neurons to clicks. *Experimental Brain Research, 103,* 174-178.

Murofushi, T., Matsuzaki, M., & Takegoshi, H. (2001). Glycerol affects vestibular evoked myogenic potentials in Meniere's disease. *Auris Nasus Larynx, 28,* 205-208.

Naito, Y., Newman, A., Lee, W.S., Beykirch, K., & Honrubia, V. (1995). Projections of the individual vestibular end-organs in the brainstem of the squirrel monkey. *Hearing Research, 87,* 141-155.

Nong, D.X., Ura, M., Kyuna, A., Owa, T., & Noda, Y. (2002). Saccular origin of acoustically evoked short latency response. *Otology & Neurotology, 23*(6), 953 -957.

Ochi, K., Ohashi, T., & Nishino, H. (2001). Variance of vestibular-evoked myogenic potentials. *Laryngoscope, 111*(3), 522-527.

Park, J.J., Tang, Y., López, I., & Ishiyama, A. (2001). Age-related change in the number of neurons in the human vestibular ganglion. *Journal of Comparative Neurology, 431*(4), 437-443.

Phillips, J., & Backous, D. (2002). Evaluation of vestibular function in young children. *Otolaryngology Clinics of North America, 35*(4), 765-790.

Popper, A.N., & Fay, R.R. (1973). Sound detection and processing by teleost fishes: A critical review. *Journal of the Acoustical Society of America, 53*(6), 1515-1529.

Qu, Y., Yang, W., Dahike, I., Ding, D., Salvi, R., Söhl, G., …Lin, X. (2007). Analysis of connexin subunits required for the survival of vestibular cells. *Journal of Comparative Neurology, 504*(5), 499-507.

Rabbitt, R.D., Boyle, R., & Highstein, S.M. (2004). Physiology of the semicircular canals after surgical plugging. *Annals of the New York Academy of Sciences, 17,* 9-16.

Rask-Anderson, H., Tylstedt, S., Kinnefors, A., & Schrott-Fischer, A. (1997). Nerve fibre interaction with large ganglion cells in the human spiral ganglion. A TEM study. *Auris Nasus Larynx, 24,* 1-11.

Rauch, S.D. (2006). Vestibular evoked myogenic potentials. *Current Opinion in Otolaryngology-Head & Neck Surgery, 14,* 299-304.

Rauch, S.D., Zhou, G., Kujawa, S.G., Guinan, J.J., & Herrmann, B.S. (2004). Vestibular evoked myogenic potentials show altered tuning in patients with meniere's disease. *Otology & Neurotology, 25*(3), 333-338.

Rebscher, S.J., Heilman, M., Bruszewski, W., Talbot, N.H., Snyder, R.L., & Merzenich, M.M. (1999). Strategies to improve electrode positioning and safety in cochlear implants. *IEEE Transactions on Biomedical Engineering, 46*(3), 340-352.

Ribaric, K., Previc, T., & Kozina, V. (1984). Frequency-following response evoked by acoustic stimuli in normal and profoundly deaf subjects. *Audiology, 23,* 388-400.

Richter, E. (1980). Quantitative study of human Scarpa's ganglion and vestibular sensory epithelia. *Acta Oto-Laryngologica, 90*(1-6), 199-208.

Robertson, D.D., & Ireland, D.J. (1995). Vestibular evoked myogenic potentials. *Journal of Otolaryngology, 24,* 3-7.

Rosenhall, U. (1970). Some morphological principles of the vestibular maculae in birds. *European Archives of Oto-Rhino-Laryngology, 197*(2), 154-182.

Sadler, T.W. (2004). *Langman's medical embryology.* Philadelphia, PA: Lippincott, Williams & Wilkins.

Saidel, W.M., & Popper, A.M. (1986). The saccule may be the transducer for directional hearing of nonstariophysine teleosts. *Experimental Brain Research, 50,* 149-152.

Salt, A.N. (2001). Regulation of endolymphatic fluid volume. *Annals of the New York Academy of Sciences, 942,* 306-312.

Salt, A.N., & DeMott, J.E. (2000). Ionic and potential charges of the endolymphatic sac induced by endolymph voltage changes. *Hearing Research, 149*(1-2). 46-54.

Sandberg, L.E., & Terkildsen, K. (1965). Caloric tests in deaf children. *Archives of Otolaryngology, 81,* 350-354.

Sato, H., Imagawa, M., Isu, N., & Uchino, Y. (1997). Properties of saccular nerve-activated vestibulospinal neurons in cats. *Experimental Brain Research, 116*(3), 381-388.

Scherer, H., & Watanabe, S. (2001). Introductory remarks on this issue on the role of the ampulla in disturbances of vestibular function. *Biological Sciences in Space, 15*(4), 350-352.

Schubert, M., & Shepard, N. (2008). Practical anatomy and physiology of the vestibular system. In G. Jacobson & N. Shepard (Eds.), *Balance function assessment and management.* San Diego, CA: Plural Publishing.

Shall, M.S. (2009). The importance of saccular function to motor development in children with hearing impairments. *International Journal of Otolaryngology.* Retrieved from http://www.hindawi.com/journals/ijol/2009/972565.html

Sheykholeslami, K., & Kaga, K. (2002). The otolith organ as a receptor of vestibular hearing revealed by vestibular-evoked myogenic potentials in patients with inner ear anomalies. *Hearing Research, 165*(1-2), 62-67.

Sheykholeslami, K., Kermany, M.H., & Kaga, K. (2001). Frequency selectivity range of the saccule to bone-conducted stimuli measured by vestibular evoked myogenic potentials. *Hearing Research, 160,* 58-62.

Sheykholeslami, K., Murofushi, T., & Kaga, K. (2001). The effect of sternocleidomastoid electrode location on vestibular evoked myogenic potential. *Auris Nasus Larynx, 28,* 41-48.

Sheykholeslami, K., Schmerber, S., Kermany, M.H., & Kaga, K. (2004). Vestibular-evoked myogenic potentials in three patients with large vestibular aqueduct. *Hearing Research, 190,* 161-168.

Shinjo, Y., Jin, Y., & Kaga, K. (2007). Assessment of vestibular function of infants and children with congenital and acquired deafness using the ice-water caloric test, rotational chair test and vestibular-evoked myogenic potential recording. *Acta Oto-Laryngologica, 127*(7), 736-747.

Snashall, S.E. (1983). Vestibular function tests in children. *Journal of the Royal Society of Medicine, 76,* 555-559.

Shumway-Cook, A., & Woollacott, M.H. (1985). The growth of stability: Postural control from a developmental prospective. *Journal of Motor Behavior, 90*(4), 1101-1112.

Staller, S.J., Goin, D.W., & Hildebrandt, M. (1986). Pediatric vestibular evaluation with harmonic acceleration. *Otolaryngology-Head & Neck Surgery, 95,* 471-476.

Steenerson, R.L., Cronin, G.W., & Gary, L.B. (2001). Vertigo after cochlear implantation. *Otology & Neurotology, 22*(6), 842-843.

Stein, M.B., & Carpenter, M.B. (1967). Central projections of portions of the vestibular ganglia innervating specific parts of the labyrinth in the rhesus monkey. *American Journal of Anatomy, 120,* 281-318.

Stevens, S.S., & Warhofsky, F. (1965). *Sound and hearing.* New York, NY: Time.

Streubel, S.-O., Cremer, P.D., Carey, J.P., Weg, N., & Minor, L.B. (2001). Vestibular-evoked myogenic potentials in the diagnosis of superior canal dehiscence syndrome. *Acta Oto-Laryngologica, 121*(545), 41-49.

Su, H.-C., Huang, T.-W., Young, Y.-H., & Cheng, P.-W. (2004). Aging effect on vestibular evoked myogenic potential. *Otology & Neurotology, 25,* 977-980.

Szirmai, Á., Ribári, O., & Répássy, G. (2001). Air caloric computer system application in monitoring vestibular function changes after cochlear implantation. *Otolaryngology-Head & Neck Surgery, 125,* 631-634.

Tien, H.-C., & Linthicum, Jr., F.H. (2002). Histopathological changes in the vestibule after cochlear implantation. *Otolaryngology-Head & Neck Surgery, 127,* 260-264.

Todt, I., Basta, D., & Ernst, A. (2008). Does the surgical approach in cochlear implantation influence the occurrence of postoperative vertigo? *Otolaryngology-Head & Neck Surgery, 138,* 8-12.

Townsend, G.L., & Cody, D.T. (1971). The averaged inion response evoked by acoustic stimulation: Its relation to the saccule. *Annals of Otology, Rhinology & Laryngology, 80*(1), 121-131.

Tribukait, A., Brantberg, K., & Bergenius, J. (2004). Function of semicircular canals, utricles and saccules in deaf children. *Acta Oto-Laryngologica, 124,* 41-48.

Tullio, P. (1938). Démonstration des methods pour la stimulation acoustique des canaux semicirculaire. *Acta Oto-Laryngologica (Stockholm), 26,* 267.

Uchino, Y. (1997). Connections between otolith receptors and neck motoneurons. *Acta Oto-Laryngologica, 30*(4), 49-51.

Uchino, Y., Sato, H., Sasaki, M., Imagawa, M., Ikegami, H., Isu, N., & Graf, W. (1997). Sacculocolic reflex arcs in cats. *Journal of Neurophysiology, 77*(6), 3003-3012.

Verbist, B.M., Ferrarini, L., Briaire, J.J., Zarowski, A., Admiraal-Behloul, F., Olofsen, H., …Frijns, J.H. (2009). Anatomic considerations of cochlear morphology and its implications for insertion trauma in cochlear implant surgery. *Otology & Neurotology, 30*(4), 471-477.

Vibert, D., Häusler, R., Kompis, M., & Vischer, M. (2001). Vestibular function in patients with cochlear implantation. *Acta Oto-Laryngologica,* Supplement 545, 29-34.

Von Békésy, G. (1935). Uber akustische Reizung des Vestibularapparetes. *Pfügers Arch, 236,* 59-76.

Wang, C.-T., & Young, Y.-H. (2006). Comparison of head elevation versus rotation method in eliciting vestibular evoked myogenic potentials. *Ear & Hearing, 27,* 376-381.

Welgampola, M., & Colebatch, J. (2001). Vestibulocollic reflexes: Normal values and the effect of age. *Clinical Neurophysiology, 112*(11), 1971-1979.

Welgampola, M.S., Rosengren, S.M., Halmagyi, G.M., & Colebatch, J.G. (2003). Vestibular activation by bone conducted sound. *Journal of Neurology, Neurosurgery & Psychiatry, 74,* 771-778.

Werner, C.F. (1933). Die differenzierung der maculae im labyrinth, insbesondere bei säugetieren. *Zeitschrift Fuer Anatomie und Entwicklungsgeschichte, 99,* 696-706.

Wersäll, J. (1956). Studies on the structure and innervation of the sensory epithelium of the cristae ampullares in the guinea pig. A light and electron microscopic investigation. *Acta Oto-Laryngologica (Stockholm),* Supplement 126, 1-85.

Wersäll, J., & Bagger-Sjöbäck, D. (1974). Morphology of the vestibular sense organ. In H.H. Kornhuber (Ed.), *Handbook of sensory physiology, vestibular system basic mechanisms.* New York, NY: Springer.

Wit, H.P., Bleeker, J.D., & Mulder, H.H. (1984). Responses of pigeon vestibular nerve fibers to sound and vibration with audiofrequencies. *Journal of the Acoustical Society of America, 75*(1), 202-208.

Worchel, P., & Dallenbach, K.M. (1950). Vestibular sensitivity in the deaf. *American Journal of Psychology, 63,* 161-175.

Wu, C.-C., & Young, Y.-H. (2002). Vestibular evoked myogenic potentials are intact for sudden deafness. *Ear & Hearing, 23*(3), 235-238.

Wu, C.H., & Murofushi, T. (1999). Tone burst-evoked myogenic potentials in human neck flexor and extensor. *Acta Oto-Laryngologica, 119,* 741-744.

Yamauchi, A., Rabbitt, R.D., Boyle, R., & Highstein, S.M. (2002). Relationship between inner-ear fluid pressure and semicircular canal afferent nerve damage. *Journal of the Association for Research in Otolaryngology, 3*(1), 26-44.

Yang, T.-H., & Young, Y.-H. (2005). Click-evoked myogenic potentials recorded on alert guinea pigs. *Hearing Research, 205,* 277-283.

Young, E.D., Fernández, C., & Goldberg, J.M. (1977). Responses of squirrel monkey vestibular neurons to audio-frequency sound and head vibrations. *Acta Oto-Laryngologica, 84*(1-6), 352-360.

Young, Y.-H., Chen, C.-N., Hsieh, W.-S., & Wan, S.J. (2009). Development of vestibular evoked myogenic potentials in early life. *European Journal of Paediatric Neurology, 13*(3), 235-239.

Young, Y.-H., Wu, C.-C., & Wu, C.-H. (2001). Augmentation of vestibular evoked myogenic potentials: An indicator for distended saccular hydrops. *Laryngoscope, 112*(3), 509-512.

Zanetti, D., Campovecchi, C.B., Belzanelli, C., & Pasini, S. (2007). Paroxysmal positional vertigo after cochlear implantation. *Acta Oto-Laryngologica, 127*(5), 452-458.

Zemlin, W.R. (1998). *Speech and hearing science: Anatomy and physiology* (4th ed.). Needham Heights, MA: Allyn & Bacon.

Zhou, G., & Cox, L.C. (2004). Vestibular evoked myogenic potentials: History and overview. *American Journal of Audiology, 13,* 135-143.

Zhou, G., Kenna, M.A., Stevens, K., & Licameli, G. (2009). Assessment of saccular function in children with sensorineural hearing loss. *Archives of Otolaryngology-Head & Neck Surgery, 135*(1), 40-44.