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Best practices for ocular and cervical VEMP tests

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BEST PRACTICES FOR OCULAR AND CERVICAL VEMP TESTS.

By

CDR (Sel) Matthew Zachary Thomas, M.A.

A Capstone Project

submitted in partial fulfillment of the

requirements for the degree of:

Doctor of Audiology

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Abstract: The VEMP is a relatively recent addition to the clinician’s test battery. The following Capstone will provide protocol for the use of both VEMP tests as part of a complete vestibular diagnostic test battery.
ACKNOWLEDGEMENTS

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I would also like to thank Dr. Belinda Sinks and Dr. Heather Monroe for their key role in introducing me to vestibular audiology and helping me learn so much on the job. Lastly I would also like to thank my family for their support for the past four years, as well as my classmates who were there for me throughout my entire scholastic career.
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<tr>
<td>CDP</td>
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Introduction

The vestibular system is one of the more complex systems in the human body, as such disorders of this system can be complicated to diagnose much less treat. Due to multiple system involvements a clinical evaluation of the vestibular system relies on a number of different and complex tests. These tests study not only the vestibular organs, but visual and proprioceptive inputs as well. As the study of the vestibular system has progressed new objective tests have been introduced that rely on electrophysiological measures to study the vestibular end organs. These are the Vestibular Evoked Myogenic Potential (VEMP) tests; the methods and reasons for their use are the subject of this capstone.

The human balance mechanism is comprised of sensory modalities from the vestibular, proprioceptive, and visual systems. These modalities provide cues which are responsible for a sensory negative feedback mechanism that works to maintain the body in an upright position during external stimuli (Mauerer, Mergner, Peterka, 2006). A deficit in one modality will often lead to balance instability (Dozza, Horak, Chiari, 2007) or an over-reliance on one of the remaining sensory modalities (Shumway-Cook, Horak, 1986). Because of this diverse nature of the vestibular system, diagnosing the causes of a vestibular deficiency requires multiple tests that investigate these modalities. To further complicate matters, the inner ear contains multiple vestibular organs: three semi-circular canals which detect rotational movement and the two organs that comprise the otoliths, the utricle and saccule which detect linear acceleration.

As mentioned, the semicircular canals and the two otolith organs are the vestibular end organs, working in conjunction with the vestibular nerve. They work together to allow the body to monitor the movement of the head, rotational and linear, as well as the orientation of the head
in regards to gravity (Murofushi, Kaga, 2009). The three semicircular canals, lateral, anterior, and posterior, lie in such a manner as to form a coordinate system that detects angular acceleration, i.e. head movement. The lateral canal lies horizontally while the anterior and posterior canals lie in the vertical position, all three lay approximately 90 degrees from the other.

All three semicircular canals have an ampulla at the end which contains the sensory portion of the canals, the crista (Murofushi, Kaga, 2009). Embedded in the crista are vestibular hair cells, or stereocilia. In turn these stereocilia are embedded in a cupula, a gelatinous mass that lies atop the ampulla. Vestibular hair cells are different from their auditory counterparts in that there is one tallest hair cell known as the kinocilium. The stereocilia are activated when fluid from one of the semicircular canals pushes against the cupula which in turn bends the stereocilia. When the stereocilia bend the tip links at the top of the hair cells open and release potassium which generates neural impulses in the VIIIth cranial nerve. If the hair cells are bent towards the kinocilium the released potassium depolarizes the hair cells and causes an increase in afferent activity, away from the kinocilium causes hyperpolarization and a decrease in afferent neural activity (Sanders, Gillig, 2010). The semicircular canals themselves work in pairs; movement in one canal triggers the opposite movement in another, which causes depolarization and hyperpolarization to occur, thus allowing the brain to detect the direction of movement of the head.

The two organs of the otolith, the utricle and the saccule lie against the wall of the inner ear between the semicircular canals and the cochlea. The sensory portion of the otolith is known as the macula which contains patches of hair cells. The saccular macula lies vertically and beneath the utricular macula which lies horizontally. The two maculae lie approximately ninety degrees to each other. The surface of the maculae are covered by a membrane which contains
calcium carbonate crystals known as otoconia. These otoconia embedded in the membrane lie atop the hair cells and their movement causes the activation of the otolithic hair cells. Because of the weight of the otoconia, any head position will cause the activation of at least some region of hair cells sending afferent activity to the VIIIth nerve. Thus the otoliths are always activated in some part due to gravitational forces consistently acting on the body. Unlike the stereocilia in the semicircular canals, the hair cells of the otoliths are not oriented in one consistent direction. Rather the hair cells of the utricle point toward a midline, known as the striola, while the hair cells of the saccule point away. Because of this orientation the utricle is most sensitive to tilt when the head is upright, while the saccule is most sensitive to tip when the head is horizontal.

Both the semicircular canals and the otolithic organs transmit afferent activity to the VIIIth cranial nerve which in turn contains both afferent and efferent nerve fibers. The vestibular portion of the VIIIth nerve is divided into two parts, the superior and inferior vestibular nerve. The superior vestibular nerve is responsible for innervating the anterior and lateral semicircular canals as well as the utricular macula. It also innervates the anterosuperior portion of the saccular macula (Murofushi, Kaga, 2009), while the inferior vestibular nerve innervates the majority of the saccular macula as well as the posterior semicircular canal. The vestibular afferent nerves fire spontaneously; they are categorized as either regularly firing fibers or irregularly firing fibers. The irregularly firing fibers have a high sensitivity to head rotation while the regularly firing fibers have low sensitivity to head rotation or linear movement.

All of the afferent nerves synapse into vestibular nuclei, which is a group of neurons located in the fourth ventricle (Sanders, Gillig, 2010). The vestibular nuclei are divided into the superior and inferior nucleus, as well as the lateral and medial nucleus. The four types of vestibular nuclei receive afferent fibers from different sources of the vestibular system. The
semicircular canals primarily project their afferents to the superior and medial vestibular nuclei. The two organs of the otolith project their afferents to all but the superior vestibular nucleus. Saccular afferents project primarily to the inferior vestibular nucleus as well as the lateral, while utricular afferents project to the inferior and medial vestibular nuclei.

The various components of the vestibular system work together to maintain not just balance, but stability, position, and overall body control. There are three vestibular reflexes that work to control these various aspects in the human body: the Vestibulospinal Reflex (VSR), the Vestibulocollic Reflex (VCR), and the Vestibuloocular Reflex (VOR). The VSR is the reflex responsible for stabilizing the head as well as preventing falls and maintaining the body’s position, both during static and dynamic positioning (Murofushi, Kaga, 2009). The three main pathways of the VSR are the lateral vestibulospinal tract (LVST), which originates in the lateral nucleus, the medial vestibulospinal tract (MVST), which originates in the medial, inferior, and lateral nuclei, and the reticulospinal tract (RST). When the otolithic end organs are stimulated they activate the vestibular nerve and the vestibular nuclei which then transmit the impulse down the LVST, MVST, and RST ultimately ending in extensor and flexor muscles in the neck. These two muscle types work in conjunction with each other; once the extensor muscles on one side are activated the flexor muscles on the opposite are activated as well, thus creating the VSR.

The second of the vestibular reflexes is the VCR which is responsible for stabilizing the head via movements of the neck. Neurons in the neck work through the MVST and the LVST to connect with the vestibular end organs to activate the VCR (Wilson, et al, 1995). The VCR is the basis of cervical VEMP testing. Although the precise pathway of the VCR isn’t known, it is believed that stimulation of the otolithic end organs causes afferent activity down the MVST, LVST and spinal accessory nerve to provide direct and indirect connections to motoneurons in
the neck muscles (Rosengren, Welgampola, Colebatch, 2010). There are three classifications of neck muscles, the extensor and flexor muscles mentioned previously, as well as rotator muscles. The motoneurons of the sternocleidomastoid muscle (SCM), a rotator muscle, is where the afferent signal terminates and activates the VCR. It is myogenic activity measured from the vestibular activation of the SCM that forms the foundation of the cervical VEMP.

The last of the reflexes is the VOR, which works to maintain a person’s gaze during movements of the head and/or body. When the head moves to one side the VOR moves the eyes together in the opposite direction and at the same time, thereby keeping the image in the same area of the retina. There are three main components that make up the VOR: the peripheral sensory component, the central processing mechanism, and the motor output (Fetter, 2007). The peripheral sensory component is made up of the vestibular organs, the semicircular canals and the two otolithic organs while the motor output consists of various eye muscles. The primary muscles involved in the VOR are the lateral rectus muscle which is responsible for rotating the eyes laterally and the medial rectus muscles which are responsible for pulling the eyes in. The lateral rectus muscle is innervated by cranial nerve VI, the abducens nerve, while the medial rectus muscle is innervated by cranial nerve III, the oculomotor nerve.

The VOR works in an inhibitory/excitatory manner; that is the activation, or excitation, of the system on one side of the body will cause the inhibition of the opposing side. Acceleration of one side of the head will cause hyperpolarization of that side’s vestibular system while simultaneously causing depolarization of the opposite side’s vestibular system (Roeser, 2013). If the head were to turn to the right, the vestibular afferents in the right horizontal semicircular canal would hyperpolarize while the left horizontal semicircular canals would depolarize. The excitation of the right vestibular system would lead to the excitation of the left
abducens nerve and the right oculomotor nerve and the inhibition of the right abducens and left oculomotor cranial nerves. As a result, as the head turns right the eye will slowly turn to the left until it reaches maximum ocular deviation, whereupon the eye will move rapidly back to the right; this is known as nystagmus (Fetter, 2007). The most commonly used method of testing the integrity of a person’s VOR is by rotational chair testing as well as the Ocular VEMP test (Rosengren, et al, 2010).

**Vestibular Disorders**

**Meniere’s Disease:**

Because the human balance system is such a complex system, there are a multitude of disorders than can affect a person’s equilibrium. For the sake of brevity only the major disorders that are commonly seen by a clinician will be discussed. Of the most common equilibrium disorders seen in a vestibular clinic the oVEMP and cVEMP will oftentimes but not always be a useful diagnostic tool. Meniere’s disease and benign paroxysmal positional vertigo (BPPV) are two of the most common disorders diagnosed by physicians during an initial consultation. Meniere’s disease is an inner ear disorder that causes episodes of spontaneous vertigo that can last up to several hours at a time, as well as fluctuating hearing loss, fullness in the ear, and tinnitus (Vassiliou, Vlastarakos, Maragoudakis, Candiloros, Nikolopoulos, 2011). Meniere’s is often unilateral in nature, though cases of bilateral Meniere’s have been reported. Doctors still do not know the exact cause of Meniere’s but the most commonly accepted reason is due to a build of endolymphatic fluid inside the membranous labyrinth.

Recent studies have suggested that the disease most often affects the saccule and the cochlea first, with the utricle and semicircular canals being affected later (Taylor, et al, 2010).
The same study showed that Meniere’s disease can produce asymmetrical amplitudes between the affected and unaffected ears or absent responses in the affected ear. In oVEMP s this was found to be the case 65% of the time, and only 45% of the time in cVEMP s. This discrepancy in sensitivity lends credence to the belief that the saccule is affected primarily with Meniere’s disease. Another study by Manzari, et al utilized oVEMP and cVEMP measures on Meniere’s patients during periods when the disease was inactive and periods of acute attacks. Utilizing bone conducted 500 Hz tone-bursts for both VEMPs the researchers tested 16 normal subjects to establish a normative baseline, then 15 Meniere’s patients during periods of acute attacks and periods of dormancy (Manzari, Tedesco, Brugess, Curthoys, 2010). The researchers found that during periods of dormancy the n10 component of the oVEMP had equal amplitude for both ipsilateral and contralateral responses. However, during periods of acute attack the n10 component of the contralateral response had significantly higher amplitude than the ipsilateral side. Conversely, the researchers found that during periods of acute attack the ipsilateral p13 component of the cVEMP showed significantly reduced amplitude compared to the contralateral response. From these results Manzari, et al (2010) conclude that during periods of acute attacks of Meniere’s disease the utricular function in the affected ear is enhanced while the saccular function is reduced.

**Benign Paroxysmal Positional Vertigo:**

Benign paroxysmal positional vertigo, or BPPV is the most common cause of vertigo in adults (Bhattacharyya, et al, 2008). The most common symptom of BPPV is periods of dizziness and unbalance typically lasting less than one minute in duration. BPPV occurs when the otoconia that are embedded in the maculae become dislodged and migrate into the semicircular canals, most typically the posterior canal. Although the causes of dislodgement aren’t always
known, head trauma is known to be one of the most common factors. Symptoms of BPPV are triggered with changes in head position, which cause the dislodged otoconia to stimulate the cupula and send the brain false signals of movement, thus triggering dizziness. BPPV can typically be rectified through treatment involving maneuvering of the head in order to work the otoconia back into place.

Clinically BPPV is most likely to be diagnosed through VNG testing with positional tests known as Dix-Hallpike maneuvers (Lopez-Escamez, 2009). Traditionally VEMPs have not been used in the diagnosis of BPPV but a recent study shows that they may be clinically useful in diagnosing BPPV. In a study by Bremova, et al 30 patients with posterior canal BPPV were given bone-conducted oVEMPs and air-conducted cVEMP tests prior to being given any treatment. Once treatment was given with the Semont maneuver the clinicians conducted further VEMP testing. The tests were administered immediately following treatment, one week and finally one month after treatment was given. The researchers found that one week later treatment was effective for 20 of the 30 participants; of these 20 subjects oVEMP amplitudes were significantly increased, whereas cVEMP responses showed no changes (Bremova, et al, 2013). The reason for the increased amplitude of the oVEMPs is believed to be due to the repositioning of the otoconia back onto the utricular cupula. The researchers further hypothesized that the reason amplitudes were increased following a week rather than immediately following treatment was likely because it took several days for the repositioned otoconia to become re-embedded into the cupula.
Vestibular Neuritis/Labyrinthitis:

Two vestibular disorders that are similar in nature to each other are vestibular neuritis and labyrinthitis. Both disorders are caused by infections in the inner ear, typically viral, that affect the VIIIth nerve. As a result of the infection a person may experience dizziness and vertigo, loss of balance, and in the case of labyrinthitis tinnitus and hearing loss may occur (Cooper, 1993). As the name suggests, vestibular neuritis is an inflammation of the VIIIth nerve that only affects the branch of the nerve associated with balance. Often a person suffering from neuritis has recently had or still is affected by an upper respiratory tract infection (Cooper, 1993). Episodes of vertigo are typically sudden and severe before gradually subsiding as the inflammation reduces, though episodes can continue up to 18 months following the initial infection. Vestibular neuritis and labyrinthitis are typically unilateral in nature.

Vestibular labyrinthitis occurs when the infection is in the vestibular labyrinth and affects both branches of the VIIIth nerve. For this reason patients typically have the same symptoms as neuritis as well as tinnitus and hearing loss (Brill, 1982). Similar to neuritis, episodes occur suddenly before typically resolving as the infection is treated. Evoked potential testing is one of the best tools a clinician has when diagnosing patients with neuritis/labyrinthitis. Patients typically presented with absent or reduced oVEMPs for the affected side, while the non-affected side showed normal results (Aw, Fetter, Cremer, Karlberg, Halmagyi, 2001). In the same study the researchers found that patients with neuritis who had present VEMPs typically had BPPV as well (>50%), while none of those patients with absent VEMPS had BPPV (Aw, et al, 2001).

In a 2014 study by Magliulo, Gagliardi, Appiani, Iannella, and Re, the role of both oVEMPs and cVEMPs as well as the video head impulse test (vHIT) was assessed in diagnosing
vestibular labyrinthitis as well as determining the location of the insult to the vestibular nerve and assessing recovery from the labyrinthitis. The cVEMP test allowed clinicians to monitor the inferior vestibular nerve as well as saccular function, the oVEMP monitored utricular function as well as the superior vestibular nerve, and the vHIT measured the VOR and provided information on the semicircular canals and their nerves (Magliulo, Gagliardi, Appiani, Iannella, Re, 2014). Forty patients diagnosed with vestibular labyrinthitis were selected and given air-conducted cVEMPs, bone-conducted oVEMPs, and vHIT testing at the initial appointment, then at follow-up appointments at 10, 20, and 30 days then monthly until the patient recovered from the labyrinthitis. Results from the initial testing revealed abnormal oVEMPs in 32 out of 40 patients and abnormal cVEMPs in 19 out of 40. The results of vHIT testing revealed deficits in the horizontal and superior semicircular canals in 35 and 31 out of 40 patients respectively, but only 19 out of 40 for posterior semicircular canal deficiency. At the three month follow-up, testing revealed 13 patients had recovered and showed normal results on all tests; at the final follow-up only 27% of the subjects showed any abnormalities during testing. The use of both VEMPs as well as the vHIT allowed researchers to study the whole vestibular system and determine the exact location of the labyrinthitis, as well as provide a clinical means of tracking recovery from the attack.

Vestibular Schwannoma:

Vestibular schwannomas, also known as acoustic neuromas, are benign, unilateral, typically slow growing tumors that develop around the VIIIth nerve. The tumors form from an overproduction of Schwann cells, which are cells covering the nerve fibers. Symptoms of schwannomas include unilateral tinnitus and hearing loss, as well as dizziness. As the tumors grow and begin to press against the trigeminal nerve facial weakness or numbness may develop.
While MRI or CT scans are the best means of positively identifying schwannomas VEMP can be a useful tool to help with an initial diagnosis. Patients with schwannomas tend to have either absent or otherwise abnormal responses both oVEMP and cVEMP testing. VEMP amplitude was found to be reduced in the majority of patients with schwannomas, and latencies were found to be reduced, especially in larger sized tumors (Suzuki, et al, 2008).

In a more recent study done by Chiarovano, et al, the efficacy of VEMP testing to identify unilateral schwannomas was studied and the two different methods of VEMP testing were compared along with more traditional caloric test. The investigators performed tests on 63 patients with diagnosed unilateral schwannomas and compared the results they obtained with air versus bone conducted tone bursts for oVEMPs, as well as air conducted clicks versus tone bursts with cVEMPs (Chiarovano, Darlington, Vidal, Lamas, de Waele, 2014). Results from the study found that 71% of the subjects displayed abnormal caloric results. For cVEMP testing, tone-bursts were found to be more sensitive with 65% having an abnormal VEMP versus only 49% with the use of clicks. With oVEMPs only 59% of subjects were initially found to have abnormal responses through the use of air conduction tone bursts. Of the 26 remaining subjects the researchers found that bone conduction tone bursts delivered directly to the mastoid were abnormal in 89% of the subjects. Ultimately the researchers found that VEMP testing, particularly oVEMP testing, was of significant clinical value when investigating a possible vestibular schwannoma, just as Suzuki, et al (2008) stated previously.
**Perilymphatic Fistula:**

A perilymphatic fistula is a small tear in one of the two windows that divides the middle ear from the inner ear, either the oval or round window. When this tear is present, perilymphatic fluid from the inner ear can leak into the typically air filled middle ear space. Because the boundary between the two spaces has been compromised, changes in air pressure in the middle ear which once had no effect on the inner ear instead can disrupt the hearing and balance system (Maitland, 2001). Symptoms of perilymphatic fistulas include ear pressure, fluctuating hearing loss, and dizziness typically without true vertigo. Because of the susceptibility to air pressure, patients with fistulas often find their symptoms worsening with changes in atmosphere such as when flying. The primary cause of fistulas is head or ear trauma, though rapid altitude descent or barotraumas from diving can also cause them to develop. Diagnosing a perilymphatic fistula can be difficult as they are often extremely small and nearly impossible to physically see. Case history along with audiogram and tympanometry is often used to try and diagnose a possible fistula.

Recently cVEMPs have begun to be used to help diagnose potential fistulas. In a study by Modugno, Magnani, Brandolini, Savastio, and Pirodda in 2006, they found a number of patients with VEMP thresholds typically found in patients with semi-circular canal dehiscence. That is, these patients had lower than normal thresholds with large amplitudes; however magnetic resonant imaging failed to show any dehiscence in the bony structure. Based on these findings as well as the patient’s symptoms, the study concluded that reduced VEMP thresholds with negative imaging are likely due to a perilymphatic fistula (Modugno, Magnani, Brandolini, Savastio, Pirodda, 2006). Treatment of fistulas is typically a wait and see approach; however if
symptoms persist then the physician may decide on a surgical approach, where a patch is placed over the affected window to seal off the tear.

**Semicircular Canal Dehiscence:**

The last vestibular disorder to be discussed is the one most commonly associated with VEMP testing for clinical diagnosis, semicircular canal dehiscence. Semicircular canal dehiscence is a condition where this is a thinning or opening in the bony structure of the wall of one of the semicircular canals, most commonly the superior semicircular canal. This hole in the bony wall can lead to both vestibular and hearing disruptions in sufferers. Symptoms typically include vertigo and oscillopsia, a visual condition where known stationary objects appear to move. Another symptom is autophonia, a condition where the resonance of one’s own voice is increased, along with a conductive loss that is seen during audiometric testing (Minor, Solomon, Zinreich, Zee, 1998). Loud noises can trigger or aggravate these symptoms, along with movements that increase pressure in the head, such as coughing or sneezing.

The exact causes of superior semicircular canal dehiscence are not completely known but it is commonly believed to have a developmental pathology that is a genetic predisposition to developing a dehiscence (Neisten, et al, 2013). In a small study done by Neisten, et al (2013), three families were observed, each with two members who presented with symptoms similar to SSCD. Audiometric and imaging results were similar amongst the family members, with symptoms being more pronounced among the elder members of the family. The authors concluded that genetics likely plays a role in the development of SSCD. This correlates with an earlier study by Carey, Minor, and Nager in 2000 that looked at the prevalence of true dehiscence and thin bone in the superior semicircular canal in the population. A post-mortem
study of 1000 temporal bones from 596 adults was conducted, with 108 random subjects being measured for bone thickness. Results from the study found five subjects with complete dehiscence with another fourteen subjects displaying significant thinning of the bone in the posterior semicircular canal (Carey, et al, 2000). The researchers therefore hypothesized that the dehiscence and thin bone structure may be the result of incomplete postnatal bone development.

Posterior SSCD is often initially diagnosed through the use of VEMP testing with follow up imaging done to verify the presence of true dehiscence. Indeed VEMPs have become the gold standard method of testing to determine the presence of dehiscence before resorting to more expensive imaging tests. When a true SSCD is present, there are very specific VEMP characteristics seen by the clinician. Specifically the threshold of the VEMP will be greatly reduced and the amplitude of the response will be magnified. In a typical VEMP seen in a healthy subject, measureable wave responses will diminish and eventually disappear around 85 dBnHL; however in subjects with SSCD thresholds are often found at 75 dB nHL (Zuniga, Janky, Nguyen, Welgampola, Carey, 2012). In addition to reduced thresholds, peak to peak amplitude has been found to be larger in ears with SSCD than normal (>25 microvolts). What Zuniga et al (2012) found during their study was that while both types of VEMPs had the same characteristics when measuring cases of SSCD, oVEMPs had much greater sensitivity and specificity than cVEMPs (90% vs. 80%) and were therefore the more reliable test for determining the presence of SSCD.
Clinical Vestibular Tests

While the oVEMP and cVEMP are the primary focus of this capstone it is important to briefly touch on the other primary tests commonly used as part of the vestibular diagnostic test battery. Tests such as rotational chair and videonystagmography (VNG) record and evaluate both ocular and inner ear responses to stimuli to help determine whether the vestibular deficit is central or peripheral, that is cerebellar or inner ear disorder (Roeser, 2013). Posturography testing allows the clinician to investigate all three sensory modalities to further help with differential diagnosis. More recently the vHIT has been introduced to assess a patient’s VOR and provide information on the semicircular canals, as demonstrated in the study by Magliulo, et al. Another electrophysiological test that is used to test for Meniere’s disease is the electrocochleography (ECochG) test.

Videonystagmography:

The primary component of the VNG test is bithermal caloric testing, a procedure where the ear is cooled and warmed, either through air or water being presented for approximately one minute in duration. The warming or cooling of the ear excites or inhibits the fluid in the inner ear triggering the body’s VOR. Bithermal caloric irrigation allows the clinician to measure caloric hyper- and hypo-sensitivity, asymmetry and directional preponderance of nystagmus for both ears (Arriaga, Chen, Cenci, 2005). While caloric testing is the primary test of the VNG, a full test battery also includes oculomotor testing such as saccades, tracking and smooth pursuit, positioning testing, positional testing for BPPV, as well as visual fixation and suppression tests for spontaneous nystagmus. A complete battery of rotational chair testing also includes oculomotor and visual fixation tests, in addition to greater assessments of the VOR.
**Rotary Chair:**

Rotational chair testing studies the VOR through a wider range of frequencies, .01 Hz to .64 Hz whereas caloric testing only corresponds to .005 Hz, a very low frequency. Another test that both the VNG and rotary chair utilize is the optokinetic test, which measures a patient’s visual-vestibular interaction. In a retrospective study done by Arriaga, et al (2005), the sensitivity of VNG testing was compared to rotary testing in order to judge which should be the preferred test method for clinicians. Initially looking at 1000 patients who had undergone evaluation for dizziness with rotational chair testing as the primary test, the researchers examined a group of 478 patients who had undergone both rotary chair and VNG testing. Of that group, they identified the patients who were diagnosed with a peripheral disorder and found that the sensitivity for rotary chair was much higher than VNG (71% vs. 31%) but the specificity was much greater for VNG (86% vs. 54%). They concluded that due to the higher sensitivity rate rotary chair testing should be the primary diagnostic tool with VNG testing used as a supplemental test to help determine specificity.

**Computerized Dynamic Posturography and vHIT:**

Computerized Dynamic Posturography (CDP) is a more recently developed test that assesses a patient’s balance rather than determining if a vestibular disorder is central or peripheral (Furman, 1994). By measuring the input from of a patient's visual, vestibular, and proprioceptive systems, CDP allows the clinician to determine where a deficiency may lie and how a patient is compensating for the weakness in his/her balance, which in turns provides some input into how physical therapy may benefit the patient with improving his/her overall balance. As mentioned previously, the vHIT is a clinical test used to detect semicircular canal
dysfunction. A video camera records eye movement as the head is rotated quickly in order to stimulate the semicircular canals. Initially it was thought the vHIT was only capable of detecting horizontal canal dysfunction with lateral movement; however a study by MacDougall, McGarvie, Halmagyi, Curthoys, and Weber (2013), utilized vertical head thrusts to detect dysfunction in the vertical semicircular canals. Recordings from healthy individuals were compared alongside subjects with identified unilateral, bilateral, and individual semicircular canal dysfunction utilizing both the vHIT and invasive scleral search coils which are inserted into the eye (MacDougall, et al, 2013). Vertical movement was oriented along the left-anterior-right-posterior (LARP) canals and the right-anterior-left-posterior (RALP) canals with the mean VOR difference between the two methods compared. The study found that the mean difference between the two methods was virtually identical, thus demonstrating that vHIT could be used to test for both horizontal and vertical semicircular canal dysfunction.

**Electrocochleography:**

Electrocochleography (ECogH) is an electrophysiological test with multiple functions, but in a vestibular clinic it is used primarily to diagnose Meniere's disease. ECogH involves the recording of the cochlear mechanoelectrical potentials in response to a sound source; ECogH testing registers all three of these mechanoelectrical potentials: the cochlear microphonic (CM), the summing potential (SM) and the action potential (AP) (Hall, 2006). The CM is generated at the most basal turn of the cochlea from outer hair cells, whereas the SP is believed by most researchers to be generated by the inner hair cells in response to acoustic stimulation. Others believe that both inner and outer hair cells are responsible for the SP (Lamounier, Gobbo, de Souza, de Oliveira, Bahmad, 2014). Lastly the AP is generated typically by click stimuli and represents neural activity in the cochlea. When used to identify Meniere's disease in subjects
only the SP and AP are considered; it is the SP/AP ratio that is examined. The amplitude of the first positive peak of the AP and the baseline is measured against the second positive peak of the AP and the baseline (Lamounier, et al 2014). Researchers have found that expanded SP/AP ratios greater than .40 are a strong indicator of endolymphatic hydrops and thus a good diagnostic tool for identifying Meniere’s disease in patients (Takeda, Kakigi, 2010).

**Cervical and Ocular VEMPs**

In the early 1990’s new electrophysiological methods were found to be an effective method of testing saccular and the inferior vestibular neural pathway. These tests, the cervical vestibular evoked myogenic potential (cVEMP), were described by Colebatch and Halmagyi in 1994 as a myogenic response to air and bone conducted sound triggered in the otolithic afferent neurons. The reason that VEMPs are called myogenic responses is because rather than measuring a neural response from the vestibular organs, electrodes are placed on the sternocleidomastoid (SCM) muscle and measure electrical response of the muscle (Rosengren, Welgampola, Colebatch, 2010). Muscular responses induced by sound are known as sonomotor responses and have been noted and studied as far back as Tullio in the early 20th century (McCaslin, 2014).

With cVEMPs, a sonomotor response from the SCM is measured after sound is delivered to the subject, typically high intensity air or bone conducted clicks though tonebursts at other frequencies have also been used clinically. The SCM is part of a group of muscles that reacts to sound, alongside the triceps, trapezius, and the quadriceps among others (Halmagyi, G, Colebatch, J, Curthoys, I, 1994). While the muscle itself responds to sound it requires tonic activity of that muscle to measure a cVEMP; without that activity the potential will be absent.
Typically patients activate their SCM by lifting and turning their head the opposite direction of the ear that is being tested in order to create tension in the muscle. While maintaining tonic activation of the SCM may be difficult for some patients, the test itself is relatively brief. Typically only 100-300 sweeps, or stimulus repetitions are needed to record a cVEMP (Rosengren, et al 2010), which typically lasts less than one minute. Unlike Auditory Brainstem Response (ABR) testing, waveform latency is not the primary component used for diagnostic interpretation. Waveform amplitude is used to measure cVEMPs, specifically the distances of the P1 to N1 wave points. Low VEMP thresholds or asymmetrical ratios may be indicators of disorders in the vestibular system.

Only recently has a new electrophysiological test which examines utricular and the superior vestibular neural pathway been introduced into the vestibular clinical battery. Ocular vestibular evoked myogenic potentials (oVEMP) record myogenic activity from the inferior oblique muscles in response to air and bone conducted sound (Kanter, Gurkov, 2012). The originators of the oVEMP potentials are considered by most researchers to be the utricle and the superior vestibular nerve as previously noted (Chiarovno, et al 2011). Just as the SCM must be active to record cVEMPs, so must the inferior oblique muscles be tonic when recording oVEMPs. When the sound stimulus is presented to the subjects they are tasked with looking directly up with their eyes, thus activating the inferior oblique muscles. Because the oVEMP utilizes the VOR, the response recorded is from the contralateral inferior oblique muscle (Kanter, Gurkov, 2012).

Similar to cVEMPs, wave point amplitude is used to determine if an oVEMP is normal, irregular, absent, or low threshold. The n1 wave point and the p1 wave point are the two points of reference and interaural amplitude is used to compare left and right (Piker, Jacobson,
If the amplitude asymmetry ratio is greater than 35% then the results are considered to be abnormal and indicative of likely utricular dysfunction, with the side with reduced amplitude having the deficiency (Murofushi, Kaga, 2009).

Another measure of dysfunction for both types of VEMP tests is threshold present; defined as the lowest level of presentation of the stimulus used to elicit the VEMP. In healthy subjects VEMP responses are elicited at presentation levels typically between 85-95 dBnHL, if a VEMP response is present below 70 dBnHL it is considered pathological and demonstrates an over-sensitivity of the utricle or saccule to sound (Colebatch, et al 1998).

While the cVEMP is elicited from the large SCM, oVEMPs rely on tonic activation from the much smaller inferior oblique (IO) muscles. Because of the smaller size of the muscle and the response, proper activation of the IO’s is a must to elicit a good response. In a study by Rosengren, et al, the effect of gaze elevation on oVEMP response was studied. The researchers conducted oVEMPs on ten subjects but varied the test protocol; electrode placement was shifted throughout the trials and eye gaze elevation was shifted from an upward position throughout to a downward gaze. Gaze elevation was begun at 24 degrees upwards through neutral (0 degrees) to a low elevation of 24 degrees downward (Rosengren, Colebatch, Straumann, Weber, 2013). The researchers found that while oVEMP amplitude did decrease somewhat as the electrodes were placed further away from directly underneath the eyes, the decrease in oVEMP amplitude was much more pronounced as gaze elevation decreased. This evidence lends credence to the thought that tonic muscle activity is of vital importance in oVEMP amplitude.
Protocol

The major objective of this paper is to share the most recent VEMP procedures with the readers. The following section outlines suggestions for best practice in clinicians’ performance of cVEMP and oVEMP testing.

**cVEMP Protocol:**

Cervical VEMPS are myogenic responses which represent the Vestibulocollic reflex and are believed to originate from the saccule (Halmagyi, Cremer, 2000). The afferent pathway of the response is from the saccule through the inferior vestibular nerve through the medial vestibulospinal tract through the accessory nerve to the ipsilateral SCM (Kushiro, Zakir, Ogawa, Sato, Uchino, 1999).

**Electrode Montage:**

The patient should be supine prior to testing. Upon stimulus presentation, the patient should raise his/her head up approximately 30 degrees (Rosengren, et al, 2010). If the patient is unable to maintain this position throughout the duration of the stimulus, it is recommended that the patient sit and lift then turn his/her head away from the ear being tested (Isaacson, Murphy, Cohen, 2006).

**Recording electrode/Non-inverting electrode:** This should be placed on the ipsilateral SCM, at approximately the middle third of the muscle. Please see examples of electrode placement in Figures 1 & 2.

**Inverting electrode:** This should be placed on the chin. If the patient has a beard, place the electrode on the chest, just above the sternum.
Ground electrode: The ground electrode is placed at Fpz which is midline on the forehead where the frontal and parietal bones meet.

Impedance should be less than 7 Ohms before testing (Wang, et al, 2014).

**Recording Parameters:**

Transducer: ER3a insert earphones are typically used.


Bandpass filter: 20-2000 Hz (Murofushi, Kaga, 2009)

Stimulus: 500 Hz tone bursts are commonly used (Murofushi, Kaga, 2009); other frequencies and click stimuli may be used as well.

Stimulus repetitions: 100-300 (Rosengren, et al, 2010)

Polarity: Rarefaction

Rate: 5 per second (McCaslin, 2014)

Analysis time: 20-100 ms (Welgampola, Colebatch, 2001)

**cVEMP Responses:**

In healthy patients, cVEMPs will be present and are marked by a positive wave (p1) that occurs at approximately 13ms and a negative wave (n1) that occurs at approximately 23ms (Murofushi, Kaga, 2009). An example of a cVEMP tracing may be seen in Figure 3. Thresholds for normal cVEMPs are between 80-90 dBnHL; anything lower is considered to be an abnormal test result (Murofushi, Kaga, 2009).
Assessment Parameters:

Amplitude is primarily used to measure cVEMP responses and determine if they are pathological or not. Amplitude is found by taking the peak distance of p1 to n1, this gives the amplitude for both ears, Amplitude Right and Amplitude Left (AR, AL). The interaural difference between the two sides is known as the asymmetry ratio and is used to determine if either ear is dysfunctional. Inter-aural amplitude is determined by using the formula absolute value of AR-AL/AR+AL x 100%. If the ratios are less than 35% then they are considered normal (Rosengren, et al, 2010); however, other studies report that less than 40% can be considered normal (McCaslin, et al, 2013). The side with the reduced amplitude is considered to be the dysfunctional ear (Murofushi, Kaga, 2009). It must be noted that patients over 60 years of age often have reduced or absent cVEMP responses; these results cannot be considered pathological (Su, Huang, Young, 2004).

oVEMP Protocol:

Ocular VEMPS are myogenic responses representing the vestibuloocular reflex (VOR). Whereas the cVEMP represents saccular function, the oVEMP is largely believed to represent function of the utricle and the superior vestibular nerve (Chiarovno, Darlington, Vidal, Lamas, de Waele, 2011). Because the oVEMP is a measure of the VOR, the contralateral ocular muscles are used to record the potential.

Electrode Montage:

The patient should be seated in a chair. Upon stimulus presentation the patient should raise his/her eyes up approximately 30 degrees (Park, Lee, Shin, Lee, Park, 2010). A bi-polar montage is the most commonly used during testing, as described below.
**Active electrode:** This should be placed at the contralateral orbital margin, approximately 1 cm below the eye and centered (Chihara, Iwasaki, Ushio, Murofushi, 2007).

**Reference electrode:** This should be placed on the chin. If the patient has a beard, place the electrode on the chest, just above the sternum.

**Ground electrode:** The ground electrode is placed at Fpz which is midline on the forehead where the frontal and parietal bones meet. Please see an example of the electrode placement in Figure 4.

Impedance should be less than 7 Ohms before testing (Wang, et al, 2014).

**Recording Parameters:** Recording parameters are listed below.

**Transducer:** ER3a insert earphones are typically used.

**Amplifier:** Because the electrical response elicited from an oVEMP is so small it requires much greater amplification of the signal than the cVEMP; 50,000-100,000 x amplification is typically used. (Kantner, Gurkov, 2012)

**Bandpass filter:** 10-1000 Hz (Kantner, Gurkov, 2012)

**Stimulus:** 500 Hz tone burst (Murofushi, Kaga, 2009) or 1000 Hz tone burst (Lewis, et al, 2010) are the most commonly used stimuli; however, clicks may be used instead of tone bursts.

**Stimulus repetitions:** 100-200 (Rosengren, et al, 2010)

**Polarity:** Rarefaction

**Rate:** 5 per second (McCaslin, 2014)
Analysis time: 50 ms

**oVEMP Responses:**

In healthy patients, oVEMPs will be present and are marked by a waveform with an initial negative peak (n1) whose latency occurs at approximately 10ms and a positive peak (p1) that occurs approximately 15ms (McCaslin, 2014). An example of oVEMP tracing may be seen in Figure 5. Thresholds for normal oVEMPs are typically between 80-90 dBnHL. Lower findings are considered to be an abnormal test result (Murofushi, Kaga, 2009).

**Assessment Parameters:**

Like cVEMPs, amplitude is the primary means used to measure oVEMP responses and determine if they are healthy or not. Amplitude is found by taking the peak distance of p1 to n1, this gives the amplitude for both ears (AR, AL). The interaural difference between the two sides is known as the asymmetry ratio and is used to determine if either ear is dysfunctional. The same formula used for cVEMPs is used to determine interaural amplitude: AR-AL/AR+AL x 100%. If the ratios are less than 35% then they are considered normal (Rosengren, et al, 2010); however, other studies have reported that less than 40% can be considered normal (McCaslin, 2014). The side with the reduced amplitude is considered to be the dysfunctional ear (Murofushi, Kaga, 2009). Age has the same effect on oVEMPs as it does for cVEMPs. Patients over 60 years of age often have reduced or absent cVEMP responses; these results cannot be considered pathological (Su, et al, 2004).
Discussion

Because VEMPs are a myogenic potential, they require activation of target muscles to elicit a response. This can be one of the great difficulties faced by the clinician when attempting to record VEMPs. Too much muscle activation can lead to increased artifact and interfere with the recordings. Too little activation may result in the VEMP not being present. While the supine position is recommended by a number of researchers as the best position to obtain cVEMPs, it is not always the most feasible. Some elderly patients may have difficulty lifting their heads up for the duration of the test. For this reason, as well as to maximize space in smaller independent clinics a reclining chair can be utilized. This method also prevents the patient from having to move positions during oVEMP and cVEMP testing; rather they can just remain in the same chair throughout.

Another potential issue facing the clinician when performing VEMP testing comes with those patients who have a conductive hearing loss. While a sensorineural hearing loss has no significant effect on the VEMP tests a conductive component does. In a study by Mahdi, et al VEMP testing was done on patients with normal hearing, sensorineural hearing loss, conductive loss, and vestibular schwannomas (Mahdi, Amali, Pourbakht, Yazdi, Bassam, 2013). The researchers found that in patients with normal hearing and those with a sensorineural hearing loss, VEMP responses were present in all subjects. Of the 32 patients with a conductive hearing loss VEMPs were present via air conduction in only 17; however when VEMPs were done via bone conduction VEMPs were present in all 32 subjects. Therefore, while bone conducted VEMPs may not be commonly used they can be extremely helpful tools when testing patients with conductive hearing losses.
VEMPs have been demonstrated to have a number of uses in a vestibular diagnostic clinic. There still remains more research to be done on the VEMPs, particularly oVEMPs as they are relatively newer. Many clinics still require normative data to be gathered before beginning oVEMP testing (Rosengren, et al, 2010). Another issue that needs to be resolved is how to properly measure myogenic activity while recording VEMPs, too much or too little muscle activation will affect the VEMP responses and can give false positive or false negative responses (McCaslin, 2014). This deficiency arises in part from the fact that many clinics use equipment that was originally designed to test other evoked potentials, such as ABR’s, but has now been modified to conduct VEMP testing. Lastly, while VEMP studies have looked at the efficacy of VEMP testing on a small scale, there have yet to be any large scale studies of VEMP testing to evaluate common vestibular disorders, (Rosengren, et al, 2010).

**Conclusion**

Vestibular disorders are some of the most difficult cases that audiologists face in a clinical setting. The clinician is faced with not only multiple symptoms and multiple causes of vestibular disorder, but multiple diagnostic tests as well. Tests such as rotary chair, CDP, and VNG often involve expensive equipment and require separate office space, creating a logistical problem for many practitioners. With the introduction of VEMP testing, audiologists now have a rather cost effective means of studying the vestibular system. VEMP testing can typically be done with the same equipment used for auditory brainstem response testing (ABR); therefore, the only equipment typically needed is a laptop, electrodes, and a recliner or table. With the introduction of oVEMPs, audiologists now have the ability to study both the utricle and saccule in an effort to determine site of lesion for vestibular dysfunction. Although VEMP test results
are not reliable for some vestibular disorders, their clinical benefit makes them an important tool for any vestibular diagnostic test battery.
References


Figures and Tables

Figure 1. *cVEMP electrode montage*
Figure 2. *cVEMP electrode montage*
Figure 3. Normal cVEMP response
Figure 4. oVEMP electrode montage
Figure 5. Normal oVEMP response