INSIGHTS in practice

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Vestibular Evoked Myogenic Potentials in Deaf and Hard of Hearing Subjects

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Abstract

Preliminary findings conducted at the Gallaudet Hearing and Balance Center corroborate clinical findings reported in the literature indicating that in subjects with vertigo or imbalance or history of these disorders vestibular evoked myogenic potentials (VEMPs) tend to be abnormal with respect to amplitude, latency and/or augmented calculations. Further, the findings suggest that subjects with vertigo or imbalance may be tested accurately and without discomfort using this procedure unlike other traditional test methods. However, there is a dearth of literature regarding clinical application of this procedure to deaf and hard of hearing subjects. The test is a measure of the saccule, inferior vestibular nerve branch of cranial nerve VIII, medulla, and neck muscles and as such are independent of hearing. Although loud acoustic signals are used to generate a response, the signal produces a pressure wave which initiates hydrodynamic stimulation of vestibular hair cells. Clinical applications of VEMP indicate that this test is useful in assessing balance function in Deaf/HH patients.

Background

Testing hearing of deaf and hard of hearing (D/HH) patients is routine and well established. There are nearly 150 discreet hearing tests which assess different aspects of the hearing mechanism. This variety of procedures assures generally accurate identification of the site of lesion of a hearing loss, which is essential if suitable treatment or medical management is to be accomplished. Determining precise configuration of the hearing loss is essential for suitable fitting of a hearing aid, but identifying possible etiology of the damage is equally critical. For example, hearing loss caused by an acoustic nerve tumor may be rehabilitated temporarily with amplification, but overlooking the cause of the loss in this instance could jeopardize future hearing, health and possibly even the life of the patient; tumors of this type can become fatal when they grow unchecked into the brainstem. In addition, an acoustic nerve tumor often affects balance, and assessing balance electrophysiologically may improve diagnostic outcome over traditional testing for tumor. Vestibular evoked myogenic potential (VEMP) testing provides this needed electrophysiological assessment tool of balance.

Approximately two-thirds of the inner ear is dedicated to vestibular function, while the other third is dedicated to the cochlea for hearing. Although inner ear balance is not essential for survival in humans as it may have been in primitive man and appears to be in other animals, balance disorders can inconvenience or handicap individuals. For example, athletes and construction workers rely on precisely coordinated balance movements. A damaged vestibular system can at least lessen the quality of life for these individuals and may even diminish their livelihood.

Only a few procedures have been developed to assess balance. The most common technique is the electronystagmography (ENG) or the more current videonystagmography (VNG) procedure. This test involves several subtests which categorize balance disorders according to: (1) central problem such as tumor or neurological disease, (2) positional disorder such as benign paroxysmal positional vertigo, and (3) inner ear disorder. The series of tests takes about an hour and an occasional result is nausea and vomiting. Unfortunately, the procedure often fails to identify the locus of the disorder or even the existence of a disorder despite the extreme discomfort the patient must usually endure.

A second balance testing procedure, used to a limited extent in the U.S., is known as Vestibular Evoked Myogenic Potential (VEMP) measurement. This procedure is designed to assess balance organ hair cells and neural linkages in the same way that the auditory brainstem response (ABR) test assesses cochlear hair cells and their neural links. Response characteristics of the VEMP electrophysiological measure are fairly well documented with respect to amplitude

continues

and latency in subjects and patients with normal hearing. However, because the test uses an intensive acoustic signal ('click' or tone burst) to stimulate a balance organ, responses from D/HH subjects may have characteristics that differ in subtle ways from subjects with normal cochlear function. Identifying and 'standardizing' the possible amplitude or latency characteristics unique to D/HH subjects is essential if the procedure is to have generalized clinical application to these patients.

Description of the Vestibular Evoked Myogenic Potential (VEMP) Procedure

The VEMP procedure is an emerging diagnostic tool for identifying vestibular lesions. At the Gallaudet Hearing and Balance Center, the VNG procedure is available but has been replaced for some patients by the better tolerated VEMP test. The VEMP procedure is non-invasive and causes little or no discomfort to the patient, unlike the caloric irrigation component of the electronystagmography (ENG) or VNG procedure. In addition, the test contributes an assessment dimension to the well-established VNG protocol. While caloric irrigation testing is focal to the lateral semicircular canal (LSC) and neural links to eye muscles, VEMP testing targets the vestibule and neural connections to the sternocleidomastoid muscles (SCM) of the neck. The VNG procedures evaluate semicircular canals and neural input to pons and cerebellar structures, while the VEMP procedure stimulates the saccule and connections through the medulla and into the neck. The procedures are complementary in that VEMP testing completes the lower brainstem and upper spinal column assessment component not targeted with VNG procedure. VEMP tests linear acceleration/gravitational orientation balance function, while VNG tests angular acceleration and central balance capability. Further, as noted, VEMP testing is tolerated well by the patient making it a useful adjunct procedure to VNG in a single test session.

Patients with true vertigo (hallucination of motion or the feeling of spinning), or a less specific feeling of imbalance or lightheadedness are routinely evaluated with a complete audiological evaluation and VNG testing. The most objective of the VNG subtests, the caloric irrigation procedure, grossly measures response of the LSC but does not assess the function of other vestibular sensory structures: namely, superior and posterior semicircular canals, saccule, and utricle (Akin and Murnane, 2001). Other procedures in the VNG sequence may identify posterior semicircular canal disorder (Dix-Hallpike Maneuver) or central lesion (direction changing nystagmus with positional change). However, often VNG findings are inconclusive or 'non-localizing' and do not always specify a locus of lesion. By including VEMP procedures in the clinical work-up of patients with imbalance, a more thorough assessment of the vestibular mechanism is accomplished, and in addition a specific vestibular sensory system is targeted.

The VEMP by definition is a short-latency electromyogram recorded from the tonically contracted SCM in response to high-intensity acoustic stimulation (Bickford et al., 1964; Cody and Bickford, 1969; Colebatch et al., 1994). With electrodes placed on the SCM muscles, waves occur at approximately 13-15 ms (p13 or wave I) and 21-24 ms (n23 or wave II) post stimulus delivery to the ear ipsilateral to the contracted SCM. The VEMP neural pathway consists of the saccule, inferior vestibular nerve, and vestibulospinal tract (Colebatch and Halmagyi, 1992; Itoh et al., 2001; Murofushi et al., 1996a; Murofushi et al., 2001).

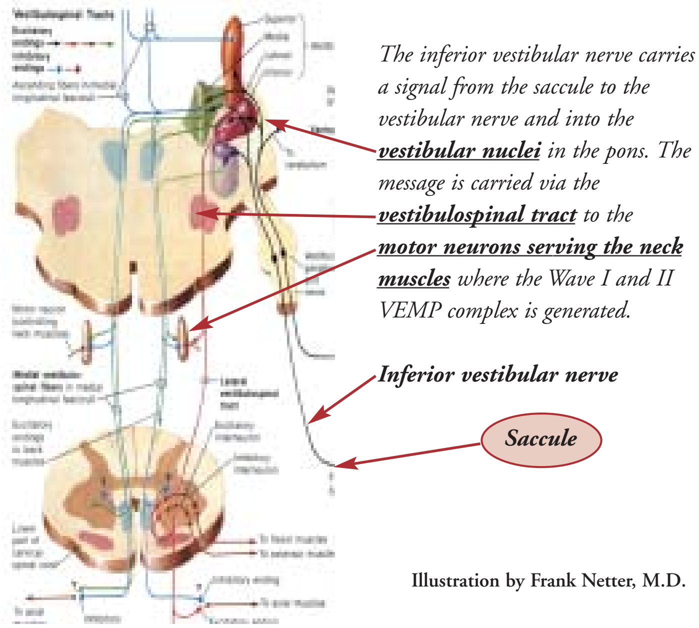


Fig. 1. *The neural route for the VEMP response is illustrated here.*

More evidence of saccular stimulation is shown with experiments on guinea pigs and cats. Loud acoustic transients are shown to stimulate saccular afferents and are recorded electrophysiologically (Didier and Cazals, 1989; McCue and Guinan, 1994; Murofushi et al., 1995, 1996b; Murofushi and Curthoys, 1997). Involvement of saccular tracts as well as the lower brainstem in normal hearing subjects are verified in additional reports (Townsend and Cody, 1971; Cazals et al., 1987; McCue and Guinan, 1994; Halmagyi and Colebatch, 1995; Robertson and Ireland, 1995; Colebatch et al., 1994; Itoh et al., 2001).

Clinical Applications

The VEMP gives important information about saccular and inferior vestibular nerve functions, which supplements caloric assessment of lateral semicircular canals, as well as other components of the VNG/ENG procedure that target non-saccular vestibular systems. Recently, a mathematical calculation of VEMP amplitude was proposed (Young, Wu & Wu, 2002). Finding the difference in amplitude between the two sides divided by the sum of the amplitudes gives a ratio. When the ratio exceeds .36 the result indicates 'distended saccule' or saccular hydrops. This condition of excessive endolymph traditionally defines Meniere's Disease. This calculation is applied in all cases when the VEMP amplitudes between the ears appears to be significant. A case demonstrating this application is presented below.

Another recent discovery in the application of VEMP procedure clinically is identification of an inner ear pathology known as 'superior canal dehiscence' (SCD), or thinning or opening of the bony superior semicircular canal of the inner ear (Minor, 2000; Streubel, et al., 2001). Other studies report VEMP sensitivity to acoustic nerve tumor (Murofushi, et al., 2001), Meniere's Disease (Murofushi, et al., 2001; Shojaku, et al., 2001), brainstem stroke (Itoh, et al., 2001), and multiple sclerosis (Versino, et al., 2002).

It is reported that VEMP is mediated by an ipsilateral pathway. That is, VEMPs are recorded when the stimulus ear and contracted SCM are on the same side (Akin and Murnane, 2001; Halmagyi and Curthoys, 2000). This suggests that the auditory stimulation to the ear contralateral to the measured SCM muscle may not affect myogenic responses, and therefore that the presence of contralateral noise may not interfere with the recording of VEMP. However, a recent report by Takegoshi and Murofushi (2003) shows a change in VEMP with contralateral noise levels of 75 and 95 dB nHL. They studied 10 normal subjects and 10 patients with hemifacial palsy. Masking levels of 75 dB and 95 dB HL significantly (p<.05) reduced VEMP amplitude. Since the effect was greater in normal subjects than in patients with hemifacial palsy, they concluded the amplitude reduction to be due to stapedius muscle reflex that reduces conduction of the stimulus to the inner ear. The finding of masking effects on VEMP is consistent with clinical observations and also noted by Gallaudet researchers (Tamaki and Ackley, 2003).

Basic clinical procedures used to acquire reliable VEMP recordings are described in the previous GN Otometrics 'Insights in Practice' (Akin and Murnane, 2004). This standard protocol and instrumentation are applied in assessing the following subjects tested at the Gallaudet Balance Center.

Gallaudet VEMP Study

Subjects are deaf and hard of hearing volunteers attending Gallaudet University. Fifteen (15) subjects have been tested to date using the GN Otometrics CHARTR Evoked Potential equipment. Electrode montage is sternum (noninverting), upper belly of the sternocleidomastoid (inverting) and forehead (ground). EMG amplitude was not monitored. Rather, test-retest comparisons were used to verify SCM muscle tension consistency. Responses were filtered (20-2k Hz), amplified (10k gain) and 128 samples averaged. All waveforms were repeated. Signals were 500 Hz tone bursts (Hanning envelope) presented at a rate of 4.7 per second at 95 dB nHL.

Results of the preliminary findings show no significant wave latency differences when comparing D/HH to normal controls **(Fig. 2)**. However, evidence of amplitude differences between the two groups is suggested, although not statistically significant **(Fig. 3)**.

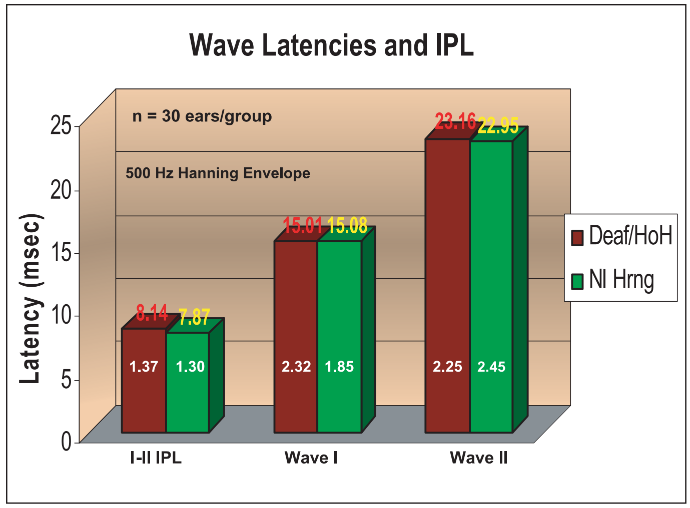


***Fig. 2**. Wave latencies for Deaf/HH subjects compared to subjects with normal hearing show no significant differences for Wave I and II latencies nor for the I-II interpeak latency (IPL). Latencies (msec) are given at the top of each bar and standard deviations are given within each bar. (Ackley and Tamaki, 2003.)*

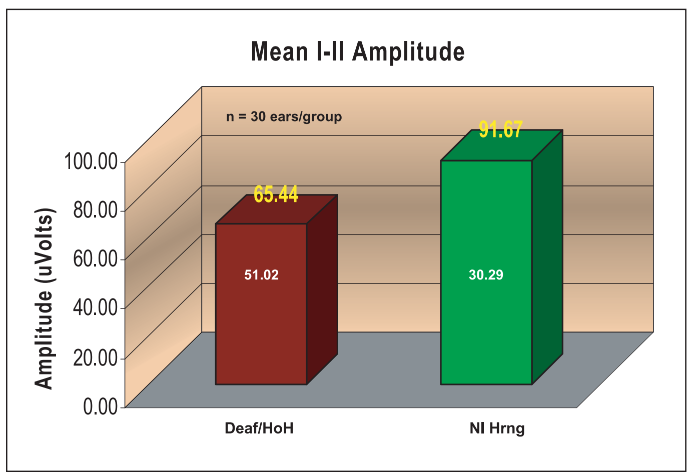


***Fig. 3.** Wave amplitudes for Deaf/HH subjects compared to subjects with normal hearing show notable differences for Wave I -II amplitude. Amplitudes (uV) are given at the top of each bar and standard deviations are given within each bar. Large standard deviations and small sample size influenced no statistical significance. (Ackley and Tamaki, 2003.)*

Several patients who are deaf or hard of hearing have been seen at the Gallaudet Balance Center for assessment or treatment. The patients are not included in the study, but some are described in the case studies section below which illustrate clinical application of VEMP when assessing D/HH patients.

Case Studies

Case 1. This is a 26 year-old student at Gallaudet who was diagnosed with Meniere's Disease. He reported symptoms of episodic vertigo and occasional buzzing tinnitus. His hearing loss was severe – profound since childhood. In addition, CT scans showed bilateral Enlarged Vestibular Aqueduct (EVA) syndrome and Mondini deformity **(Fig. 4)**. Such a finding suggests possible Pendred Syndrome, which has not yet been verified by DNA study.

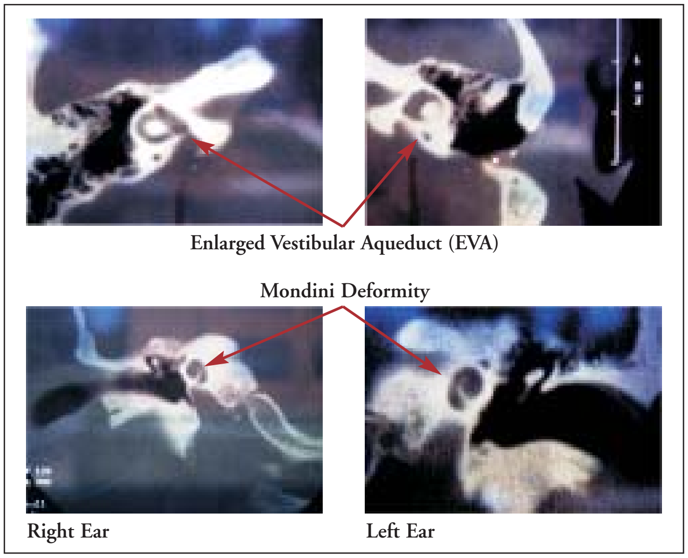
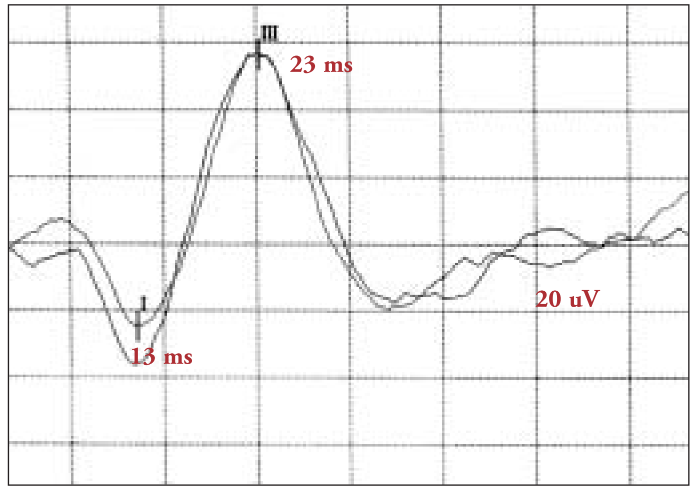


Fig. 4. *CT Scan of case #1 showing enlarged vestibular aqueduct (EVA) and Mondini deformity for right and left ears suggesting possibility of Pendred syndrome. This patient was diagnosed with Meniere's disease at age 26 and was managed with surgical repair of perilymphatic fistula. VEMP testing determined the affected ear.*

Determining the disordered ear is often difficult in deaf patients who may not identify tinnitus or hearing loss. Accurate balance testing is essential to find the damaged ear in order for surgical management of that ear to be successful. This patient had been seen by otolaryngologists who had made the diagnosis and was being seen in the Gallaudet Hearing Clinic for further testing and amplification. He was referred for VEMP testing, having refused ENG or VNG assessment because of prior unpleasant experiences with the procedure. Results showed the following:



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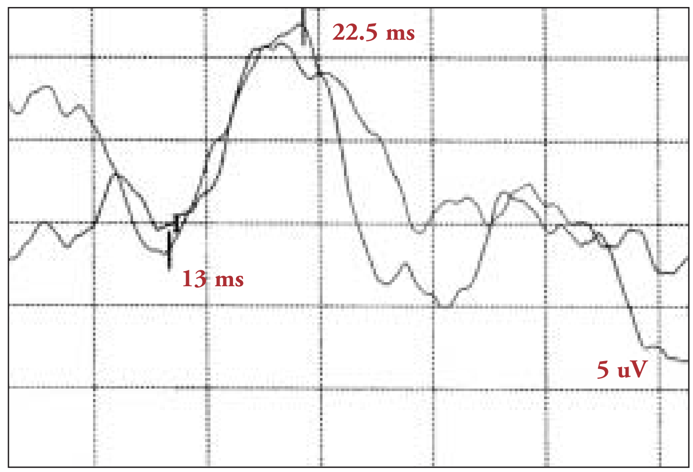


Left Side

Fig. 5. *This result shows a normal response for the right side with normal latencies and an amplitude of 110 uV. However the left side has an amplitude of only 12 uV identifying the left ear as the probable cause of the vertigo symptoms. Furthermore, calculation of the augmented VEMP (described in the text below) indicates saccular hydrops (left) consistent with a diagnosis of Meniere's Disease. (Note: The waveforms are 'reversed' because of the noninverting ('active') electrode placement on the sternum. The waves are then easily inverted using this function with the GN Otometrics EP software which would then display the waveforms more conventionally with wave I positive consistent with the 'P13' nomenclature common in the literature for this wave.)*

The findings indicated that the left saccule was damaged and because of the significant right vs. left amplitude difference in the responses, calculation of the "augmented VEMP" (Young, Wu & Wu, 2002) was done and found to be consistent with diagnosis of 'saccular hydrops'. The augmented VEMP calculation uses the following formula to determine a significant VEMP amplitude difference between ears: LA –SA/R+L > 0.36, where 'LA' and 'SA' are the large and small amplitude; 'R' and 'L' = right and left side amplitudes. When the resultant ratio exceeds 0.36, excessive endolymphatic volume in the saccule is suspected. In this case the ratio is: 110-12/110+12 = 0.80 suggesting saccular hydrops. This is consistent with the diagnosis of Meniere's Disease which involves a pathophysiological inner ear mechanism of 'endolymphatic hydrops', or distended endolymphatic compartment including the saccule. However, it should be noted that a possible cause of endolymphatic hydrops and therefore Meniere's symptoms is reportedly 'Perilymphatic Fistula' (Ackley, et al., 1994). The findings were reported to an otolaryngologist who confirmed a diagnosis of saccular hydrops secondary to perilymphatic fistula, and surgical repair of the fistula was accomplished. In sum, the VEMP procedure accurately identified a damaged saccule which was not recognizable to the patient nor to medical examination, which led to surgical management of the disorder.

Case 2. This is a 43 year-old profoundly deaf subject who was referred to the Balance Center because of a complaint of vertigo when she hears loud sound. This problem, although frequently mistakenly diagnosed as psychogenic, is known as 'Tullio Phenomenon' and is thought to be a condition affecting the saccule of the inner ear. In this case, the patient discovered that when she turned on her right hearing aid to full volume with loud noise in the environment, she became vertiginous. This was verified in the Balance Center after attempting to induce vertigo with the left hearing aid and failing. With the right hearing aid on but without noise in the environment, similarly no vertigo was induced. However, after 45 seconds of environmental noise including multi-talker loud conversation and music and amplified through her right hearing aid, vertigo resulted and was documented as nystagmus (horizontal, non-torsional) with videonystagmography. VEMP testing was performed **(Fig. 6)** with the following results:

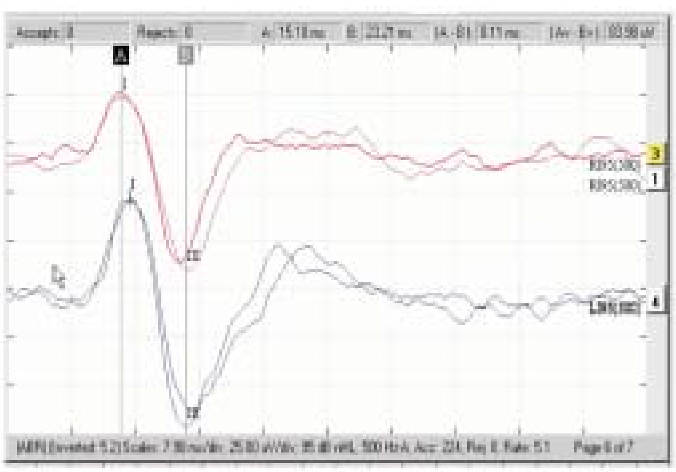


Fig. 6. *This VEMP tracing for right (top) and left (bottom) shows a symmetrical response with respect to wave latency (time delay of waves), but shows a reduced amplitude for the right side. Response amplitude for the right is 80 uV and for the left is 110 uV suggesting a disorder of the right saccule. Calculation of the 'augmented VEMP' (LA – SA/R + L >.36; where 'LA' and 'SA' are large and small amplitudes and 'R' and 'L' are 'right' and 'left' amplitudes) is not significant for saccular hydrops (Young, Wu & Wu, 2002). Further VEMP testing discovered a reduced VEMP threshold which may be consistent with SCD, but CT scans of the superior and posterior semicircular canals were negative for SCD. Follow-up of this patient by an otoneurologist is ongoing.*

Case 3. Perhaps this case best illustrates the importance of the Gallaudet study. This is a 24 year-old deaf subject with autosomal dominant deafness and a student at Gallaudet who reports no imbalance, vertigo or disequilibrium of any description **(Fig. 7)**. Her VEMP responses are very poor which would indicate a balance disorder in a subject with normal hearing.

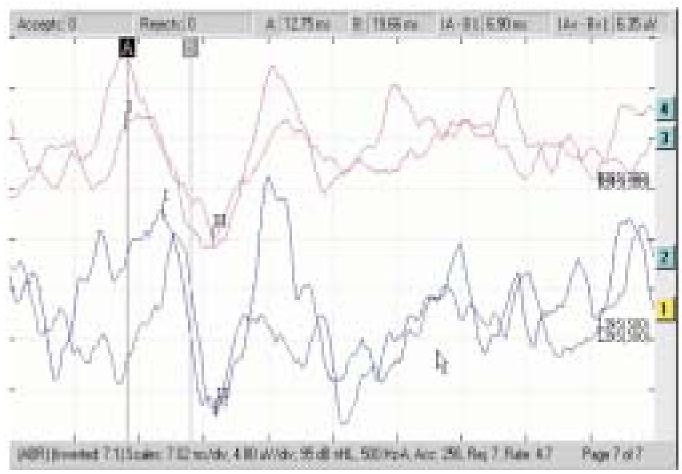
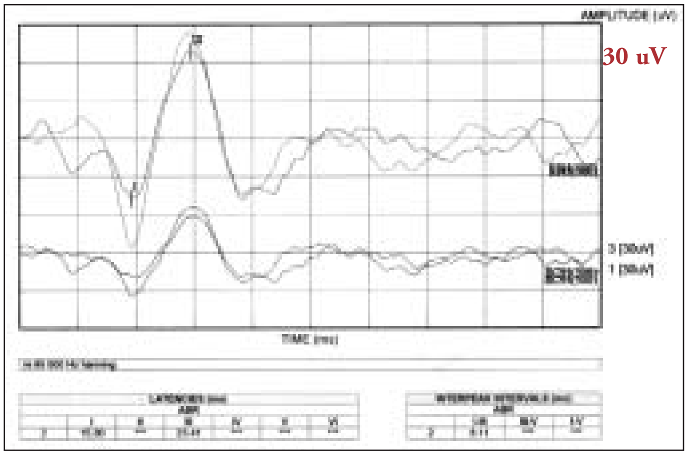


Fig. 7. *Autosomal dominant deafness diagnosed in a 24 y/o student at Gallaudet with no complaint or history of a balance disorder. VEMP responses are very poorly defined bilaterally and amplitudes are several standard deviations below controls with normal hearing and no complaint of imbalance.*

Possible explanation for the poor VEMP response in this subject could be congenital stapes ankylosis, which may accompany her genetic hearing loss and would prevent a normal VEMP reading owing to a loss of sound conduction through the middle ear. Immittance measurements did not indicate middle ear abnormality, however. Additional testing to include CT scan of the middle ear may help to identify a possible conductive component as explanation for the poor VEMP. Alternatively, the poor response may be of saccular origin reflecting type I/type II hair cell loss or maldevelopment.

Case 4. This patient is a 45 year-old male who has been deaf since birth and from a deaf family. He has Connexin 26 and 30 deafness, according to genetic studies conducted at the Gallaudet Genetics Clinic. He was diagnosed in 1976 with Meniere's disease, and he was treated several months later with migraine medication. He continued to take the medication until recently when he stopped because of rapid and irregular heart rate. He became vertiginous again and was gradually reintroduced to a low dose of his original migraine medication. He was seen for VEMP testing at the Gallaudet Balance Center.



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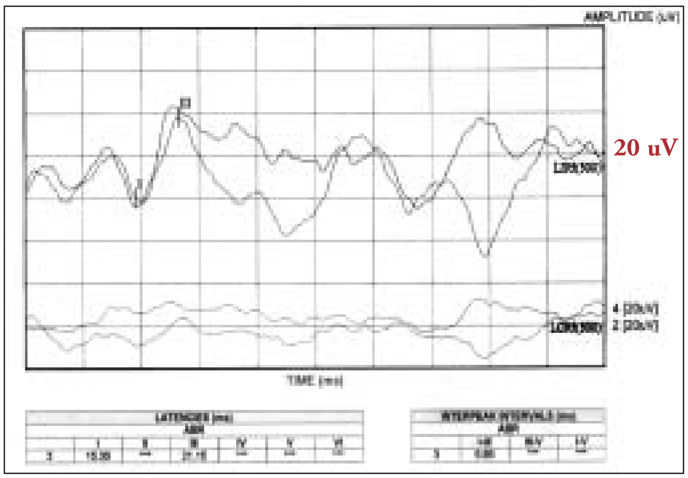

Left Side

Fig. 8. *VEMP results for a 45 year-old deaf patient (case # 4) showing ipsilateral (upper tracing) and contralateral (lower tracing) records for right and left sides. Wave latencies are within normal limits bilaterally (lower grid). Amplitude for the right side (127 uV) is normal, but left side amplitude (39 uV) is significantly reduced giving an augmented VEMP calculation of 127-39/ 127+39 = 0.53 which is significant for saccular hydrops. Follow-up evaluation by an otoneurologist is ongoing. Tracings are 'reversed' because of active (non-inverting) electrode placement on the sternum and referencing each SCM muscle as in case # 1. The GN Otometrics CHARTR EP print-out shows the VEMP application of the ABR program.*

VEMP results in case # 4 identified the left side as the probable site of saccular damage, which should assist in this patient's ongoing otoneurologic treatment. ENG caloric test results showed a 21% left reduced vestibular response which is marginally indicative of left side damage also.

Summary and Conclusions

These cases illustrate the potential clinical value of the VEMP procedure with D/HH persons, and further study involving several dozen subjects at Gallaudet should improve the diagnostic application with this population. An important aspect of the study is measuring reliability of the VEMP with re-testing conducted days or weeks following the initial test. To date, after measurement of 15 subjects, reliability is consistent. Also, segregating according to cause of deafness or hearing loss will be useful. This may be more challenging. In cases of acquired deafness this may never be precisely determined. For example, deaf persons may have always thought that their condition was caused by being dropped as a baby, because this was the explanation they understood. One subject reported that he became deaf because of an infection, but hospital records also showed that he suffered a fever of >105 degrees F. for several hours or perhaps even days. Genetic testing is more conclusive, but costly. Six (6) subjects with documented Connexin 26 autosomal recessive deafness have been tested in this series. This is the largest subgroup of subjects to date. Finally, comparison of VEMP results with caloric responses and other subtests of the VNG procedure will help to standardize the procedure. This is also an aspect of the Gallaudet study.

References

Ackley, R.S., Tamaki, C. (2003). Application of VEMP to Deaf and HoH Patients: A Clinical Study. American Speech-Language and Hearing Association National Convention. Chicago, IL. November, 2003.

Ackley, R.S., Ferraro, J., Arenberg, I.K. (1994). Diagnosis of Patients with Perilymphatic Fistula. *Seminars in Hearing*, Vol. 15, No. 1, pp. 37-42

Akin, F.W., Murnane, O.D., 2001. Vestibular evoked myogenic potentials: preliminary report. *J. Am. Acad. Audiol.* 12, 445-452.

Akin, F.W., Murnane, O.D., 2004. Vestibular evoked myogenic potentials. *Insights in Practice: Clinical Topics in Otoneurology*. GN Otometrics. April, 2004.

Aran, J.M., Pajor, A.M., de Sauvage, R.C., Erre, J.P., 2000. Role of the efferent medial olivocochlear system in contralateral masking and binaural interactions: an electrophysiological study in guinea pigs. *Audiology* 39(6), 311-21.

Bickford, R.G., Jacobson, J.L., Cody, D.T.R., 1964. Nature of averaged evoked potentials to sound and other stimuli in man. *Ann. N.Y. Acad. Sci.* 112, 204-223.

Cazals, Y., Erre, J.P., Aurousseau, C., 1987. Eighth nerve auditory evoked responses recorded at the base of the vestibular nucleus in the guinea pig. *Hear. Res.* 31, 93-98.

Cody, D.T.R., Bickford, R.G., 1969. Averaged evoked myogenic responses in normal man. *Laryngoscope* 79, 400-416.

Colebatch, J.G., Halmagyi, G.M., 1992. Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. *Neurology* 42, 1635-1636.

Colebatch, J.G., Halmagyi, G.M., Skuse, N.F., 1994. Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J. Neurol. Neurosurg. Psychiatry* 57, 190-197.

Didier, A., Cazals, Y., 1989. Acoustic responses recorded from the saccular bundle on the eighth nerve of the guinea pig. *Hear. Res.* 37, 123-128.

Ferber-Viart, C., Dubreuil, C., Duclaux, R., 1999. Vestibular evoked myogenic potentials in humans: a review. *Acta Otolaryngol.* 119, 6-15.

Halmagyi, G.M., Colebatch, J.G., 1995. Vestibular evoked myogenic potentials in the sternomastoid muscle are not of lateral canal origin. *Acta Otolaryngol. Suppl. (Stockholm)* 520, 1-3.

Halmagyi, G.M., Curthoys, I., 2000. Otolith function tests. In: Herdman, S.J. (Ed). Vestibular Rehabilitation. *Philadelphia: FA Davis,* 196-214.

Itoh, A., Kim, Y.S., Yoshioka, K., Kanaya, M., Enomoto, H., Hiraiwa, F., Mizuno, M., 2001. Clinical study of vestibular-evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. *Acta Otolaryngol. Suppl.* 545, 116-119.

McCue, M.P., Guinan, J.J. Jr., 1994. Acoustically responsive fibers in the vestibular potentials in the vestibular nerve of the cat. *J. Neurosci.* 14, 6058-6070.

Minor, L.B., 2000. Superior canal dehiscence syndrome. *Am J Otol.* 21(1):9-19.

Murofushi, T., Curthoys, I.S., 1997. Physiological and anatomical study of click-sensitive primary vestibular afferents in the guinea pig. *Acta Otolaryngol. (Stockholm)* 117, 66-72.

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

Murofushi, T., Halmagyi, G.M., Yavor, R.A., Colebatch, J.G., 1996a. Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis: an indicator of inferior vestibular nerve involvement? *Arch. Otolaryngol. Head Neck Surg.* 122, 845-848.

Murofushi, T., Curthoys, I.S., Gilchrist, D.P., 1996b. Responses of guinea pig vestibular nucleus neurons to clicks. *Exp. Brain Res.,* 111, 149-152.

Murofushi, T., Shimizu, K., Takegoshi, H., Cheng, P.-W., 2001. Diagnostic value of prolonged latencies in the vestibular evoked myogenic potential. *Arch. Otolaryngol. Head Neck Surg.* 127, 1069-1072.

Ochi, K., Ohashi, T., Nishino, H., 1999. Variance of vestibular-evoked myogenic potentials. *Laryngoscope* 111, 522-527.

Polyakov, A., Pratt, H., 2003. The cumulative effect of high click rate on monaural and binaural processing in the human auditory brainstem. *Clin. Neurophysiology* 114(2), 366-75.

Robertson, D,D., Ireland, D.J., 1995. Vestibular evoked myogenic potentials. J. Otolaryngol. 24, 3-8.

Shojaku, H., Takemori, S., Kobayashi, K., Watanabe, Y., 2001. Clinical usefulness of glycerol vestibular-evoked myogenic potentials: preliminary report. *Acta Otolaryngol. Suppl.*; 545:65-8.

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 Otolaryngol. Suppl.* 2001; 545:41-9.

Tamaki, C., Ackley, R.S., 2003. Vestibular Evoked Myogenic Potentials: Contralateral Masking Effects. *Hearing Research.* In Review.

Takegoshi, H., Murofushi, T., 2003. Effect of white noise on vestibular evoked myogenic potentials. *Hear. Res.* 176, 59-64.

Townsend, G.L., Cody, D.T.R., 1971. The averaged inion response evoked by acoustic stimulation: its relationship to the saccule. *Ann. Otol. Rhinol. Laryngol.* 80, 121-131.

Tsutsumi, T., Komatsuzaki, A., Noguchi, Y., Tokano, H., Kitamura, K., 2001. Postoperative vestibular-evoked myogenic potentials in cases with vestibular schwannomas. *Acta Otolaryngol.* 121, 490-493.

Ungan, P., Yagcioglu, S., 2002. Origin of the binaural interaction component in wave P4 of the short-latency auditory evoked potentials in the cat: evaluation of serial depth recording from the brainstem. *Hear. Res.* 167(1-2), 81-101.

Wu, C.-H., Murofushi, T., 1999. The effect of click repetition rate on vestibular evoked myogenic potential. *Acta Otolaryngol. (Stockholm)* 119, 29-32.

Young, Wu,C.C., Wu, C.-H. (2002) Augmentation of vestibular evoked myogenic potentials: an indication for distended saccular hydrops. *Laryngoscope.* 112 (3). 509-512.

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