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# Vestibular Evoked Myogenic Potentials: Preliminary Report

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**Abstract**

Vestibular evoked myogenic potentials (VEMPs) are short-latency electromyograms evoked by high-level acoustic stimuli recorded from surface electrodes over the tonically contracted sternocleidomastoid (SCM) muscle. These responses are presumed to originate in the saccule. The purpose of this preliminary report is to provide an overview of our initial experience with the VEMP by describing the responses obtained in five subjects. Click-evoked VEMPs were present at short latencies in two normal-hearing subjects, one patient with profound congenital sensorineural hearing loss, and one patient with a severe sensorineural hearing loss due to Meniere's disease. Additionally, VEMPs were absent in a patient with profound sensorineural hearing loss following removal of a cerebellopontine angle tumor. The amplitude of the VEMP was influenced by the amount of background activity of the SCM muscle, stimulus level, and stimulus frequency. Tone-burst evoked responses showed an inverse relationship between stimulus frequency and response latency. VEMPs may prove to be a reliable technique in the clinical assessment of vestibular function.

**Key Words:** Electromyography, motor evoked potentials, saccule, sternocleidomastoid muscle, vestibular function tests, vestibular nerve

**Abbreviations:** EMG = electromyography, SCM = sternocleidomastoid, VEMP = vestibular evoked myogenic potential

The vestibular sensory organs include three semicircular canals and two otolithic organs. Most routine clinical vestibular tests evaluate the horizontal semicircular canal and fail to evaluate the remaining four vestibular organs. Recent research is focusing on developing clinical tests to assess otolith function. Vestibular evoked myogenic potentials (VEMPs) are short-latency electromyograms (EMGs) evoked by high-level acoustic stimuli recorded from surface electrodes over the tonically contracted sternocleidomastoid (SCM) muscle. These responses are presumed to originate in the saccule (Colebatch and Halmagyi, 1992). VEMPs have been proposed as a clinical test of saccular and/or inferior vestibular nerve function (Colebatch, 2001).

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In some lower vertebrates such as amphibians and fish, the saccule often serves as the organ of hearing (Moffat and Capranica, 1976; Popper et al, 1982). Although the cochlea has replaced the saccule as the primary organ of hearing in mammals, there is neurophysiologic evidence that the mammalian saccule remains responsive to sound. Recordings from single afferent fibers of the inferior vestibular nerve in the squirrel monkey (Young et al, 1977), cat (McCue and Guinan, 1994), and guinea pig (Murofushi et al, 1995) have demonstrated responsiveness to sound at frequencies and levels within the range of human hearing.

Sound-evoked vestibular symptoms have been observed in humans since the early work of Tullio (1929). Indirect physiologic evidence of the human vestibular system response to auditory stimuli was initially provided by Bickford and colleagues (1964), who described a short-latency myogenic potential recorded just below the inion in response to high-level click stimuli. They showed that the response was generated by the EMG of neck muscles and that the amplitude of the response was dependent on stimulus level and tonic EMG amplitude. In this and

a subsequent series of experiments, the saccule was implicated as the origin of the response (Cody et al, 1964; Townsend and Cody, 1971). The specificity of the response, however, was questioned, and the vestibular origin was not widely accepted (Meier-Ewert et al, 1974; Douek, 1982). More recently, the VEMP response has been recorded from a site over the SCM muscle, and studies using human subjects with well-documented peripheral audiovestibular lesions have confirmed the vestibular origin of the VEMP (Colebatch and Halmagyi, 1992). These authors demonstrated that the VEMP response is abolished following unilateral vestibular neurectomy. In addition, several studies have shown that the VEMP is preserved in patients with profound sensorineural hearing loss and normal vestibular function and in a patient with extreme atrophy of the cochlear branch of the eighth cranial nerve (Colebatch et al, 1994; Murofushi et al, 1999; Ozeki et al, 1999; Ito et al, 2001). These findings suggest that the VEMP response is not mediated by the cochlea. The neurophysiologic and clinical data indicate that VEMPs are mediated by a pathway that includes the saccular macula, inferior vestibular nerve, lateral vestibular nucleus, lateral vestibulospinal tract, and motoneurons of the ipsilateral SCM muscle (Halmagyi and Curthoys, 2000). The purpose of this preliminary report is to provide an overview of our initial experience with the VEMP by describing the responses obtained in five subjects, two with normal hearing and three with hearing loss.

## METHOD

### Subjects

Subjects 1 (45-year-old male) and 2 (33-year-old male) had normal hearing sensitivity and no history of vestibular or neurologic disease. Subjects 3, 4, and 5 had unilateral severe or profound sensorineural hearing loss. Subject 3 (40-year-old female) had a congenital profound sensorineural hearing loss in her left ear and normal vestibular function as determined by electronystagmography. Subject 4 (59-year-old male) had experienced loss of hearing and loss of vestibular function in his left ear following removal of a cerebellopontine angle tumor. Subject 5 had a severe sensorineural hearing loss in the right ear. He was diagnosed with Meniere's disease and had undergone a right endolymphatic shunt procedure.

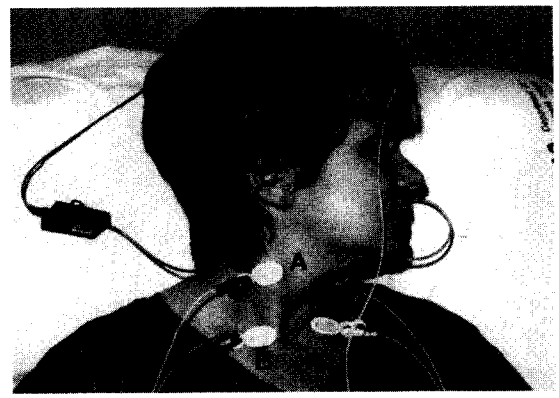



**Figure 1** Electrode sites for recording vestibular evoked myogenic potentials (VEMP): (A) noninverting electrode on the sternocleidomastoid muscle and (B) inverting electrode on the upper sternum. Ground electrode is located on the forehead. The EMG differential surface electrode (not shown in this figure) was located just below A.

### Procedures

VEMPs were obtained by averaging the acoustically evoked EMG of the SCM muscle during tonic contraction. The subjects were seated upright and asked to turn their heads to one side to activate unilaterally the SCM muscle. For example, in Figure 1, the right ear is stimulated during activation of the right SCM muscle by head rotation to the left. A two-channel recording of the VEMP was obtained with noninverting electrodes placed at the midpoint of the SCM muscle on each side of the neck (A in Fig. 1). The inverting electrode sites were at the sternoclavicular junctions (B in Fig. 1), and the ground electrode was placed on the forehead. Stimuli were presented monaurally to the ear ipsilateral to the activated SCM muscle via ER3A insert earphones at a repetition rate of 5/sec. The response was amplified (5000) and bandpass filtered from 20 to 1500 Hz with a 12 dB/octave slope (Nicolet Spirit 2000). The 100-msec epochs (including a 20-msec prestimulus baseline) were digitized at 5 kHz. Each waveform consisted of responses to 128 stimuli, and three waveforms were obtained from each side. Peak-to-peak amplitudes and absolute latencies were calculated from the mean of the three responses for each subject. To assess the effects of stimulus level, responses were obtained from subject 2 using rarefaction click stimuli presented at levels from 85 to 100 dB nHL in 5-dB steps. To determine the effect of frequency on the VEMP, responses were obtained from subject 2 using tone bursts at 250, 500, 1000, and 2000 Hz (rarefaction onset phase, Blackman gating func-

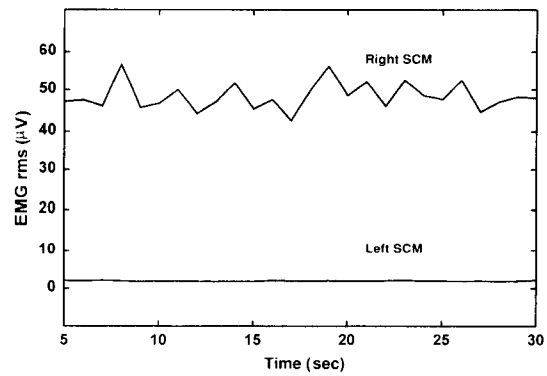



**Figure 2** An example of EMG rms amplitude level measured during a VEMP recording while the right SCM muscle was activated (i.e., head turned left as shown in Fig. 1). This display provided each subject with the visual feedback necessary to maintain the target EMG amplitude.

tion, two-cycle rise–fall time with no plateau) presented at 120 dB $_{peak}$SPL.

To control for the effects of tonic EMG level on the VEMP, a two-channel EMG recording was obtained simultaneously with the evoked potential recordings. An EMG stand-alone differential surface electrode (DelSys, Inc., DE-2.1) was placed on the SCM muscle midway between the mastoid process and sternoclavicular junction on each side of the neck (although not shown in Fig. 1, placement was below A), and a reference electrode was attached to the wrist. The EMG signals from each channel were amplified (10,000), bandpass filtered from 20 to 450 Hz (12 dB/octave), and digitized at 1024 Hz via a portable EMG unit (DelSys, Inc., Bagnoli-2). During head rotation to one side, the subjects were provided with visual feedback of their EMG amplitude via the computer monitor and software (Delsys, Inc., EMGworks Signal Acquisition and Analysis Software). Figure 2 is an example of the visual feedback display of EMG amplitude provided to subject 2 during 25 sec of head rotation to the left (i.e., activation of the right SCM muscle). During right SCM muscle activation, subject 2 was able to maintain EMG amplitude at a level that approximated the 50-μV target. In contrast, the EMG amplitude recorded from the unactivated left SCM muscle approximated 0 μV. Each subject was instructed to maintain the rectified EMG rms amplitude within a target range of 30 to 50 μV during the recording of each evoked potential waveform.

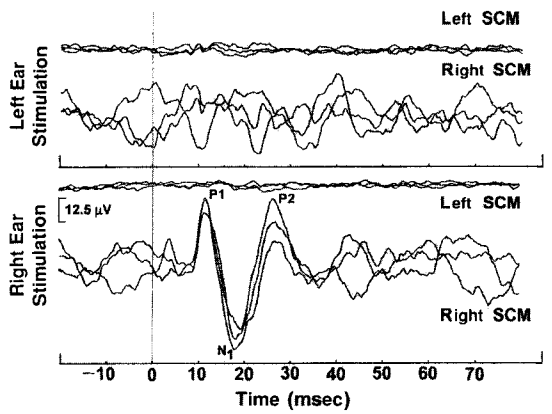



**Figure 3** Two-channel VEMP recordings obtained from a normal-hearing subject (1) with 100 dB nHL click stimuli delivered to the left ear (upper two waveforms) and the right ear (lower two waveforms). For all conditions, the subject's head was turned to the left, which activated the right SCM. The vertical line at 0 msec indicates the onset of the stimulus. Note that VEMPs are present only when the acoustic stimulus is delivered to the ear (right) ipsilateral to the side of SCM muscle activation.

## RESULTS AND DISCUSSION

### Subjects with Normal Hearing

Short-latency, large-amplitude responses were evoked with 100 dB nHL click stimuli during tonic SCM muscle activation from both subjects with normal hearing. The ipsilateral (right ear stimulation/right SCM activation) and contralateral (left ear stimulation/right SCM activation) responses obtained from subject 1 are illustrated in Figure 3. For all conditions in Figure 3, the head was turned to the left, which activated the right SCM muscle. The vertical line at 0 msec indicates the onset of the click. In the upper panel of Figure 3, the stimulus was presented to the left ear during activation of the right SCM muscle. In this condition, a VEMP was not obtained from either the left or right SCM muscle. Notice, however, that the overall amplitude of the recording from the right (activated) SCM muscle was greater than the amplitude from the left (unactivated) SCM muscle. This difference in overall amplitude reflects the difference in the level of tonic muscle activation (see Fig. 2). In the lower panel of Figure 3, the stimulus was presented to the right ear during activation of the right SCM muscle. In this condition, a VEMP was obtained only from the right (activated) SCM muscle, and no response was obtained over the left (unactivated) SCM muscle. Thus, VEMPs were obtained only when the stimulus was ipsilateral to the activated SCM muscle, which is consistent with the results of Li et al (1999) in humans and Kushiro et al

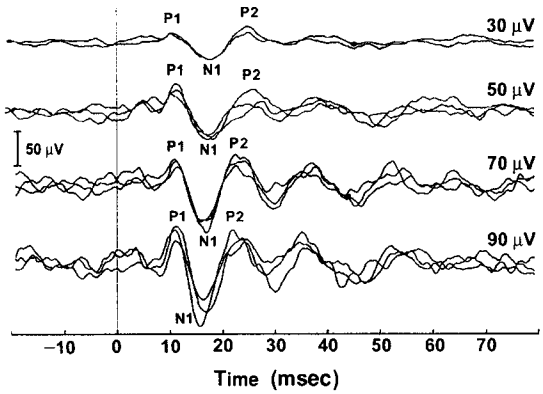



**Figure 4** VEMP recordings obtained with 100 dB nHL click stimuli from the left SCM muscle of subject 2 showing the effect of SCM muscle EMG amplitude on evoked potential amplitude. Target EMG rms levels resulting from different degrees of head rotation are indicated beside each waveform. The vertical line at 0 msec indicates the onset of the stimulus.

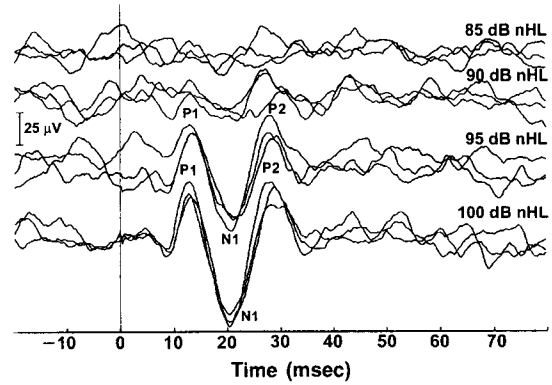



**Figure 5** VEMPs obtained from the left SCM muscle of subject 2 illustrating the effect of click stimulus level on VEMP amplitude. Stimuli were delivered to the left ear, and the stimulus levels are indicated beside each waveform. Target EMG rms amplitude was maintained at 50 μV.

(1999) in cats, suggesting that the VEMP is mediated by an ipsilateral pathway.

The salient features of the response waveform (lower panel in Fig. 3) were a positive peak (P1) at 10.9 msec, a negative peak (N1) at 17.7 msec, and a second positive peak (P2) at 25.7 msec. Peak-to-peak amplitudes were 69.5 μV for P1–N1 and 60.7 μV for N1–P2. These response characteristics agree with descriptions of the VEMP contained in previous reports (e.g., Colebatch et al, 1994).

To determine the effect of tonic EMG rms amplitude on the VEMP amplitude, subject 2 was instructed to vary the amount of unilateral SCM muscle activation (by increasing or decreasing the degree of head rotation) to target EMG rms values of 30, 50, 70, and 90 μV. The click stimulus level was 100 dB nHL. Figure 4 shows VEMP responses obtained from subject 2 during SCM activation at the four target EMG rms values. The responses in Figure 4 reveal that VEMP amplitude increased with an increase in EMG rms amplitude (target EMG rms values are indicated at the top right of each waveform), whereas response latency remained fairly constant (±1 msec). Lim et al (1995) also demonstrated that VEMP amplitude was positively correlated with both stimulus level and background EMG. As VEMP amplitude may prove to be a diagnostically useful metric, the importance of controlling the level of tonic EMG activity has been emphasized (Colebatch et al, 1994; Colebatch, 2001).

To demonstrate the effect of stimulus level on the VEMP, recordings were obtained from subject 2 at four click levels (85, 90, 95, and 100 dB nHL) while maintaining tonic EMG amplitude at ~ 50 μV. Figure 5 shows VEMP waveforms obtained at the four click levels. No response was obtained at 85 dB nHL, and a questionable response was obtained at 90 dB nHL. The VEMP threshold for this subject was determined to be 95 dB nHL with the largest amplitude response obtained at 100 dB nHL. Although the amplitude of the VEMP increased as a function of stimulus level, the response latency remained constant (at 95 dB nHL, P1 = 12.5 msec, N1 = 20.1 msec; at 100 dB nHL, P1 = 12.1 msec, N1 = 19.5 msec). These preliminary findings are similar to results obtained in previous studies (Colebatch et al, 1994; Lim et al, 1995; Ochi et al, 2001).

To determine the effect of stimulus frequency on the VEMP, evoked responses were elicited with tone-burst stimuli. VEMP recordings obtained from subject 2 at each tone-burst frequency are shown in Figure 6, with latencies and amplitudes listed in Table 1. VEMPs were present at 250, 500, and 1000 Hz and absent at 2000 Hz. These preliminary findings revealed an inverse relationship between VEMP latency and stimulus frequency. Response amplitudes also varied with stimulus frequency, and the largest amplitude VEMP was obtained with 500-Hz tone bursts. Previous studies have reported similar frequency effects on the VEMP. Todd et al (2000) demonstrated that the VEMP has well-defined frequency tuning with a maximum in response amplitude occurring between 300 and 350 Hz. Similarly, Murofushi et al (1999) observed larger VEMPs with 500-Hz tone bursts

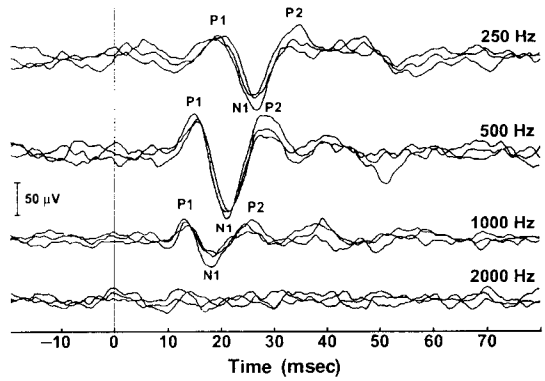



**Figure 6** VEMPs obtained from the left SCM muscle of subject 2 illustrating the effect of frequency on VEMP amplitude and latency. Stimuli were delivered to the left ear, and the tone-burst frequencies are indicated.

**Table 1 VEMP Latencies and Amplitudes for Tone-Burst Stimuli Obtained in a Normal Subject (2)**

| *Frequency (Hz)* | *Latency (msec)* | | | *Amplitude (μV)* | |
|---|---|---|---|---|---|
| | *P1* | *N1* | *P2* | *P1–N1* | *N1–P2* |
| 250 | 18.3 | 25.5 | 32.9 | 104.4 | 102.8 |
| 500 | 14.3 | 20.3 | 27.3 | 150.2 | 136.0 |
| 1000 | 12.5 | 17.3 | 24.3 | 61.1 | 54.9 |
| 2000 | NR | NR | NR | NR | NR |

NR = no response.

than with 1000- and 2000-Hz tone bursts. The VEMP frequency response may be due to a residual auditory function of the saccule and/or inferior vestibular nerve. This theory is supported by the neurophysiologic findings that acoustically responsive afferent fibers in the mammalian inferior vestibular nerve have broad, V-shaped tuning curves with best frequencies between 500 and 1000 Hz (McCue and Guinan, 1994, 1995). Alternatively, Todd et al (2000) related VEMP frequency tuning to the resonant properties of the saccule and effectively modeled this resonance as a mass-spring damper system.

## Subjects with Hearing Impairment

Figures 7, 8, and 9 depict the VEMPs in three subjects (3, 4, and 5, respectively) with either severe or profound unilateral sensorineural hearing loss. Subject 3 had a history of congenital profound sensorineural hearing loss in her left ear and normal hearing sensitivity in her right ear. The bithermal caloric test revealed normal and symmetric nystagmic responses. Figure 7 illustrates VEMPs present in response to 100 dB nHL click stimuli presented to either ear. In this case, the VEMP and the caloric test results suggested normal saccular and horizontal semicircular canal function, respectively.

Subject 4 experienced loss of hearing and vestibular function in his left ear following sectioning of the eighth cranial nerve secondary to removal of a left cerebellopontine angle tumor. Subject 4 had a moderate-to-profound high-frequency sensorineural hearing loss in the right ear and a profound sensorineural hearing loss in the left ear. Nystagmic responses to bithermal water caloric irrigation were absent in the left ear and present in the right ear. As shown in Figure 8, VEMPs were absent with left ear

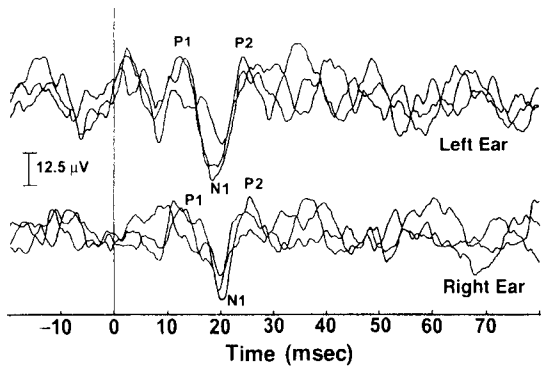
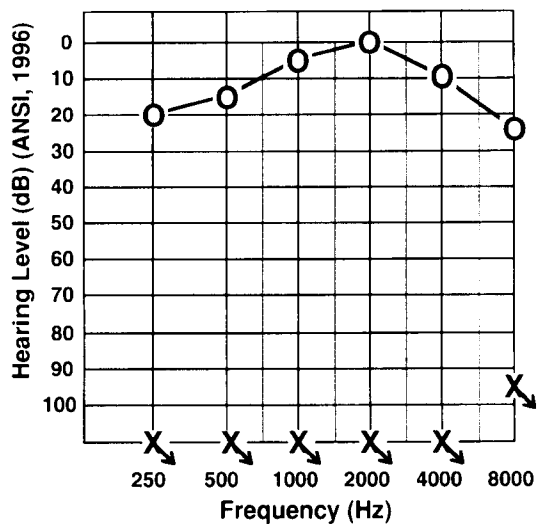
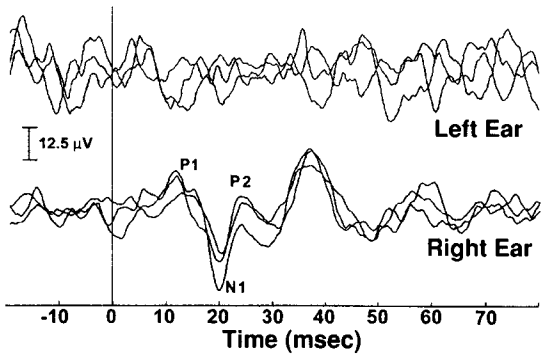



**Figure 7** VEMPs (left panel) obtained from subject 3 in response to 100 dB nHL clicks were present in both ears. Subject 3 has a history of left congenital unilateral sensorineural hearing loss. The audiogram (right panel) revealed profound sensorineural hearing loss on the left, and the caloric test yielded symmetric nystagmic responses.





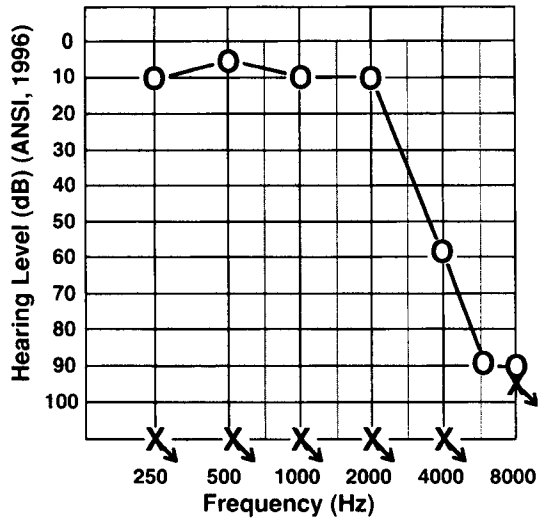
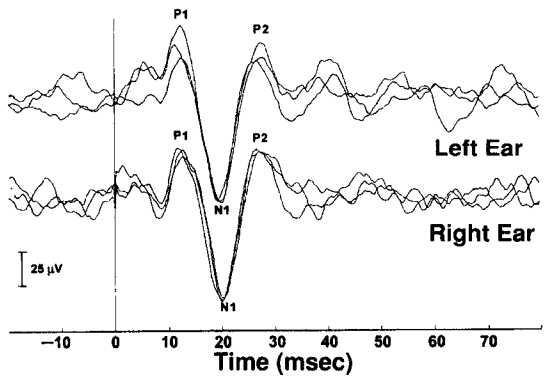




**Figure 8** VEMPs (left panel) obtained from subject 4 in response to 100 dB nHL clicks were absent with left ear stimulation and present with right ear stimulation. The audiogram (right panel) revealed profound sensorineural hearing loss on the left following removal of a left cerebellopontine angle tumor, and the caloric test yielded a 100 percent left unilateral weakness.

stimulation and present with right ear stimulation. For this subject, both the VEMP and the caloric responses were absent, which is consistent with decreased vestibular function presumably caused by the loss of vestibular nerve function.

Subject 5 had a diagnosis of Meniere's disease and had undergone a right endolymphatic shunt procedure. He had a mild-to-moderate high-frequency sensorineural hearing loss in the left ear and a severe sensorineural hearing loss in the right ear. Nystagmic responses to bithermal water caloric irrigation were absent in the right ear and present in the left ear. Figure 9 shows VEMPs present in response to 100 dB nHL click stimuli presented to either ear. In this case, the VEMPs suggested normal bilateral saccular function, whereas the caloric test results were consistent with abnormal right horizontal semicircular canal function.

VEMP recordings in the three hearing-impaired subjects are consistent with previous studies demonstrating that the VEMP response is independent of the degree of sensorineural hearing loss (Bickford et al, 1964; Colebatch et al, 1994; Ozeki et al, 1999). Recently, Ito et al (2001) reported the presence of VEMPs bilaterally in a patient with unilateral profound sensorineural hearing loss caused by extreme narrowing of the internal auditory meatus with absent or extremely thin cochlear nerve. These findings provide structural and functional evidence that the VEMP is not mediated by the cochlea.



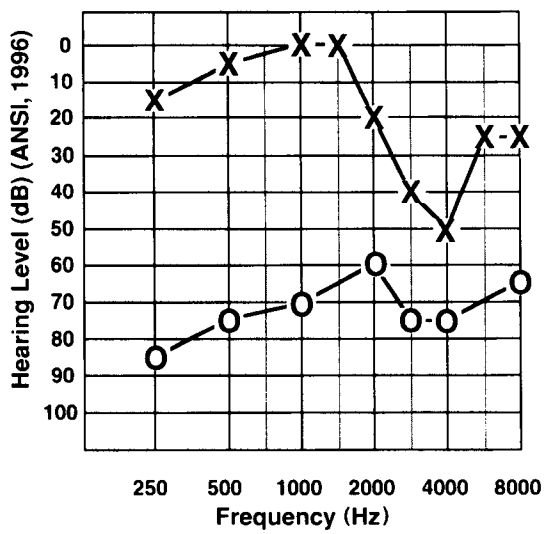




**Figure 9** VEMPs (left panel) obtained from subject 5 in response to 100 dB nHL clicks were present in both ears. Subject 5 had a diagnosis of Meniere's disease and had undergone a right endolymphatic shunt procedure. The audiogram (right panel) revealed a severe sensorineural hearing loss on the right, and the caloric test yielded a 100 percent right unilateral weakness.

Consistent with previous reports, the data from the three subjects with hearing loss indicated that the presence or absence of the VEMP does not appear to be related to the caloric test results. For example, Murofushi et al (1998) demonstrated that patients with acoustic neuromas may present with normal caloric responses and abnormal VEMPs or with abnormal caloric responses and normal VEMPs. Similarly, de Waele et al (1999) reported no relationship between caloric responses and VEMPs in patients with Meniere's disease. These studies suggest that the VEMP and the caloric test most likely reflect the function of two different vestibular structures.

## CONCLUSIONS

In summary, click-evoked VEMPs were present at short latencies in both subjects with normal hearing and in two subjects with either severe or profound sensorineural hearing loss. In these subjects, VEMPs were obtained only when the stimulus was ipsilateral to the activated SCM muscle, consistent with previous findings indicating that the VEMP is mediated by an ipsilateral pathway. The amplitude of the VEMP is influenced by the amount of background activity of the SCM muscle, stimulus level, and stimulus frequency. Tone-burst evoked responses showed an inverse relationship between stimulus frequency and the response latency of the VEMP.

VEMPs may prove to be a reliable technique in the clinical assessment of vestibular function. Currently, conventional vestibular assessment is limited to evaluation of one of the five peripheral vestibular end organs, the horizontal semicircular canal. VEMP recordings may supplement the current test battery by providing diagnostic information about saccular and/or inferior vestibular nerve function. Prior to routine clinical application of this response, further study is needed to determine the effects on the VEMP of hearing and vestibular loss, stimulus parameters, tonic EMG level, and aging. Normative data must be established to determine the diagnostic utility of this response in various audiovestibular disorders.

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