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The influence of voluntary tonic EMG level on the vestibular-evoked myogenic potential

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Abstract—Vestibular-evoked myogenic potentials (VEMPs) are proposed as a reliable test to supplement the current vestibular test battery by providing diagnostic information about saccular and/or inferior vestibular nerve function. VEMPs are short-latency electromyograms (EMGs) evoked by high-level acoustic stimuli and recorded from surface electrodes over the tonically contracted sternocleidomastoid muscle. VEMP amplitude is influenced by the EMG level, which must be controlled. This study examined the ability of subjects to achieve the EMG target levels over a range of target levels typically used during VEMP recordings. In addition, the influence of target EMG level on the latency and amplitude of the click- and tone-evoked VEMP was examined. The VEMP amplitude increased as a function of EMG target level, and the latency remained constant. EMG target levels ranging from 30 µV to 50 µV are suggested for clinical application of the VEMP.

Key words: electromyography, motor-evoked potentials, saccule, sternocleidomastoid muscle, vestibular function tests, vestibular nerve.

INTRODUCTION

Vestibular-evoked myogenic potentials (VEMPs) are short-latency electromyograms (EMGs) recorded from the tonically contracted sternocleidomastoid (SCM) muscle (m.) in response to acoustic stimuli at relatively high levels. The VEMP waveform consists of an early positive-negative component that occurs at 13 ms to 23 ms poststimulus (p13-n23 or P1-N1) and a later negative-positive component that occurs at 34 ms to 44 ms poststimulus (n34-p44 or N3-P4) [1]. The early component of the VEMP depends on the integrity of vestibular afferents as the response is abolished after vestibular nerve section but preserved in subjects with severe-to-profound sensorineural

Abbreviations: ANOVA = analysis of variance, EMG = electromyogram, HL = hearing level, m. = muscle, SCM = sternocleidomastoid, VEMP = vestibular-evoked myogenic potential, VRT = vestibular rehabilitation therapy.

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hearing loss [2]. It has been hypothesized that the later component of the VEMP is mediated by cochlear afferents [1], although recent evidence suggests that the source of the later component has not been delineated [3].

Single-unit recordings from the afferent inferior vestibular nerve in the squirrel monkey, cat, and guinea pig demonstrate responsiveness to acoustic stimuli at frequencies and levels within the range of human hearing [4–6]. Recordings of single motor unit action potentials in the human SCM m. provide direct evidence that the surface-recorded VEMP reflects a short period of inhibition of SCM motoneuron firing [7]. In addition, direct recordings from SCM motoneurons of decerebrate cats confirm that saccular afferent stimulation produces inhibitory postsynaptic potentials [8]. Neurophysiological and clinical data indicate that VEMPs recorded from the SCM m. following unilateral activation are mediated by a pathway that includes the saccular macula, inferior vestibular nerve, the lateral vestibular nucleus, the vestibulospinal tract, and the motoneurons of the ipsilateral SCM m. [9].

Conventional vestibular assessment (caloric and rotational testing) is limited to the evaluation of the horizontal semicircular canal, which is one of the five vestibular end organs (three semicircular canals, the utricle, and the saccule). VEMPs have been proposed as a reliable test that may supplement the current vestibular test battery by providing diagnostic information about saccular and/or inferior vestibular nerve function [10]. The diagnostic utility of the VEMP has been examined for various audiovestibular and neurological disorders, including vestibular labyrinthitis, Ménière's disease, benign paroxysmal positional vertigo, superior canal dehiscence, the Tullio phenomenon, vestibular schwannoma, multiple sclerosis, and spinocerebellar degeneration [11–22].

The clinical interpretation of the VEMP has focused primarily on amplitude or threshold asymmetries between the right and left ears and, thereby, an indication of the likely side of the pathology [23]. VEMP amplitude is positively correlated with both click-stimulus level and EMG level, whereas VEMP latency is independent of both factors [1,24,25]. Controlling the level of tonic EMG, therefore, is a prerequisite for the accurate interpretation of interaural VEMP amplitude differences. To control the level of the tonic EMG, subjects are instructed to maintain a constant rectified EMG target level for the duration of the evoked potential recording via some form of visual feedback [1,26–28]. Although several studies have established the positive linear relationship between click-evoked VEMP amplitude and tonic EMG level [1,24,25], no data exist concerning the ability of subjects to achieve specific EMG target levels during recording of the VEMP.

Most investigators measure VEMPs using click stimuli, although animal studies suggest that acoustically responsive afferent fibers in the inferior vestibular nerve are most sensitive to low-frequency acoustic stimuli [6,29]. Low-frequency tone bursts, therefore, may produce a more robust VEMP response than broadband clicks. Relatively few data, however, report VEMP response characteristics using tone-burst stimuli [30–33], and no data can be found on the influence of voluntary EMG level on tone-evoked VEMP amplitude.

This study determined the ability of subjects to achieve EMG target levels over a range of target levels typically used during recording of the VEMP. The influence of target EMG level on the latency and amplitude of the click- and tone-evoked VEMP was also examined.

METHODS

Subjects

Eleven subjects (1 male and 10 females) ranging in age from 18 to 34 years (mean = 25.3 years; standard deviation [SD] = 4.3) participated in the study. All subjects had normal hearing sensitivity (20 dB hearing level [HL], American National Standards Institute [ANSI], 1996) at the octave frequencies from 250 Hz to 8,000 Hz, negative histories of middle-ear pathology, and no history of vestibular or neurological disease. In addition, histories were negative for open or closed head injury and cervical injury. Approval was obtained from all subjects, and the procedures followed the standards of the institutional review board.

Procedures

To determine the influence of target EMG level on the VEMP, we recorded tonic EMG and VEMPs concurrently from one side of each subject. A randomization was performed prior to the experiment in order to achieve a balance between the number of right (n = 5) and left (n = 6) sides. The subjects were seated in an upright and erect position with both feet resting on a foot support. A lumbar half-roll was positioned comfortably behind the low back to maintain the normal lordotic curve of the

lumbar spine. Postural alignment was achieved with the subjects maintaining hip flexion at 90°, knee flexion at 90°, and ankle dorsiflexion at neutral. Both upper limbs were positioned with the shoulders internally rotated ~20° and the elbows resting in alignment with the trunk and flexed to 90°. The forearms were supported with two towels placed on the thighs. The wrists and fingers were in a resting position. The subjects were asked to turn their heads to one side to activate unilaterally the SCM m. An acoustic stimulus was delivered to the ear ipsilateral to the activated SCM m., and the tonic EMG level and evoked potential recording (VEMP) were simultaneously recorded from the activated side.

Tonic EMG Measurement

A single-channel EMG recording was obtained with an EMG stand-alone differential surface electrode (DelSys, DE-2.1) placed on the SCM m. midway between the mastoid process and sternoclavicular junction on one side of the neck with a reference electrode attached to the wrist. The EMG signal was amplified (10,000), bandpass filtered from 20 to 450 Hz (12 dB/octave), and digitized at 1024 Hz via a portable EMG unit (DelSys, Bagnoli-2). The subjects were provided visual feedback of their rectified EMG amplitude via the computer monitor and software (Delsys, EMGworks Signal Acquisition and Analysis Software) [28]. During head rotation, the subjects were asked to maintain the rectified EMG amplitude at the target level for the duration of each trial (~40 s). The EMG amplitude for each trial was calculated from a 20 s window applied to the steady-state portion of the EMG recording. The mean EMG amplitude of the three trials for each stimulus at each EMG target level was used for data analysis.

VEMP Measurement

VEMPs were recorded by an averaging of the acoustically evoked electromyogram of the SCM m. during tonic contraction. The recording methods were similar to those reported previously [28]. A single-channel recording of the evoked potential was obtained with a noninverting electrode placed at the midpoint of the SCM m., the inverting electrode site on the sternoclavicular junction, and the ground electrode on the forehead. Absolute electrode impedances were ≤5,000 Ω and interelectrode impedances were ≤2,000 Ω. VEMPs were obtained from each subject with the use of 100 μs clicks presented at 100 dB normal hearing level [nHL] (134 dB peak sound pressure level [SPL]) and 500 Hz tone bursts (rarefaction onset phase, Blackman gating function, two-cycle rise-fall time with no plateau) presented at 120 dB_{peak} SPL. These stimuli have been shown to produce reliable and robust VEMPs in human subjects with normal audiovestibular function [1,29–32]. The stimuli were presented monaurally to the ear ipsilateral to the activated SCM m. via ER3A (Etymotic Research) insert earphones at 5/s. The stimulus order (click versus 500 Hz tone burst) was randomized for each subject. Responses to each stimulus were obtained at rectified EMG root-mean-square target levels of 0 μV, 30 μV, 50 μV, 70 μV, and 90 μV. The order of the EMG target levels was selected randomly and applied to the first stimulus and repeated in the same order for the second stimulus.

The response was amplified (5,000) and bandpass filtered from 20 Hz to 1500 Hz with a 12 dB/octave slope (Nicolet Spirit 2000). The 100 ms epochs included a 20 ms prestimulus baseline. The responses to 128 stimuli were averaged, and three waveforms were obtained for each stimulus at each rectified EMG target. Peak-to-peak amplitudes and absolute latencies were calculated from the mean of the three responses to each stimulus at each EMG target level.

RESULTS

Representative waveforms obtained from one subject at each EMG target level and for each stimulus are shown in **Figure 1**. The responses to the 500-Hz tone burst are shown on the left and responses to the click stimulus are shown on the right. The target EMG levels are indicated in the center of the figure. The three waveforms for each condition show little variation in amplitude and latency and represent the intrasubject variability. VEMP amplitude increased as a function of EMG target level, whereas VEMP latency is relatively constant. In addition, VEMP amplitude was larger for the 500 Hz tone bursts than for the clicks at each EMG target level. No response was recorded when the SCM m. was not activated (EMG target level = 0 μV).

Figure 2 shows bivariate plots of the individual EMG amplitude as a function of the target EMG level and illustrates the ability of the subjects to achieve EMG target levels over a range of target levels typically employed during recording of the VEMP. The upper panel shows the data for the click stimulus, and the lower

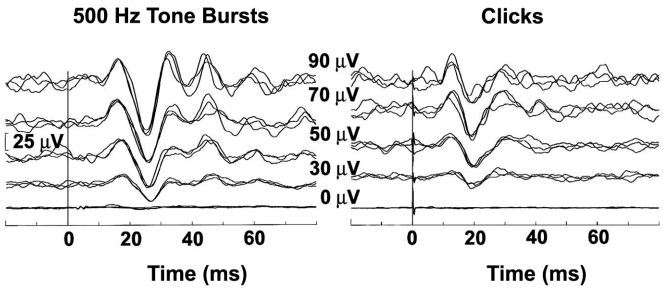

Figure 1.
VEMPs obtained from single subject at each EMG target level and for 500 Hz tone bursts (left) and clicks (right). Target EMG levels are indicated in center of figure.

panel shows the data for the 500 Hz tone burst. The solid diagonal line in each panel represents the condition in which the actual EMG level equals the target level. The dashed lines fit to the data in each panel are linear functions of target EMG level. The equations that describe each function and the r^2 values are indicated in the lower right corner of each panel. For each stimulus, the EMG amplitude increased as a function of EMG target level. In addition, the variability of the EMG amplitude increased as a function of EMG target level, with the least variability obtained at the 30 μV and 50 μV target levels. The correlations between the target EMG level and the actual EMG amplitude were significant for both clicks (n = 55, $p \leq 0.0001$, $r^2 = 0.98$) and 500 Hz tone bursts (n = 55, $p \leq 0.0001$, $r^2 = 0.98$).

The individual P1-N1 VEMP amplitude is plotted as a function of EMG target level in **Figure 3**. The upper panel shows the data for the click stimulus, and the lower panel shows the data for the 500 Hz tone burst. For each stimulus, the VEMP amplitude increased as a function of EMG target level. The solid lines fit to the data in each panel are linear functions of target EMG level. The equations that describe each function and the r^2 values are indicated within each panel on the left side. The correlation between target EMG level and P1-N1 amplitude was significant for both clicks (n = 55, $p < 0.0001$, $r^2 = 0.56$) and 500 Hz tone bursts (n = 55, $p < 0.0001$, $r^2 = 0.71$).

Separate 2 × 5 (ear × target EMG level) repeated measures analyses of variance (ANOVAs) were computed for the click and 500 Hz tone burst amplitude data depicted in **Figure 3**. The main effect of ear was not significant for either the click data [$F(1,9) = 0.03$, $p = 0.86$] or the 500 Hz tone burst data [$F(1,9) = 0.01$, $p = 0.93$], whereas the main effect of target EMG level was significant for both stimuli [clicks: $F(4,9) = 29.73$, $p < 0.0001$; 500 Hz tone bursts: $F(4,9) = 78.94$, $p < 0.0001$]. Although a division of the subject pool into two groups (i.e., left ear and right ear) may have decreased the power of the ANOVA, the p-values for the main effects were robust.

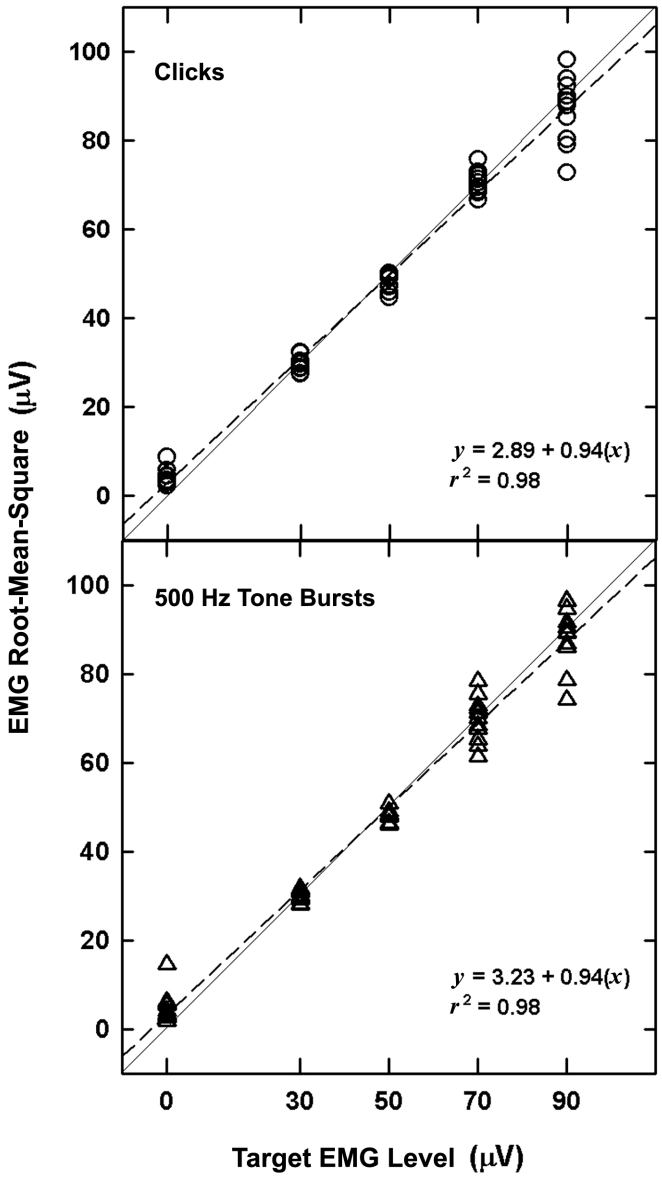

Figure 2.
Bivariate plots of individual EMG amplitude as function of target EMG level for clicks (upper panel) and 500 Hz tone bursts (lower panel). Solid diagonal line in each panel represents condition in which actual EMG level equals target level. Dashed lines fit to data in each panel are linear functions of target EMG level. Equations that describe each function and r^2 values are indicated in lower right corner of each panel.

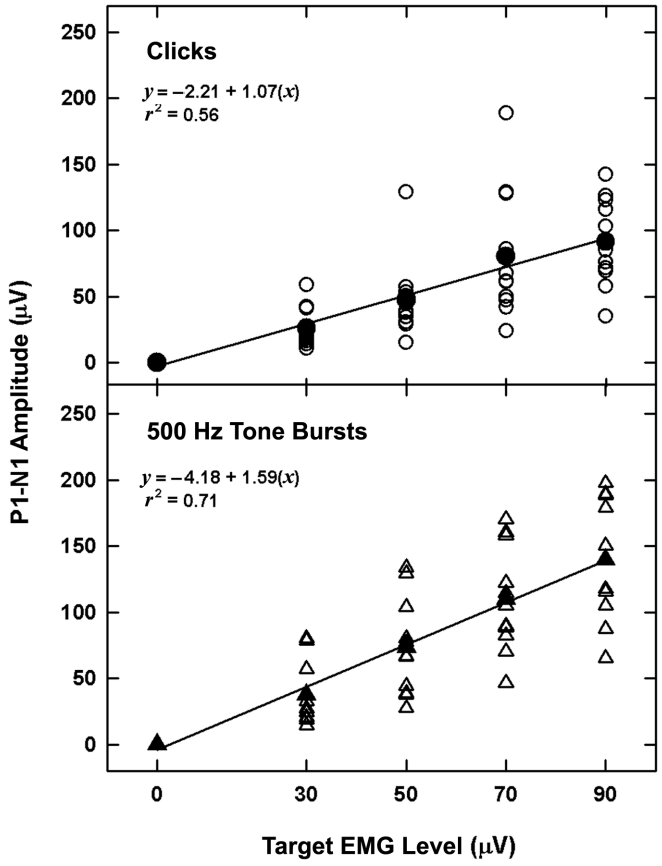

Figure 3.
Individual P1-N1 VEMP amplitude as function of EMG target level for clicks (upper panel) and 500 Hz tone bursts (lower panel). Solid lines fit to data in each panel are linear functions of target EMG level. Equations that describe each function and r^2 values are indicated within each panel on left side.

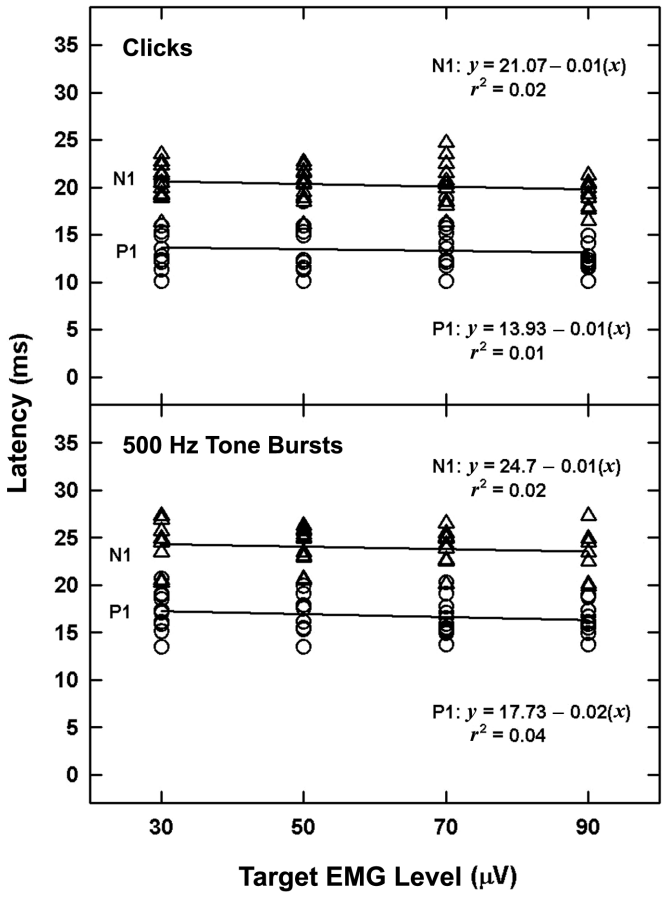

Figure 4.
Individual P1 and N1 latencies as function of target EMG level for clicks (upper panel) and 500 Hz tone bursts (lower panel). Solid lines fit to data in each panel are linear functions of target EMG level. Equations that describe each function and r^2 values are indicated within each panel for both N1 and P1.

Post hoc means contrasts for the click data indicated that all pairwise comparisons were significant ($p \leq 0.03$), with the exception of the 90 μV versus 70 μV ($p = 0.25$) and the 50 μV versus 30 μV ($p = 0.09$) comparisons. Post hoc means contrasts for the 500 Hz tone burst data indicated that all pairwise comparisons were significant ($p \leq 0.017$). No significant interaction effects were observed.

The individual P1 and N1 latencies are plotted as a function of target EMG level in **Figure 4** for clicks (upper panel) and 500 Hz tone bursts (lower panel). The solid lines fit to the data in each panel are linear functions of target EMG level. The equations that describe each function and the r^2 values are indicated within each panel for both N1 and P1. The correlation coefficients were not significant for either clicks or 500 Hz tone bursts ($p > 0.05$).

Separate 2 × 2 × 4 (ear × wave × target EMG level) repeated measures ANOVAs were computed for the click and 500 Hz tone burst latency data shown in **Figure 4**. The main effect of ear was not significant for either the click data [$F(1,9) = 0.29$, $p = 0.60$] or the 500 Hz tone burst data [$F(1,9) = 0.80$, $p = 0.40$], whereas the main effect of wave (P1 versus N1) was significant for both stimuli [clicks: $F(1,9) = 104.85$, $p < 0.0001$; 500 Hz tone bursts: $F(1,9) = 212.72$, $p < 0.0001$]. The main effect of target EMG level was not significant for the click data [$F(3,9) = 3.45$, $p = 0.08$]. Although the main effect of target EMG level was significant for the 500 Hz tone burst data [$F(3,9) = 7.43$, $p < 0.01$], the differences were not considered to be clinically significant, because the

maximum difference among the mean latencies of the four target EMG levels was 0.8 ms. In contrast to the amplitude functions in **Figure 3** that show a significant positive linear correlation between P1-N1 VEMP amplitude and target EMG level, the slopes of the latency functions are essentially unity. Post hoc means contrasts for the 500 Hz tone burst latency data indicated significant differences for two of the six pairwise comparisons: 90 μV versus 30 μV and 70 μV versus 30 μV ($p < 0.01$). No significant interaction effects were observed.

DISCUSSION AND CONCLUSION

The tonic state of the SCM m. is a critical parameter in the recording method of the VEMP [1,10,24]. Thus, controlling the level of tonic EMG would appear to be a prerequisite for the accurate interpretation of the VEMP. The present study demonstrates that the EMG target levels were achieved during unilateral activation of the SCM m. and that EMG amplitude increased as a function of target level. **Figure 2** shows that the EMG amplitude was least variable at the 30 μV and 50 μV target levels. This finding suggests that EMG target levels of 30 μV to 50 μV are optimal for clinical recording of the VEMP because tonic EMG levels are less variable at least for the age range (18–34 years) of the subjects included in this study.

It is noteworthy that the SCM m. was activated unilaterally with the subject sitting upright and the head turned to one side; however, other methods have also been used to activate the SCM m. during VEMP recording (e.g., bilateral SCM m. activation in the upright position, unilateral SCM m. activation in the supine position, bilateral SCM m. activation in the supine position) [1,20,32]. The 30 μV to 50 μV EMG target levels may not be optimal when the SCM m. is activated bilaterally or from the supine position. The method for activating the SCM m. in the present study was selected to activate unilaterally the SCM m. and allow optimal viewing of the tonic EMG target levels displayed on a computer monitor.

The VEMP amplitudes were positively correlated with tonic EMG level for both the click and tone burst stimuli (**Figure 3**). This finding is consistent with previous experiments that determined click-evoked VEMP amplitude is dependent on tonic EMG level [1,24,25]; however, the observed increase in amplitude as a function of EMG target level for tone burst stimuli has not previously been reported.

VEMP amplitude was larger for 500 Hz tone bursts than for clicks at each EMG target level. Previous studies comparing responses to tone burst and click stimuli have shown inconsistent findings. Welgampola and Colebatch determined that tone-evoked responses required lower stimulus levels than click-evoked responses to produce equivalent VEMP amplitudes [31]. Similarly, Akin et al. demonstrated that tone-evoked VEMP amplitudes were larger than click-evoked amplitudes when comparisons were made at equal peak SPLs [32]. In contrast, Cheng et al. reported that VEMP amplitudes for clicks were larger than VEMP amplitudes for 500 Hz tone bursts when each stimulus was presented at 95 dB nHL [33]. The contradictory findings are likely due to calibration differences resulting in different spectrum levels across experiments.

Because the VEMP amplitude is a parameter used to interpret the response clinically, the influence of tonic EMG level on the VEMP amplitude is important. Thus, controlling the level of tonic EMG is likely a prerequisite for the accurate interpretation of interaural VEMP amplitude differences. The following two techniques have been proposed to control/monitor the tonicity of the SCM m. during VEMP recording: (1) direct control of the magnitude of tonic neck muscle activity through monitoring of the amplitude of the rectified EMG at a constant target level [24,28,31] and (2) calculation of a corrected reflex amplitude by division of the peak-to-peak amplitude by the mean rectified EMG level [1,23,30,32]. It is unclear if clinical interpretation of the VEMP is influenced by the technique used to control the tonic SCM m. activity.

Typically, clinical assessment does not include tests of otolith function. Rather, electronystagmography and rotational tests assess the horizontal semicircular canal (one of five vestibular sensory organs). VEMPs may prove to be a useful clinical test to assess saccular and/or inferior vestibular nerve function, and the identification of saccular involvement may have implications in the management of patients with balance disorders. Vestibular rehabilitation therapy (VRT) exercises are typically based on principles of vestibular adaptation of semicircular canal input. If otolith organ involvement is identified, then VRT exercises designed to stimulate otolithic adaptation may be more effective for managing a patient's symptoms.

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