EMG Normalization Eliminates cVEMP Amplitude Asymmetries in Normal Subjects

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Introduction

The vestibular evoked myogenic potential (VEMP) is a short latency, sound-evoked sonomotor response evoked by acoustical stimulation of the vestibular system. This evoked response was first described by Colebatch and colleagues (e.g. 1992, 1994) and can be recorded from a number of muscles, the most common of which is the sternocleidomastoid muscle (SCM). There is general agreement that the peripheral receptor for the VEMP is the saccule (Young et al., 1977; Colebatch et al., 1994; Murofushi et al., 1995; Murofushi et al., 1996; Bath et al., 1998) and that its sensitivity to high intensity acoustical stimulation is due to its close proximity to the middle ear. The integrity of the saccule can be assessed by measuring cVEMPs through mechanical, acoustical and galvanic stimulation (Fitzpatrick & Day, 2004), though acoustical stimulation of the saccule is used most commonly. Numerous studies have shown that the 500 Hz tone-burst stimuli can elicit cVEMP responses at the lowest intensity, as compared to tone-bursts at other frequencies or click-evoked stimuli (Akin & Murnane, 2001; Welgampola & Colebatch, 2001b).

Once the saccular macula has been translated by an acoustical stimulus of sufficient intensity, the saccular afferents are depolarized and these electrical signals are routed through the inferior vestibular nerve to the inferior and medial vestibular nuclei. From there, the signal is routed to the descending medial vestibulospinal pathway to the nucleus of the spinal accessory nerve to cranial nerve XI to terminate on the SCM. (Fitzgerald et al., 1982; Buttner-Ennevera, 1999). If the SCM is being tonically contracted when the saccule is stimulated, a stimulus-evoked reduction in the electromyographic (EMG) activity will occur. When this reduction of stimulus-induced EMG activity is recorded using surface electrodes and then signal-averaged,

the resulting evoked potential waveform consists of a prominent positive wave that occurs at approximately 13 msec (i.e. referred to as P13) and this is followed by a negative-going wave that occurs at approximately 23 msec (i.e. that is referred to as N23).

Several different SCM activation procedures have been described in previous research: 1) lifting the head 30 degrees from the supine position to activate both SCMs, 2) turning the head toward the contralateral ear while in a sitting position, 3) lifting the head from a supine position to push against a blood pressure cuff, and 4) turning and lifting the head toward the contralateral ear from a supine position. All of these different SCM activation mechanisms create EMG activity in the production of a cVEMP (Rosengren et al., 2010). In a comparison of different activation techniques, Wang and Young (2006) found that activation by turning and lifting the head toward the contralateral ear in the supine position produced a cVEMP in 100% of subjects. Alternatively, rotation of the head in the sitting position produced a cVEMP in only 70% of patients; cVEMPs also had smaller amplitudes when compared to supine activation. Generally, turning and lifting the head from the supine position is considered to be the optimal activation technique for obtaining the most reliable and repeatable cVEMP response (Zapala & Brey, 2004; Rosengren et al., 2010; Isaacson et al., 2006; Isaradisaikul et al., 2008).

The determination that a cVEMP is abnormal is based on absolute and interaural latency differences, and interaural amplitude asymmetry. Interaural asymmetry measurements of at least 47% suggest the presence of a vestibular impairment affecting either or both the saccule and/or inferior vestibular nerve (McCaslin et al., 2013). It is well accepted that cVEMP amplitude has a monotonic relationship with EMG level (Lim et al, 1995). Since amplitude measures are dependent on the magnitude of tonic EMG, it is important that EMG levels are carefully

monitored or considered in the interpretation of cVEMP asymmetry measures (Akin & Murnane, 2008).

There are two methods that can be used to ensure that the tonic EMG activity of the SCM is equivalent from one side to the other: 1) through patient self-monitoring and 2) through mathematical normalization. The first method is accomplished when patients take an active role in monitoring their own EMG during a cVEMP assessment. With this method, patients use visual feedback from an oscilloscope to ensure that a constant EMG level is maintained. Patients may also make determination about how their EMG compares to the desired target level. Multiple studies have found this method minimizes variability in EMG amplitude (Vanspauwen et al., 2006; Eleftheridaou & Koudounarakis, 2011). Akin et al. (2004) also used patient self-monitoring to study the use of EMG targets in determining patients' ability to achieve prescribed levels. Eleven young subjects (ages 18-34 years) with normal hearing sensitivity produced cVEMP responses following unilateral activation of the SCM (i.e. head turned toward the contralateral ear in a sitting position). Responses were obtained with tonic EMG target levels between 0 μV and 90 μV. Results revealed that cVEMP amplitude increased with EMG target levels. Because less intersubject variability was observed at lower target levels, the authors recommended that EMG targets of 30μV and 50μV be utilized for monitoring patient EMG. Of note, McCaslin et al., (2013) found that when the supine optimal activation method is used, young normal subjects produced mean EMG levels of approximately 300 μV without the use of targets.

The second way that clinicians and researchers may account for differences in tonic EMG is through the use of EMG normalization or rectification. cVEMP amplitudes may be "normalized" by attempting to remove the influence of EMG on cVEMP amplitudes.

Normalization ratios are calculated by measuring tonic EMG activity in 20-100 ms blocks preceding the stimulus onset. The mean value is then calculated, which represents the overall EMG level that was recorded during signal averaging. Next, the mean EMG value is divided into the amplitude voltage value for each data point that represents the cVEMP (McCaslin et al., 2013). Interaural asymmetries in amplitude can vary greatly, even in normal subjects; however, there is conflicting research regarding the clinical usefulness of normalization (Bogle et al., 2013; McCaslin et al., 2013). Lee and colleagues (2008) studied 22 young, normal subjects with cVEMP testing using the optimal activation method. cVEMP amplitudes were normalized by using the method described above, and all subjects produced normal, symmetrical cVEMP responses bilaterally. When normalization was applied, data showed that the upper limit of the interaural amplitude asymmetry value was significantly reduced when compared to the uncorrected data.

In the end, past research has suggested that patient EMG monitoring and amplitude normalization methods can ensure that observed asymmetries in cVEMP responses are due to vestibular impairment rather than an artifact stemming from side to side differences in tonic EMG activity. Though the importance of controlling EMG is well-documented, several aspects relating to EMG's influence on cVEMP values are unknown. The relationship between normalization, different EMG levels, and interaural asymmetry has not been investigated significantly. Accordingly, the purpose of this study was to address the effects of EMG normalization on interaural asymmetry.

Methods

This investigation was approved by the Institutional Review Board (IRB) of Vanderbilt University (IRB# 090841), and full consent was obtained from each participant. All subjects were recruited through the Vanderbilt community. Subjects reported no history of vertigo, or otologic /neurologic disease. The study included 24 subjects age 21-29 [mean = 24.2].

All subjects underwent audiological evaluation on the same day that cVEMP testing was completed. Participants demonstrated thresholds within normal limits (< 20 dB without air bone gaps) from 250-8000 Hz. Tympanometry and stapedial reflex threshold testing at 500 and 1000 Hz were normal. Prior to testing, a cVEMP screening examination was completed to ensure that participants could generate at least 50 μV RMS tonic EMG activity from both the left and right SCM muscles.

Electrodes were applied to the surface of the skin using a conventional clean electrode preparation technique with impedances < 5000 Ω and interelectrode impedances < 3000 Ω. The non-inverting electrode was applied to the middle third of the SCM bilaterally. A reference was applied to the chin, and the ground electrode was placed at Fpz.

Subjects were situated in a supine position with the head elevated 30 degrees. Subjects were instructed to lift and rotate their head away from the ear being stimulated for unilateral SCM activation. All testing was completed using the Interacoustics Eclipse evoked potential system. cVEMPs were obtained using a single-channel recording in accordance with the ear being stimulated. A band-pass filter of 15-500 Hz was used with amplification gain of 5000x. Artifact rejection was disabled. The recording epoch began 10 ms before the stimulus onset and continued for 90ms post-stimulus for a total of 100 ms epoch. A total of 150 single samples per averaging block were collected. Air-conducted stimuli were presented at 500 Hz at 95 dB nHL

with a Blackman gating function (Jacobson & McCaslin, 2007). The 500 Hz tone burst had a 2 cycle rise time, 1 cycle plateau and a 2 cycle fall time, and the stimulus was presented at a rate of 5.1/sec through Ear Tone insert earphones. An oscilloscope providing visual feedback of EMG activity was presented on a large screen in front of the subject.

Pilot testing was completed prior to this investigation with 9 subjects age 20-24. Data was gathered to determine the value of appropriate EMG targets for producing cVEMPs using the supine activation methods. Subjects were initially asked to produce their maximal EMG activity. Before formal testing was completed, the patient was encouraged to use the oscilloscope to produce the most EMG activity as possible while still maintaining proper positioning. Then, 80% of the maximum EMG level was calculated to create a high target level that would be sustainable and comfortable for patients (Vanspauwen, et al., 2006). This value was termed a "high EMG target level." Next, 67% of the high EMG target level value was calculated to establish a "medium EMG target value." Finally, 33% of the high EMG target level was calculated to establish a "low EMG target value." These values were unique to each patient and varied across ears in accordance with the maximum EMG. Results of P13-N23 amplitude and EMG results are displayed in Figure 1.

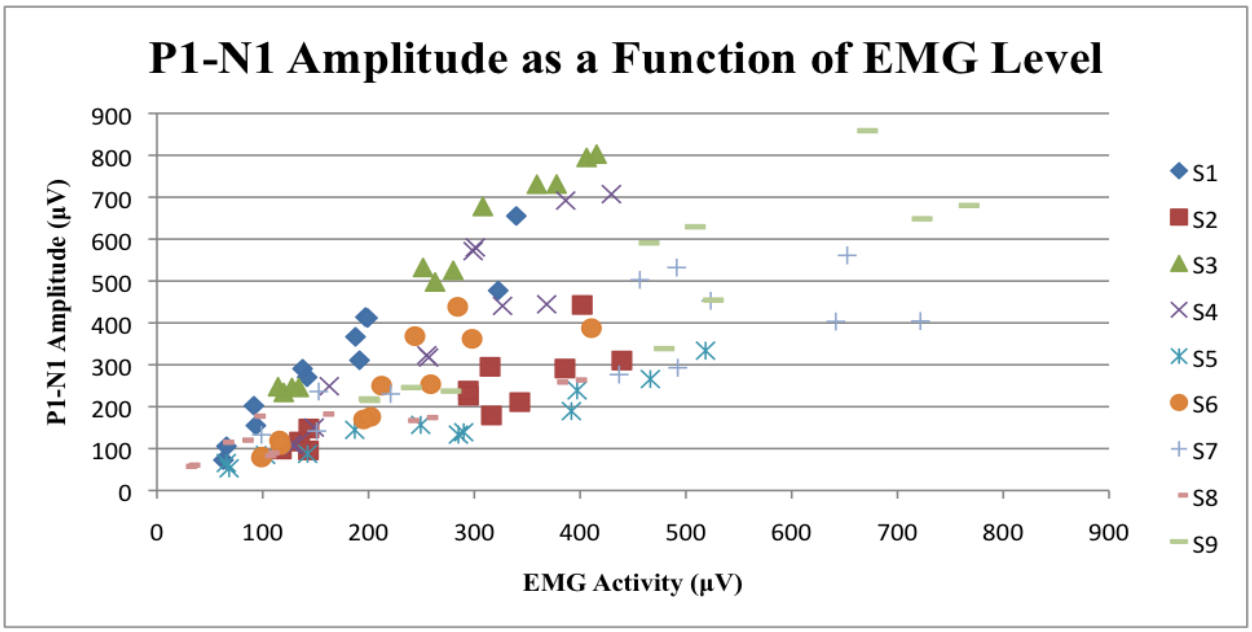

Figure 1. P13-N23 cVEMP amplitude as a function of target EMG level.

Participants' subjective maximal SCM activation produced EMG levels from 236.8 µV-951 µV (mean = 489.3), with all participants able to match their individual targets from 72.65 (low target) to 760.8 µV (high target).

As a result of this pilot data, EMG target levels were set for the present study at 100 µV, 200 µV, 300 µV, and 400 µV. Patients briefly practiced creating the target level prior to completing the two runs for each EMG level. The order of presentation and EMG level were counterbalanced using a Latin Square design. Each averaging block was replicated at least one time and participants were given breaks as needed. P13 latency, P13-N23 amplitude, and mean and standard deviation of EMG were recorded for each condition. Interaural amplitude asymmetry was calculated using the following formula:

$$\frac{\text{(Amplitude right cVEMP-Amplitude left cVEMP)}}{\text{(Amplitude left cVEMP + Amplitude right cVEMP)}} \times 100$$

A cVEMP asymmetry ratio of greater than 47% was considered significantly asymmetric (McCaslin et al., 2013). EMG normalization procedures were conducted by collecting the tonic EMG activity during the 100ms pre-stimulus onset. The full-wave rectified EMG was sampled (30,000 Hz) during this period and the mean value of these samples was calculated. After signal averaging, the mean individual RMS values from the pre-stimulus interval were calculated. This value represented the estimate of the average tonic EMG level that occurred during signal averaging, and this value was used to normalize the cVEMP. The mean RMS value was divided into the amplitude value of each data point in the cVEMP to produce a "normalized" cVEMP waveform.

Results

A general linear model was used for data analyses using SPSS version 21.0 (SPSS, Inc., Chicago, IL). An initial repeated measures analysis of variance (ANOVA) was conducted where cVEMP amplitude, RMS amplitude, and separately cVEMP latency, served as dependent variables, and ear served as the independent variable. The results of these analyses showed that there was not a significant main effect for ear (i.e. $p < 0.05$). Because no ear effects were calculated, data generated from the left and right ears were averaged together for subsequent

analysis (e.g. P13 latency, P13-N23 amplitude, and RMS of pre-stimulus EMG). The collapsed data were then analyzed using repeated measures ANOVAs in an attempt to detect tonic EMG level dependent differences separately for P13 latency, P13-N23 peak-to-peak amplitude and mean RMS amplitude. Responses collected following stimulation of the left and right side were used to calculate interaural amplitude asymmetry for subsequent analyses. A one-way multivariate analysis of variance (MANOVA) was used to determine if interaural amplitude asymmetries were significantly reduced when amplitude normalization techniques were employed. Any differences found were investigated using unplanned linear contrasts.

Effects of Tonic EMG Level on Uncorrected P13-N23 Amplitude

A repeated measures ANOVA with a Greenhouse-Geisser correction determined that mean uncorrected P13-N23 amplitude differed statistically as a function of the EMG target levels ($F_{(2.1, 49.8)} = 34.2$, $p < .000$). Post hoc tests using the Bonferroni correction revealed that mean P13-N23 amplitudes differed significantly ($p < 0.05$) across tonic EMG target levels from 100-300 μV. P13-N23 amplitude was shown to increase with increases in EMG target level. Specifically, EMG targets at 100, 200, 300 μV RMS produced significantly larger cVEMP amplitudes respectively ($p < .05$). However, although significant differences existed between P13-N23 amplitudes recorded using 100, 200 and 300 μV targets, no significant differences in mean amplitude were observed between the P13-N23 recorded using 300 and 400 μV EMG target levels. That is, the results of the analysis suggested that P13-N23 amplitude saturated for EMG targets between 300 and 400 μV. Figure 2 illustrates uncorrected P13-N23 amplitudes plotted as a function of target EMG level.

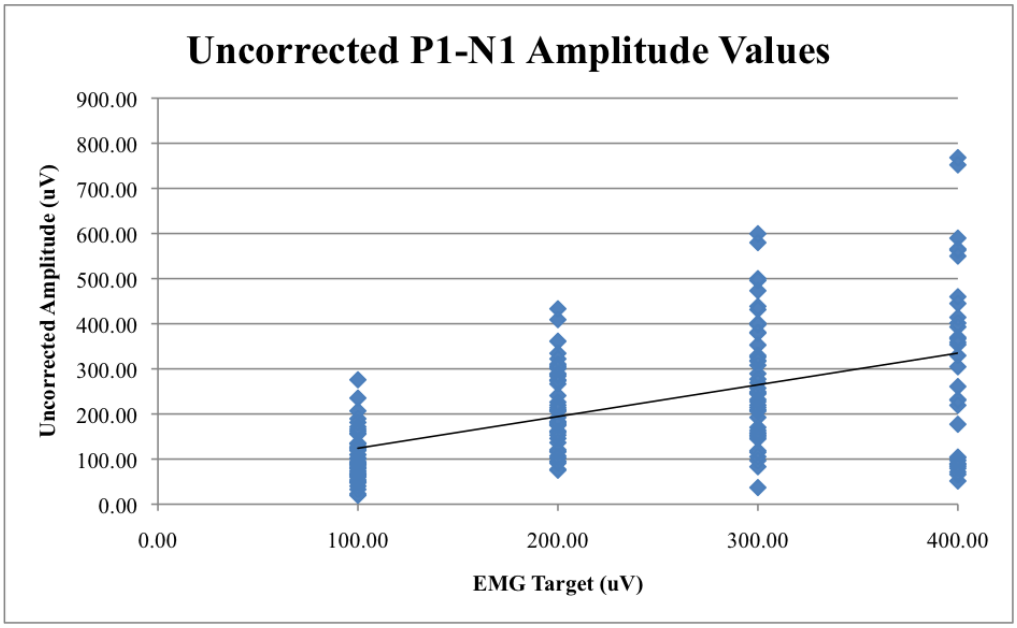

Figure 2. Uncorrected amplitude (uV) compared to EMG target level

P13-N23 amplitude increased as a function of EMG target level. The line represents the linear regression analysis of target EMG level for the uncorrected data. A significance level was set at p <.05. The correlation between target EMG level and P13-N23 amplitude was significant for uncorrected P13-N23 peak-to-peak amplitude (N =145, p <0.00, $r2$ = 0.45).

Effects of Tonic EMG Level on Corrected P13-N23 Amplitude

The results of a repeated measures ANOVA with a Greenhouse-Geisser correction showed that following amplitude correction, mean P13-N23 amplitude did not differ significantly using increasing EMG target levels ($F_{(1.682, 40.360)}$ = 1.064, p=.344). Post hoc tests using the Bonferroni correction revealed that when amplitude correction was applied, there were no significant differences in P13-N23 amplitude between any of the EMG target levels (p<.05).

Figure 3 illustrates amplitude normalized P13-N23 waveform mean amplitudes plotted as a function of target EMG level.

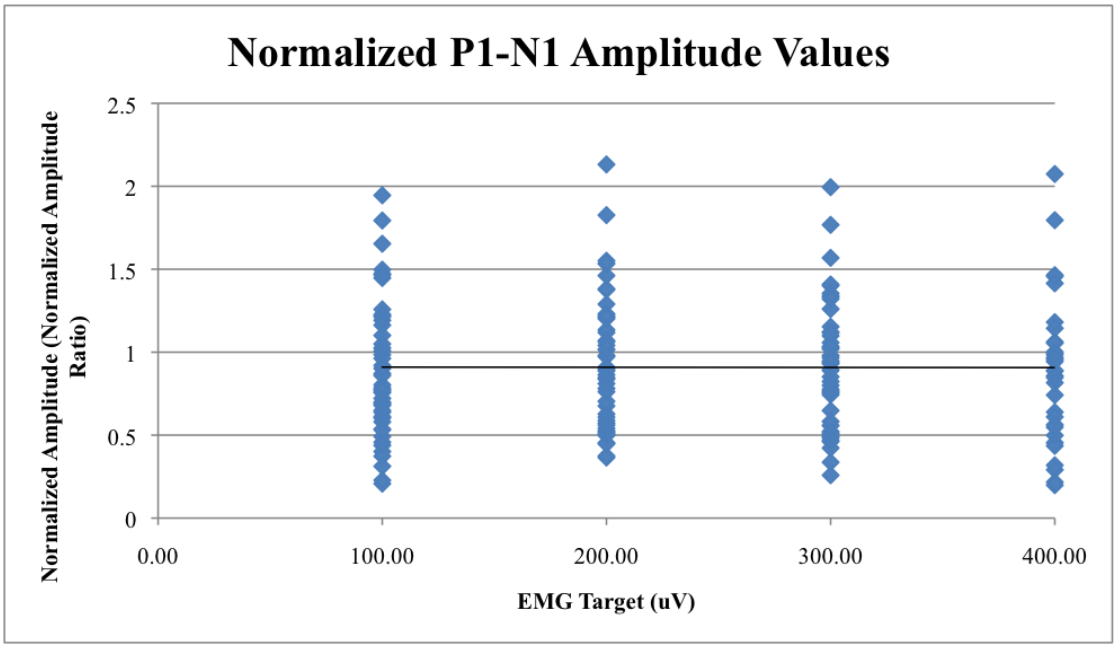

Figure 3. Normalized amplitude (normalized amplitude ratio) compared to EMG target level (uV).

Following amplitude correction, P3-N23 amplitude no longer increased significantly as a function of EMG target level. The correlation between target EMG level and P13-N23 amplitude was not significant for normalized P13-N23 peak-to-peak amplitude (N =145, $p <$ 0.864, $r2$ = 2.060E-4).

Effects of Tonic Level of EMG on P13-N23 Peak Latency

A repeated measures ANOVA (sphericity assumed) was conducted to determine whether EMG target level had a significant effect on P13-N23 peak-to-peak latency. The result of this analysis was not significant ($P > 0.05$). That is, P13-N23 latency did not increase or decrease

with changes in EMG target level, regardless of age.

Effects of Tonic Level of EMG on RMS Amplitude

A repeated measures ANOVA with a Greenhouse-Geisser correction determined that mean RMS amplitude differed between all EMG target levels ($F_{(1.91, 20.99)} = 326.12$, $p < .000$). Post hoc tests using the Bonferroni correction revealed that mean EMG RMS amplitude increased significantly with increases in EMG target level. That is, as the EMG target increased (i.e. from 100, to 200, to 300, to 400 μV) RMS pre-stimulus EMG estimates increased accordingly (p<.05; Figure 4).

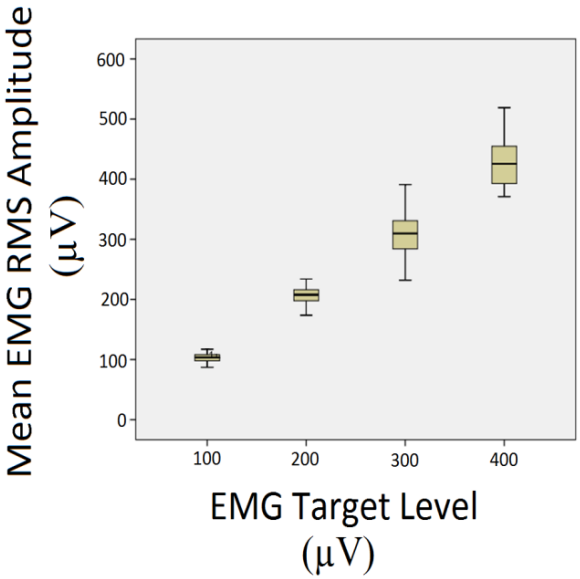

Figure 4. Mean EMG RMS amplitude (uV) compared to target EMG target level (uV).

Effects of Amplitude Normalization on Interaural Asymmetry

The primary aim of the current study was to determine whether amplitude normalization reduced interaural asymmetry differences in the presence of progressively asymmetrical levels of tonic EMG. To accomplish this, interaural asymmetries were compared at four tonic EMG target levels (e.g. P13-N23 amplitude at 100 µV, 200 µV, 300 µV, 400 µV) in both the uncorrected and corrected conditions. If amplitude correction did not neutralize the effects of varying EMG magnitudes, then the differences in P13-N23 amplitude between the corrected and uncorrected conditions would not be significant.

Accordingly, a one-way multivariate analysis of variance (MANOVA) was conducted to test the hypothesis that there were significant differences between uncorrected and normalized interaural asymmetry. For this analysis, interaural asymmetry served as the dependent variable and recording condition (i.e. uncorrected or amplitude normalized) served as the independent variable. The result of this analysis showed that there was a significant main effect for condition (i.e. normalized versus uncorrected; $F_{(10,10)} = 10.65$, $p < .001$). Post hoc testing (Bonferroni adjusted) showed that there were significant amplitude differences for the mean comparisons between 100 µV versus 200 µV EMG targets ($p < 0.05$), 100 µV versus 300 µV EMG targets ($p < 0.01$), 100 µV versus 400 µV EMG targets ($p < 0.01$), and 200 µV versus 300 µV EMG targets ($p < 0.04$). The comparison of interaural amplitude asymmetries for 300 uV versus 400 uV targets was not significant (i.e. $p > 0.05$).

Discussion

The purpose of this study was to investigate the effect of amplitude normalization on interaural asymmetry in a group of young subjects. P13-N23 amplitude values for four different

EMG target levels (100 μV, 200 μV, 300 μV, and 400 μV) were measured and then used to calculate interaural asymmetries for each condition (e.g. 100 μV vs. 200 μV, 100 μV vs. 300 μV). The technique of comparing P13-N23 amplitude values at different target afforded us the ability to artificially create different degrees of EMG asymmetry. Interaural asymmetries using both uncorrected and corrected interaural asymmetry results were compared.

Interaural asymmetry values above 47% were considered abnormal and suggest the presence of a vestibular impairment. Even in young, normal adults, the possibility exists that SCM muscle contraction may vary significantly within the same patient. Consequently, cVEMP amplitudes may also differ within a patient, and if significantly asymmetrical, could potentially result in a false positive judgment (e.g. in that instance the clinician might errantly state that the patient had a unilateral saccular/inferior vestibular nerve impairment on the side with the smaller cVEMP). Additionally, impairments such as a cervical spine injury or other underlying neck weaknesses may impair a patient's ability to generate equivalent amounts of EMG within each sternocleidomastoid muscle.

For example, a patient with an impairment of the cervical spine could also have a unilateral saccular impairment. In such a case it is possible that a vestibular impairment could be masked if the patient were unable to tonically activate both SCMs equally. In this instance, the patient may be capable of activating the SCM on the side of the impaired end-organ vestibular end-organ at a level of approximately 300 μV. On the side of the healthy vestibular system, the patient may only be able to generate a tonic level of EMG of approximately 100 μV due to the impairment in the neck. In this instance, results of this assessment may fail to identify the vestibular impairment due to the influence of the asymmetrical EMG. Numerous studies have shown that cVEMP amplitude is highly correlated with tonic EMG activity (Akin et al., 2004;

Colebatch et al., 1994; Welgampola et al., 2003). In situations such as the one described above, the asymmetry in the level of EMG must be controlled to ensure that what is being evaluated is the vestibular system and not the level of EMG. When significant interaural asymmetries are observed clinically, it is imperative for examiners to determine whether the amplitude asymmetries are occurring due to asymmetries in EMG or represent evidence of an actual vestibular impairment. This investigation has shown that the magnitude of the tonic EMG can have a significant impact cVEMP amplitude. Further, our results demonstrate that amplitude normalization neutralizes spurious cVEMP interaural amplitude asymmetries. The downstream effect would be fewer misdiagnoses.

Results of the primary analyses were consistent with past studies, as subjects experienced increases in P13-N23 amplitude with increases in tonic EMG (Lim et al., 1995; Akin et al., 2004; Akin et al., 2011; Bogle et al., 2013). However, when amplitudes were normalized, no significant differences in mean amplitude were found across all target EMG levels, as expected. Previous studies have also found a linear relationship between tonic EMG and cVEMP amplitude , and similar findings were observed in this study for low-medium EMG levels (100-300 μV). When the relationship between EMG target level and uncorrected amplitude were assessed, a significant finding was observed at higher EMG levels. Results showed that there were no significant differences between the mean P13-N23 amplitude at 300 μV and 400 μV EMG target levels. Instead, cVEMP amplitude seemed to saturate for tonic EMG levels that exceeded 300 uV.

This same result was recently found by Bogle et al. (2013), who recorded cVEMPs at maximum, moderate, and minimal muscle contraction in 10 healthy subjects. In 8 of the subjects, after EMG reached a certain “threshold“ level, cVEMP amplitude increased and then

saturated shortly thereafter. This finding suggests that we may not fully understand the relationship between EMG and amplitude. The possibility of amplitude saturation in the cVEMP could also significantly affect the application of amplitude normalization. The calculation of amplitude normalization is based on the previously held assumption that the relationship between tonic EMG amplitude and cVEMP amplitude is monotonic. However, when the relationship changes between these mechanisms (i.e. high EMG levels result in lower-than-expected amplitudes), normalization may not be as effective. Though this is a consideration in the clinical utilization of normalization, it is also unlikely that patients maximally activate the SCM muscles during testing, producing very high EMG levels. Still, the precise impact of normalization in this situation is largely unknown at the present time.

When uncorrected mean interaural asymmetry data was analyzed, it was determined that there were two conditions in which asymmetries were considered to be abnormal, or greater than 47% (McCaslin et al., 2013). These occurred at 100 μV versus 300 μV (mean difference = 50.58%) and 100 μV versus 400 μV (mean difference = 56.24%). These two conditions also represent the largest differences in EMG target values, making a significant difference in amplitude an expected finding. The results of this analysis are displayed in Figure 5.

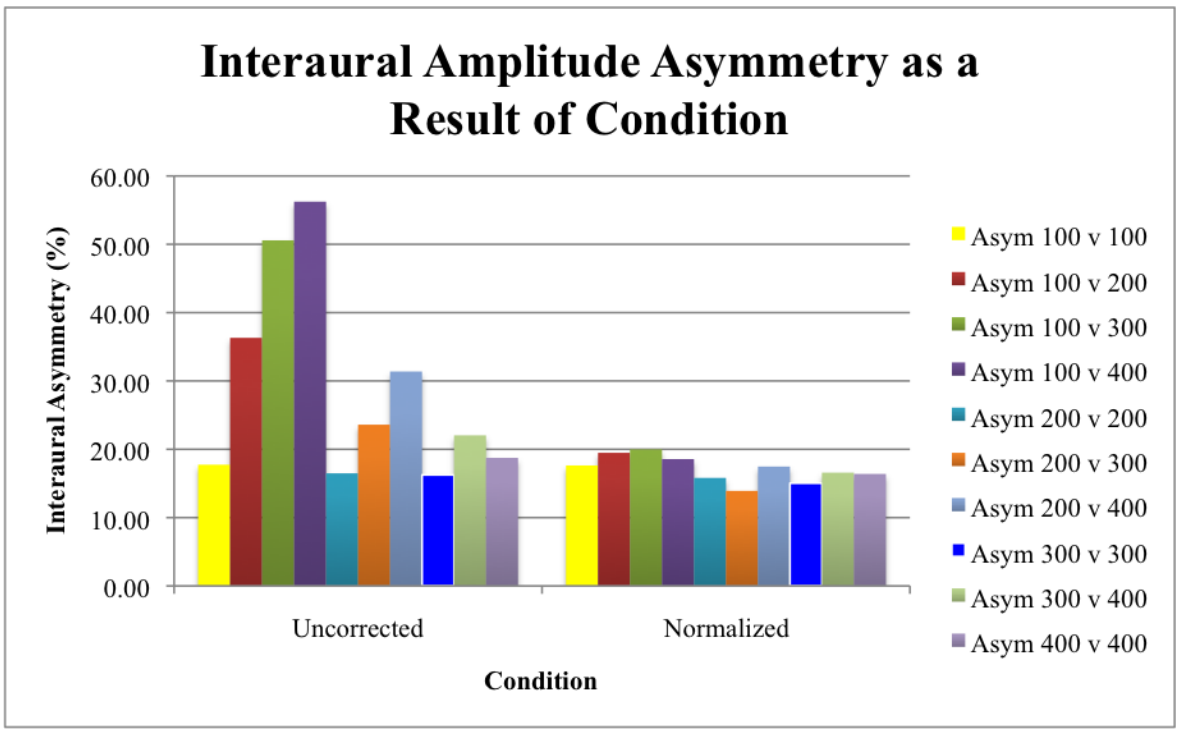

Figure 5. Uncorrected and corrected P13-N23 amplitudes as a result of condition. .

In conditions when the interaural asymmetries from the same amplitude targets were compared (i.e. 100 µV versus 100 µV, 200 µV versus 200 µV, etc.), differences in interaural asymmetry ranged from .1-2.4%. In other words, when EMG targets were exactly the same, normalization had a very small effect on interaural asymmetry, as expected. However, in conditions where EMG targets differed by a more extreme degree, the effect of normalization was substantial. For example, when P13-N23 derived from EMG targets of 100 µV versus 400 µV were compared, normalization reduced amplitude asymmetry values by an average of 37.6%. Following normalization, all interaural asymmetry measures were reduced to less than 20%. Therefore, it can be concluded that for a group of young, normal subjects, amplitude normalization was very effective in reducing asymmetry values when interaural EMG values were significantly different.

McCaslin et al. (2013) reported for a group of 97 subjects with a mean age of 31 years (sd = 16.33 years) of all ages, they were able to self-monitor their EMG, even without the use of a formal visual feedback system. As a result, the investigators reported that normalization did not provide significant benefit in reducing interaural asymmetry values. However, there is a subgroup of patients who are unable to generate symmetrical SCM muscle contraction. The results of the present investigation have suggested that these patients would benefit from the use of amplitude normalization. Additionally, in conditions when normalization was not necessarily needed (i.e. comparing 100 μV to 100 μV), it was found that normalization always kept interaural asymmetry values consistent or even reduced asymmetry values in a group of young, normal subjects.

Variation in P13-N23 amplitude also changed with EMG target level. At 100 μV, mean uncorrected amplitude varied from 19.7-189.5 μV, while at 400 μV, amplitude ranged from 79.54-768.2 μV. One explanation for this variability was the ability for subjects to meet EMG targets. At 100 μV, most participants were successful at meeting the EMG target. However, with increases in target level, the EMG became more variable, and subjects became less able to meet the targets.

Conclusion

To obtain the most accurate cVEMP measures and make appropriate interpretations, it is imperative that optimal recording techniques, with the least variability, are employed in the clinical setting. The results of the present study support the use of amplitude normalization to reduce the statistical upper limits of cVEMP interaural asymmetry measurement variables.

Analyses of P13-N23 amplitudes indicated that amplitude normalization reduced interaural amplitude asymmetry across all four target EMG conditions. In fact after normalization, all interaural asymmetry upper limits of normal were less than 19.97%. Therefore, it can be concluded that in a group of young, normal participants, normalization was very effective in reducing asymmetry values, even when interaural EMG values differed significantly. Further research is recommended to determine how normalization will influence interaural asymmetry in the presence of amplitude saturation at high EMG levels.

Though most patients in the clinic are able to symmetrically activate the SCM muscles without the use of feedback, patients with cervical pathologies or other weaknesses may not be able to produce symmetrical EMG levels. This EMG asymmetry may produce false-positive or false-negative results that could easily lead an examiner to misdiagnose the presence or absence of saccular/inferior vestibular nerve impairment. Thus, the use of amplitude normalization in cVEMPs is an integral part of the vestibular evaluation for these patients.

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