The Effect of Noise Exposure on the Cervical Vestibular Evoked Myogenic Potential

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Objective: The purpose of this study was to investigate the effects of noise exposure on the cervical vestibular evoked myogenic potential (cVEMP) in individuals with asymmetric noise-induced sensorineural hearing loss (NIHL).

Design: A cross-sectional observational study was used to compare cVEMP characteristics in 43 individuals with a history of noise exposure greater in one ear (e.g., the left ear of a right-handed rifle shooter) and asymmetric sensorineural hearing loss consistent with the history of noise exposure and in 14 age-matched controls. The characteristics of hearing loss were examined further for the noise-exposed participants with abnormal cVEMPs and the noise-exposed participants with normal cVEMPs.

Results: Thirty-three percent of the noise-exposed participants had abnormal cVEMPs, whereas cVEMPs were present and symmetrical in 100% of the age-matched controls, and cVEMP threshold was greater in the noise-exposed group than in the control group. Abnormal cVEMPs occurred most often in the ears with poorer hearing (or greater NIHL), and the noise-exposed participants who had abnormal cVEMPs had poorer high-frequency pure-tone thresholds (greater NIHL) and greater interaural high-frequency pure-tone threshold differences than the noise-exposed participants with normal cVEMPs.

Conclusions: These findings are consistent with previous studies that suggest that the sacculocollic pathway may be susceptible to noise-related damage. There is emerging evidence that the severity of NIHL is associated with the presence or absence of cVEMPs.

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INTRODUCTION

The effect of noise exposure is well established in both the animal and the human auditory systems (Henderson et al. 1976). In contrast, there is substantial evidence for noise-related damage to the animal vestibular system (Ylikoski 1987; Akdogan et al. 2009; Hsu et al. 2008; Fetoni et al. 2009), but the evidence for an effect of noise exposure on the human vestibular system is less clear. Most previous studies in humans have measured horizontal semicircular canal function after noise exposure (Man et al. 1980; Oosterveld et al. 1980; Golz et al. 2001); however, histological studies in animals have provided some evidence that the saccule may be more susceptible to noise-related damage than other vestibular sensory organs (McCabe & Lawrence 1958; Mangabeira-Albernaz et al. 1959). More recently, Perez et al. (2002) determined changes in the linear (otolith) vestibular evoked potential in rats exposed to noise whereas the angular (semicircular canal) vestibular evoked potential remained unchanged. Because vestibular evoked potentials are difficult to record in humans, cervical vestibular evoked myogenic potentials (cVEMPs) may provide a comfortable and reliable method to measure noise-induced changes in otolith function.

The cVEMP has been established as a clinical test of saccular and inferior vestibular nerve function (Colebatch 2001). cVEMPs are short latency electromyograms (EMG) evoked by high-level acoustic stimuli recorded from surface electrodes over the sternocleidomastoid (SCM) muscle during prolonged contraction. An ipsilateral reflex pathway mediates cVEMPs and includes the saccule, inferior vestibular nerve, brainstem vestibular nuclei, the descending medial vestibulospinal tract, the accessory nucleus, the accessory nerve, and the motoneurons of the SCM muscle. cVEMPs may be sensitive to noise-induced damage to the vestibular system, because the response originates predominantly from the saccule, which is in close proximity to the stapes footplate.

There is emerging evidence in animals and humans that noise exposure affects the VEMP. Hsu et al. (2008) determined that noise exposure causes a loss of VEMPs in guinea pigs; and, by using light and electron microscopy, they confirmed that the loss of VEMPs after noise exposure correlated with a saccular lesion. Similarly, Wang and colleagues have reported abnormal cVEMPs in humans after acute acoustic trauma (Wang et al. 2006) and in individuals with chronic bilateral noise-induced hearing loss (NIHL) (Wang & Young 2007). Asymmetric hearing loss has been described in individuals exposed to occupational noise and gunfire (Clark & Popelka 1989; Pirilä et al. 1991; Fernandes & Fernandes 2010), and there are reports that unilateral 4000 Hz notched audiograms are more common than bilateral 4000 Hz notched audiograms in Veteran and non-Veteran populations (Osei-Lah & Yeoh 2010; Wilson 2011). It is unclear whether asymmetric NIHL is related to asymmetric susceptibility to noise exposure or other factors (Pirilä et al. 1991; Job et al. 1998), and it is unknown whether asymmetric NIHL causes asymmetric loss of the cVEMP. We hypothesized that asymmetric NIHL may be associated with a greater prevalence of cVEMP loss in the poorer-hearing ear than in the better-hearing ear of individuals with a history of noise exposure. The purpose of this study was to investigate the effects of noise exposure on the cVEMP in individuals with asymmetric noise-induced sensorineural hearing loss.

PATIENTS AND METHODS

Subjects

Study participants were 43 individuals with a history of noise exposure and 14 age-matched controls. The noise exposure (NE) group comprised 42 male veterans and 1 female veteran (aged 25–63 years; mean = 52.0, SD = 9.9) with a history of military or occupational noise exposure that was greater in one ear (e.g., the left ear of a right-handed rifle shooter) and

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asymmetric sensorineural hearing loss consistent with the history of noise exposure. Asymmetric, noise-induced sensorineural hearing loss was defined as a noise notch at 4000 kHz of ≥35 dB HL in the poorer-hearing ear and a minimum interaural asymmetry of 20 dB HL at the affected frequencies. Exclusion criteria included neurological disease, middle ear pathology, open or closed head injury, and cervical injury. The control group comprised 14 age-matched controls (age 27–59 years; mean = 47.4, SD = 10.0) with normal-hearing sensitivity and a negative history of military or occupational noise exposure. Normal hearing was defined as pure-tone thresholds ≤20 dB HL (ANSI 2004) at the octave frequencies 250 to 8000 Hz. This study was approved by the East Tennessee State University/Veterans Affairs Institutional Review Board, and each participant signed an informed consent.

Procedures

Participants in the NE group were surveyed to determine the following: (1) the source of noise to which they were exposed; and (2) the presence or absence of dizziness. The sources of noise exposure included aircraft, heavy equipment/engines, firearms, artillery, explosives, radio/sonar, and the type of noise exposure was categorized as continuous, impulse, or both. The NE participants who reported dizziness were asked to categorize the quality of their symptoms as vertigo, imbalance, or lightheadedness.

The NE group underwent screening for horizontal semicircular canal function using slow harmonic acceleration (SHA) on a rotary chair (Micromedical, Schaumburg, IL). The participant was seated in a chair enclosed in a light-proof booth and the head was positioned upright so that the rotation occurred in the plane of both horizontal semicircular canals. The SHA stimulus was symmetrical angular chair displacement in clockwise and counterclockwise directions at frequencies ranging from 0.01 to 0.64 Hz. The participant was instructed to perform mental alerting tasks to prevent central suppression of the vestibulo-ocular reflex (VOR) response. The VOR response to angular acceleration (nystagmus) was averaged and compared with chair velocity for each frequency to obtain the response parameters of phase, gain, and asymmetry. Normal vestibular function was defined as slow-component eye velocity phase, gain, and asymmetry data within normal threshold values at 0.01 to 0.64 Hz.

Ocular motor and vestibular suppression tests were performed on NE participants as a measure of central vestibular function. Ocular motor tests included the following: (1) testing for pathologic nystagmus with changes in fixation and gaze position; (2) random saccades; (3) smooth pursuit of a target moving sinusoidally at several frequencies; and (4) optokinetic nystagmus performed in full field. Vestibular fixation suppression was evaluated during SHA at 0.16 Hz on the rotary chair or during caloric irrigation.

cVEMPs were recorded in both the NE and control groups. To record cVEMPs, the participants were seated upright and instructed to turn their heads to one side to activate unilaterally the SCM muscle. A two-channel recording of the evoked response was obtained with noninverting electrodes placed at the midpoint of the SCM muscle, inverting electrode sites at the sternoclavicular junctions, and the ground electrode on the forehead (Intelligent Hearing Systems, Miami FL). cVEMPs were recorded using a 500 Hz Blackman-gated tone burst (rarefaction onset, two-cycle rise-fall time with no plateau) presented to the ear ipsilateral to the activated SCM muscle via ER3A (Etymotic Research, Elk Grove Village, IL) insert earphones at a repetition rate of 5/sec. Tone burst stimuli were presented from 110 to 120 dB_{peak} SPL (80–90 dB nHL), and the stimulus level was increased to 125 dB_{peak} SPL in participants with no response recorded at 120 dB_{peak} SPL. The cVEMP threshold was defined as the lowest stimulus level that elicited a replicated, visually detectable P1-N1 wave. The cVEMP response was amplified (×5000) and band-pass filtered from 20 to 1500 Hz with a 12 dB/octave slope. Responses to 128 stimuli were averaged and 3 responses were obtained from each side at each stimulus level in randomized order.

To control for the influence of tonic EMG level on the cVEMP, EMG and cVEMPs were recorded simultaneously from each side of each subject (Akin & Murnane 2001). Subjects were seated upright and asked to rotate their heads to activate unilaterally the SCM muscle. A two-channel EMG recording was obtained with a stand-alone differential surface electrode (DE-2.1; DelSys, Inc., Boston, MA) placed at the midpoint of the SCM muscle, and a reference electrode was attached to the wrist. The EMG signals were amplified (×10,000), band-pass filtered from 20 to 450 Hz, and digitized at 1024 Hz via a portable EMG unit (Bagnoli-2; DelSys, Inc.). The subjects were provided visual feedback of their EMG amplitude via the computer monitor and software (EMGworks Signal Acquisition and Analysis Software; DelSys, Inc.). Each participant was instructed to maintain the rectified EMG root mean square amplitude at 50 μV during the recording of each cVEMP waveform. Participants were excluded from the study if they were unable to maintain rectified EMG root mean square amplitude at 50 μV.

The following measurements were calculated from the mean of three responses for each participant and compared across groups: (1) peak to peak cVEMP amplitudes (P1-N1) at 120 dB_{peak} SPL; (2) cVEMP thresholds; and (3) P1 and N1 latencies at 120 dB_{peak} SPL. Using P1-N1 amplitudes obtained at 120 or 125 dB_{peak} SPL, signed asymmetry ratios (ARs) were calculated for the NE and control groups (Manzari et al. 2010). Specifically, for the NE group,

$$AR = \frac{(\text{Poorer-Hearing Ear P1-N1} - \text{Better-Hearing Ear P1-N1}) \times 100}{(\text{Poorer-Hearing Ear P1-N1} + \text{Better-Hearing Ear P1-N1})}$$

For the control group,

$$AR = \frac{(\text{Left Side P1-N1} - \text{Right Side P1-N1}) \times 100}{(\text{Left Side P1-N1} + \text{Right Side P1-N1})}$$

The signed ARs vary from −100% to 100%. For the NE group, −100% indicates the cVEMP is absent from the poorer ear, whereas 100% indicates the cVEMP is absent from the better ear. For the control group, ARs can also vary, with negative values indicating that the P1-N1 amplitude is greater on the left side and positive values indicating that the P1-N1 amplitude is greater on the right side. Signed AR values near 0% indicate that P1-N1 amplitudes are symmetrical. Previous studies have indicated that normal individuals have ARs ≤40% (Akin & Murnane 2008); therefore, signed ARs ≤40% and ≥−40% were considered within normal limits.

Data Analyses

Two separate dependent-samples *t* tests were conducted to assess the significance of the interaural audiometric threshold differences for the normal controls and the NE group. Four separate one-way analyses of variance were conducted to determine the effect of group (control group, better ear of NE group, poorer ear of NE group) on cVEMP amplitude, threshold, P1 latency, and N1 latency. Absent responses were assigned an amplitude value of 0 μV; absent responses were not included in the latency analyses. An independent-samples *t* test was used to determine the significance of group (controls versus NE) on the signed AR. The NE group was subsequently divided into two subgroups (cVEMPs present versus cVEMPs absent) and two separate 2 × 6 (group × frequency) mixed-model analyses of variance were conducted to determine the effect of subgroup on the audiometric thresholds in the poorer ear and on the interaural audiometric threshold differences. Post hoc tests consisted of pairwise comparisons and the *p*-values were adjusted using the Bonferroni procedure to correct for experimentwise error. The degrees of freedom for main effects were corrected using Greenhouse-Geisser estimates of sphericity whenever Mauchly's test indicated that the assumption of sphericity had been violated. We considered $p < 0.05$ as statistically significant. All analyses were conducted using SPSS statistical software (version 14.0; SPSS Inc., Chicago, IL).

RESULTS

Figure 1 shows the means and ranges of the pure-tone thresholds for the better- and poorer-hearing ears of the NE group and the mean pure-tone threshold for the left and right ears of the control group. Dependent-samples *t* tests revealed that the difference between the thresholds for the better- and poorer-hearing ears of the NE group was significant ($t = -8.91$, $p = 0.000$). There was no significant difference between the right ear and the left ear thresholds for the control group ($t = -1.11$, $p = 0.27$).

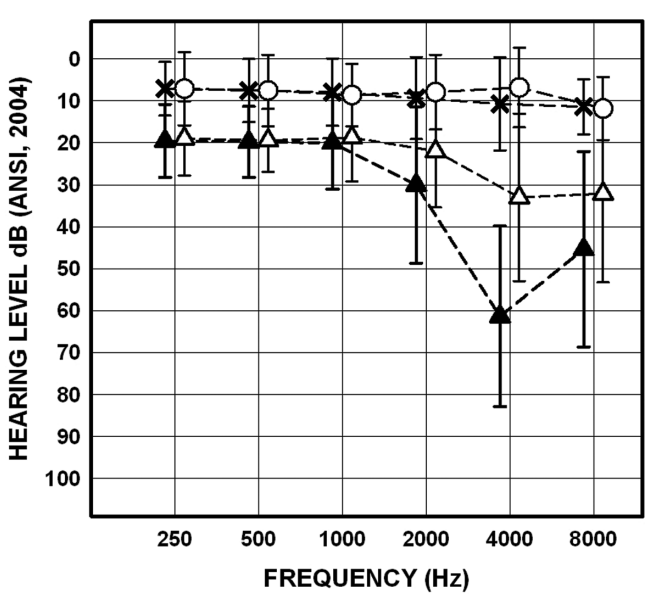

Fig. 1. Mean and SDs for pure-tone thresholds for the left (x) and right (o) ears of the age-matched control group and for the better-hearing ear and poorer-hearing ear of the NE group (indicated by open and filled triangles, respectively). NE, noise exposure.

Rotary chair testing revealed that all NE participants had normal phase, gain, and asymmetry during SHA, and these findings suggest normal horizontal semicircular canal/superior vestibular nerve function. Ocular motor function was within normal limits for all of the NE participants, and no subjects had spontaneous or positional nystagmus. Twenty-one of the 43 NE participants (49%) reported dizziness and were asked to specify the quality of the dizziness symptoms. Eight NE participants (35%) described vertigo, 15 (65%) described imbalance, and 20 (87%) described lightheadedness.

Table 1 summarizes the noise exposure history and cVEMP characteristics in the poorer- and better-hearing ears of the NE group. Most participants reported a history of exposure to both continuous and impulse noise. The latency and P1-N1 amplitude values shown in Table 1 are based on cVEMPs obtained at 120 dB_{peak} SPL. cVEMPs were present at 120 dB_{peak} SPL in 29 of the poorer-hearing ears and 37 of the better-hearing ears. For six participants, the cVEMPs were absent bilaterally at 120 dB_{peak} SPL; therefore, the stimulus level was increased to 125 dB_{peak} SPL. Four of the six participants with cVEMPS absent bilaterally had a cVEMP on at least one side at the higher stimulus level. cVEMPs were absent unilaterally for 11 of the 43 NE participants (26%), and cVEMPs were absent in the poorer-hearing ear for 10 of these 11 participants. The signed ARs in Table 1 were determined using the P1-N1 amplitudes obtained at either the 120 or the 125 dB_{peak} SPL stimulus levels.

Figure 2 shows signed ARs for 41 NE participants (circles) and the 14 control participants (triangles). Two of the 43 NE participants had cVEMPs absent bilaterally, and were excluded from this figure. The data points between the horizontal dotted lines indicate participants within normal limits. Twenty-nine of the 41 NE participants (71%) had normal AR (i.e., ≤40% and ≥−40%) indicating normal and symmetrical cVEMPs. Ten NE participants had −100% ARs indicating an absent cVEMP response from the poorer-hearing ear and a present cVEMP response from the better-hearing ear. One NE participant had a −51% AR indicating an abnormally reduced cVEMP amplitude from the poorer-hearing ear. One NE participant had an absent cVEMP from the better-hearing ear and a present cVEMP from the poorer-hearing ear. All of the control participants had cVEMPs present bilaterally and normal ARs. The results of an independent-samples *t* test indicated that the signed AR for the NE group (mean = −20.3%, SD = 51.2) was significantly greater than the signed AR for the control group (mean = −0.04%, SD = 9.2) ($t = 2.44$, $p = 0.009$).

Figure 3 shows the relationship between cVEMP asymmetry and hearing loss for the NE participants. A correlation analysis indicated that signed ARs were significantly related to the high-frequency pure-tone hearing average (2000, 4000, and 8000 Hz) in the poorer-hearing ear for the NE participants ($r = -0.346$, $p = 0.027$). The characteristics of the hearing loss in the poorer-hearing ears were examined further for the NE participants with abnormal cVEMPs (i.e., absent in poorer-hearing ear or signed AR ≥−40) and the NE participants with normal cVEMPs (i.e., present in poorer-hearing ear and AR <40%). Figure 4 shows that the pure-tone thresholds for the poorer-hearing ears were better for the NE participants with normal cVEMPs (open triangles) than the NE participants with abnormal cVEMPs (filled triangles). There was a significant main effect of frequency on pure-tone threshold ($F[2.1,80] = 96.7$, $p = 0.000$), and post hoc comparisons for

TABLE 1. Noise exposure history and cVEMP characteristics in the poorer- and better-hearing ears of the 43 NE participants

Participant No.	Noise History	P1 Latency (msec)		N1 Latency (msec)		P1-N1 (μV)		Threshold (dB_{peak} SPL)		Signed AR (%)
		Poorer	Better	Poorer	Better	Poorer	Better	Poorer	Better	
1	Both	NR	16.2	NR	22.0	NR	26	NR	120	–100
2	Both	NR	NR	NR	NR	NR	NR	125	125	–29*
3	Cont	16.8	15.6	23.0	21.2	35	56	115	115	–23
4	Cont	14.2	13.0	20.8	18.0	58	38	115	110	20
5	Impulse	NR	NR	NR	NR	NR	NR	NR	125	–100*
6	Both	15.4	15.6	20.8	22.6	57	65	115	110	–7
7	Cont	14.0	13.8	19.0	18.8	44	46	110	115	–2
8	Both	14.6	14.8	21.8	19.6	22	34	115	115	–22
9	Both	NR	15.0	NR	24.0	NR	152	NR	110	–100
10	Both	15.0	15.4	21.8	22.6	71	33	115	115	37
11	Both	16.4	15.2	23.4	23.2	39	24	120	120	24
12	Both	16.4	14.2	25.4	28.0	105	49	105	105	36
13	Cont	15.0	16.0	26.6	24.4	36	36	120	120	0
14	Both	14.2	14.0	21.6	20.4	76	134	110	110	–28
15	Both	14.4	16.2	20.6	26.6	40	26	120	120	21
16	Both	14.6	16.2	23.2	23.8	116	69	115	115	26
17	Both	17.2	14.2	22.2	22.2	41	85	120	115	–35
18	Both	NR	NR	NR	NR	NR	NR	NR	NR	NR
19	Both	16.2	15.8	22.4	23.4	120	87	115	110	16
20	Both	15.8	16.8	22.2	22.4	132	110	110	110	9
21	Both	16.8	15.4	22.2	23.0	164	226	115	115	–16
22	Both	14.6	15.0	23.0	23.0	181	93	115	115	32
23	Both	16.0	16.8	23.4	25.8	304	156	105	115	32
24	Both	NR	16.4	NR	23.0	NR	54	NR	115	–100
25	Both	15.2	13.6	24.2	22.8	26	80	120	110	–51
26	Both	15.8	17.2	25.8	26.6	324	348	110	105	–4
27	Cont	16.0	15.4	22.6	22.2	59	89	115	110	–21
28	Cont	NR	17.6	NR	23.4	NR	45	NR	120	–100
29	Both	NR	15.6	NR	23.0	NR	52	NR	120	–100
30	Both	16.0	15.4	27.0	26.0	132	249	115	105	–31
31	Both	NR	NR	NR	NR	NR	NR	NR	NR	NR
32	Both	NR	NR	NR	NR	NR	NR	125	125	–15*
33	Both	15.8	12.2	20.8	19.0	63	98	120	110	–22
34	Both	NR	13.6	NR	21.0	NR	105	NR	120	–100
35	Both	NR	NR	NR	NR	NR	NR	NR	125	–100*
36	Both	17.4	19.4	25.8	25.0	98	58	120	120	25
37	Cont	16.2	NR	21.8	NR	96	NR	120	NR	100
38	Both	15.0	15.6	22.8	26.0	224	205	110	115	4
39	Both	NR	18.0	NR	26.2	NR	65	NR	120	–100
40	Both	19.0	16.2	24.6	23.0	201	196	110	115	1
41	Cont	14.6	14.0	25.0	27.0	88	42	115	120	35
42	Cont	15.4	14.2	25.0	23.8	278	215	110	105	13
43	Impulse	NR	16.2	NR	25.0	NR	92	NR	115	–100

Latency and amplitude data are shown for cVEMPs obtained at 120 dB_{peak} SPL
**Signed AR obtained at 125 dB_{peak} SPL.*
NR, no response; Cont, continuous noise; Both, continuous and impulse noise; poorer, poorer-hearing ear; better, better-hearing ear; AR, asymmetry ratio.

frequency revealed that the thresholds at 2000, 4000, and 8000 Hz were significantly poorer than lower frequency thresholds ($p = 0.000$). The main effect of group on pure-tone threshold was not significant (F [1,38] = 2.7, $p = 0.11$). The group × frequency interaction was significant (F [2,80] = 4.05, $p = 0.02$), and post hoc comparisons revealed that pure-tone thresholds for the NE participants with abnormal cVEMPs were significantly poorer than pure-tone thresholds for the NE participants with normal cVEMPs at 2000 and 4000 Hz ($p < 0.04$); there were no significant differences in pure-tone thresholds at 250, 500, 1000, or 8000 Hz ($p > 0.25$).

Interaural pure-tone threshold differences are plotted in Figure 5 for the NE participants with abnormal cVEMPS and the NE participants with normal cVEMPs. The main effect of frequency on interaural differences was significant (F [5,190] = 45.09, $p = 0.000$), and post hoc comparisons revealed that the interaural threshold differences at 2000, 4000, and 8000 Hz were significantly greater than lower-frequency interaural differences ($p \leq 0.038$). The main effect of group on interaural pure-tone threshold differences was significant (F [1,38] = 7.44, $p = 0.010$) with greater interaural differences for the NE participants with abnormal cVEMPs than the NE participants with

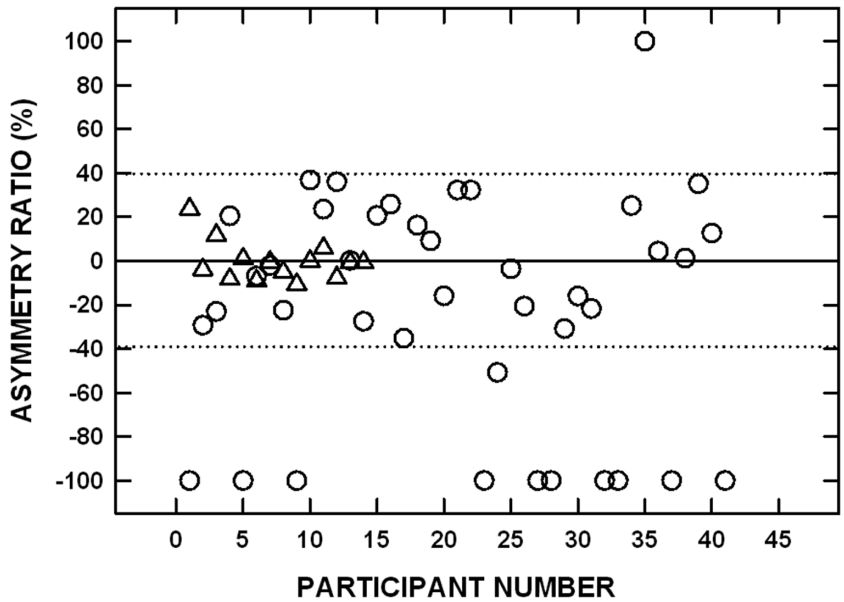

Fig. 2. Signed asymmetry ratios for 14 age-matched control (triangles) and 41 NE (circles) participants (two NE participants with cVEMPs absent bilaterally, ear not shown). The area between the dotted horizontal lines indicates asymmetry ratios within normal limits. NE, noise exposure; cVEMP, cervical vestibular evoked myogenic potential.

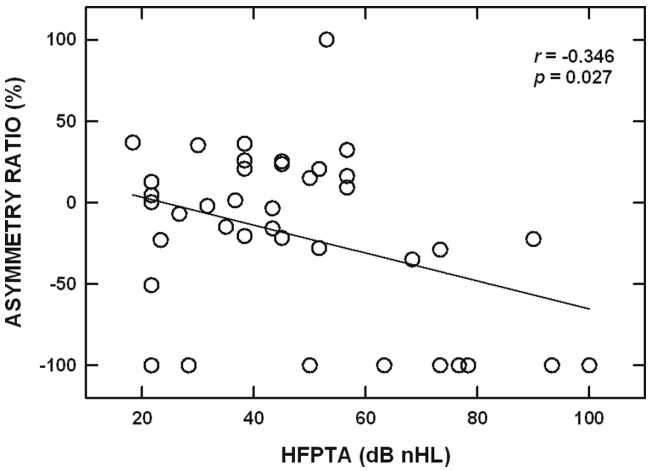

Fig. 3. Signed asymmetry ratios as a function of high-frequency pure-tone average (2000, 4000, and 8000 Hz) in the poorer-hearing ear of the noise-exposed participants. The solid line represents the linear regression analysis ($y = 20.41 - 0.86x$; $r^2 = 0.12$).

normal cVEMPs. The group × frequency interaction was significant ($F[5,190] = 2.51$, $p = 0.032$), and post hoc comparisons revealed that interaural differences for the NE participants with abnormal cVEMPs were significantly poorer than interaural differences for the NE participants with normal cVEMPs at 250, 2000, and 4000 Hz ($p \leq 0.018$); all other pairwise comparisons were not significant ($p \geq 0.264$).

The box plots in Figure 6 show individual and mean data for P1-N1 amplitude at 120 dB_{peak} SPL (top panel) and cVEMP threshold (lower panel) in the poorer and better ears of the NE group and for the mean of the right and left ears in the control group. All control subjects had cVEMPs present on both sides at 120 dB_{peak} SPL. The mean P1-N1 amplitude was 75 μV (SD = 87) for the poorer-hearing ear of the NE group, 82 μV (SD = 78) for the better-hearing ear of the NE group, and 79 μV (SD = 40) for the mean of the control group. There was no significant difference among groups for P1-N1 amplitude (F [2,111] = 0.102, p = 0.903). The mean cVEMP threshold was 117 dB_{peak} SPL (SD = 6) for the poorer-hearing ear of the NE group, 115 dB (SD = 6 dB) for the better-hearing ear of the NE group, and 111 dB (5 dB) for the control group. There was a significant main effect of group on cVEMP threshold (F [2,103] = 7.558, p = 0.001), and post hoc comparisons revealed that the difference in cVEMP threshold between the control group and either ear of the NE group was significant ($p \leq 0.014$). The difference in cVEMP threshold between the better- and poorer-hearing ears of the NE group was not significant ($p = 0.518$).

The box plots in Figure 7 show individual and mean latencies at 120 dB_{peak} SPL for P1 and N1 in the poorer and better ears of the NE group and for the mean of the right and left ears in the control group. The mean P1 latency was 15.7 msec (SD = 1.1) for the poorer-hearing ear of the NE group, 15.4 msec (SD = 1.4) for the better-hearing ear of the NE group, and 15.0 msec (SD = 0.7) for the control group. The mean N1 latency was 23.1 msec (SD = 2.0) for the poorer-hearing ear of the NE group, 23.3 msec (SD = 2.4) for the better-hearing ear of the NE group, and 22.1 msec (SD = 1.7) for the control group. There were no significant differences among the three groups for P1 latency (F [2,90] = 2.54, p = 0.09) or N1 latency (F [2,90] = 2.9, p = 0.06).

DISCUSSION

These results provide further evidence of noise-induced vestibular loss in humans. In this study, cVEMPs were recorded in 43 participants with asymmetric sensorineural hearing loss consistent with a history of noise exposure greater in one ear than the other, and cVEMPs were recorded in 14 age-matched controls. Fourteen of the NE participants (33%) had abnormal cVEMPs, whereas cVEMPs were present and symmetrical in 100% of the age-matched controls. Furthermore, cVEMP threshold was higher in the NE group than in the control group. These findings are consistent with previous studies in humans that have demonstrated effects of noise exposure on the cVEMP (Wang et al. 2006; Wang & Young 2007; Wu & Young 2009) and suggest that the sacculocollic pathway may be susceptible

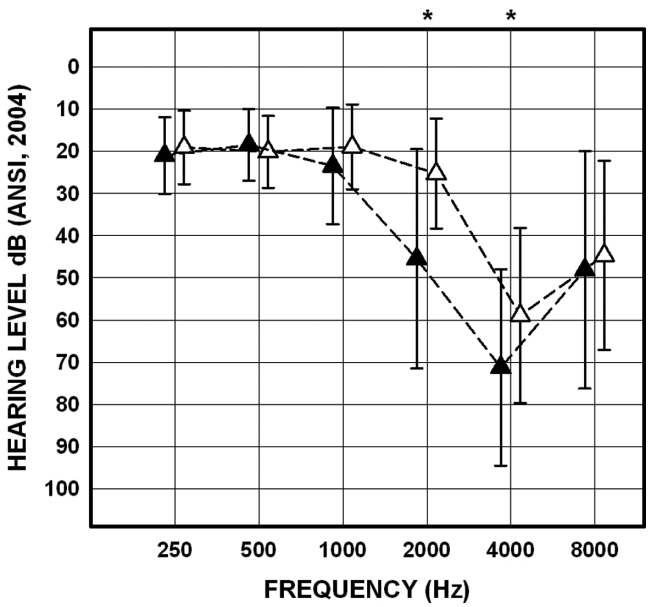

Fig. 4. Mean and SDs for pure-tone thresholds for the poorer-hearing ear of the NE participants with cVEMPs present (open triangles) and for the poorer-hearing ear of the NE participants with cVEMPs absent (filled triangles). Asterisks indicate significant post hoc comparisons. NE, noise-exposed; cVEMP, cervical vestibular evoked myogenic potential.

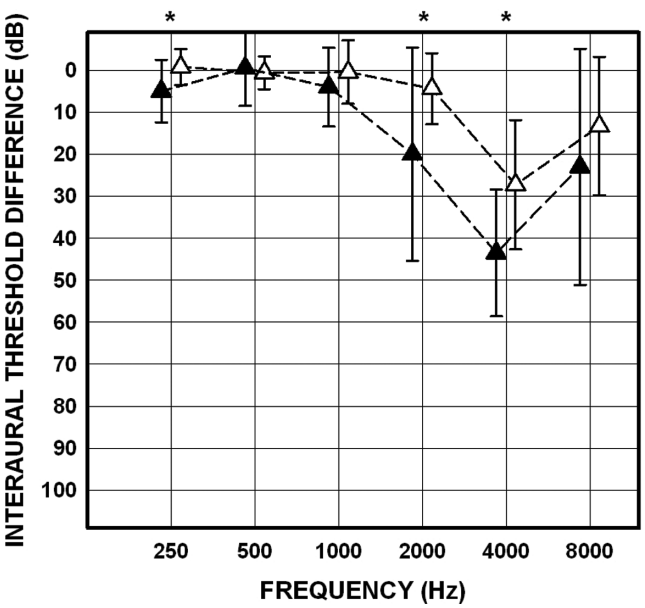

Fig. 5. Mean and SDs for interaural threshold differences for the NE participants with cVEMPS present (open triangles) and the NE participants with cVEMPs absent (filled triangles). Asterisks indicate significant post hoc comparisons. NE, noise exposure; cVEMP, cervical vestibular evoked myogenic potential.

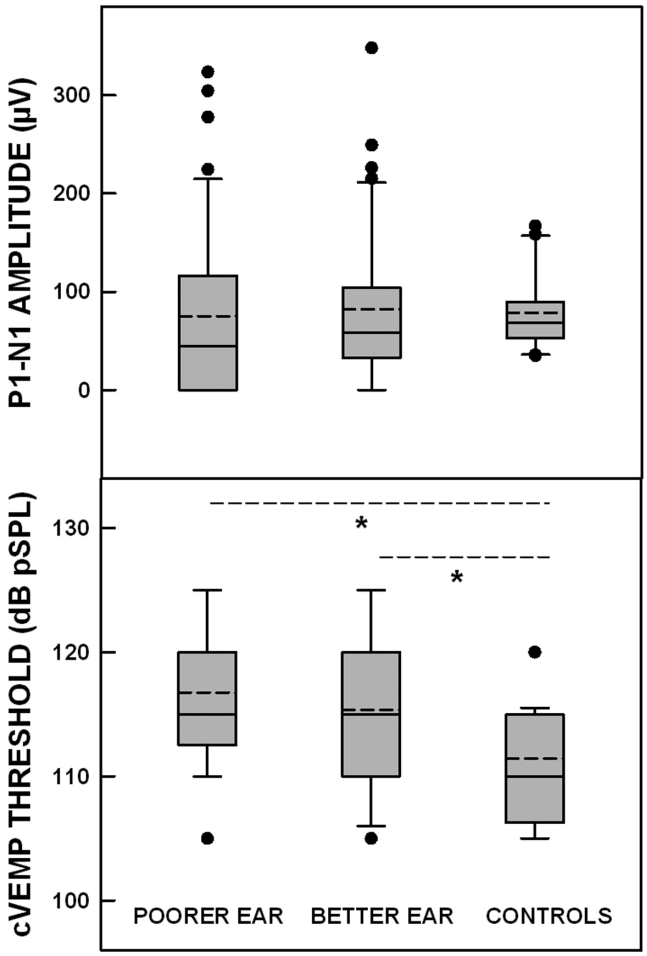

Fig. 6. Box plots of P1-N1 amplitude (top panel) and cVEMP threshold (lower panel) for the poorer-hearing ear and better-hearing ear of the NE group and for the mean of the right and left ears in the age-matched control group. The solid and dashed lines within the box mark the median and the mean, respectively. The boundary of the box closest to zero indicates the 25th percentile and the boundary of the box farthest from zero indicates the 75th percentile. The error bars above and below the box indicate the 90th and 10th percentiles, respectively. Outliers are represented by the solid circles. Asterisks indicate significant post hoc comparisons. NE, noise-exposed; cVEMP, cervical vestibular evoked myogenic potential.

to noise-related damage. Hsu et al. (2008) correlated the loss of VEMPs after long-term noise exposure in guinea pigs to morphological changes in the saccule. Specifically, the cell bodies of hair cells in the saccular maculae showed signs of atrophy and disruption in the guinea pigs with post-NE VEMP loss.

There is emerging evidence that there is a relationship between the loss of cVEMP and the degree of NIHL. In the present study, abnormal cVEMPs occurred most often in the ears with poorer-hearing (or greater NIHL), and the NE participants who had abnormal cVEMPs had poorer high-frequency pure-tone thresholds (greater NIHL) and greater interaural high-frequency pure-tone threshold differences than the NE participants with normal cVEMPs. Similarly, Wang and Young (2007) found that VEMPs were often absent or delayed in subjects with hearing thresholds >40 dB at 4000 Hz and a bilateral 4000 Hz notched audiogram. The changes in cVEMP observed in individuals with NIHL are most likely related to the consequences of noise exposure on the sacculocollic pathway rather than related to hearing loss, because it is well established that the presence of cVEMP is independent of cochlear function (Bickford et al. 1964; Colebatch et al. 1994; Ozeki et al. 1999).

In contrast with the present and previous studies that determined an amplitude decrement or cVEMP loss after noise

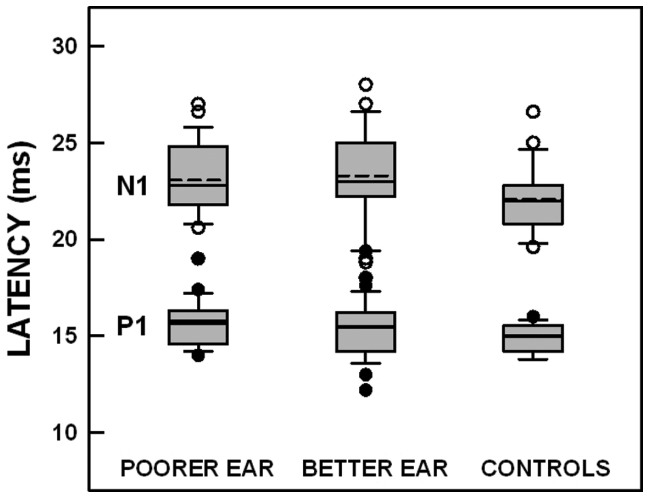

Fig. 7. Box plots of P1 and N1 latencies for the poorer-hearing ear and better-hearing ear of the NE group and for the mean of the right and left ears in the age-matched control group. The solid and dashed lines within the box mark the median and the mean, respectively. The boundary of the box closest to zero indicates the 25th percentile and the boundary of the box farthest from zero indicates the 75th percentile. The error bars above and below the box indicate the 90th and 10th percentiles, respectively. Outliers are represented by filled circles for P1 and by unfilled circles for N1. NE, noise exposure; cVEMP, cervical vestibular evoked myogenic potential.

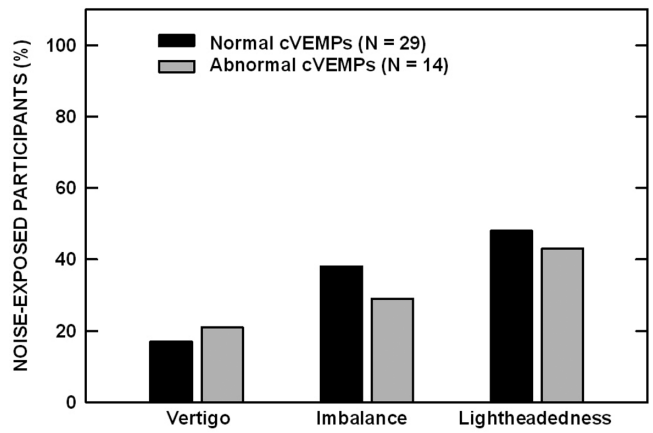

Fig. 8. Frequency of quality of dizziness categories in percentage for the NE participants with normal cVEMPs and the NE participants with abnormal cVEMPs. NE, noise exposure; cVEMP, cervical vestibular evoked myogenic potential.

exposure, Cassandro et al. (2003) reported an increase in cVEMP amplitude in university students after a 3 hr exposure to disco music (128 dB(C)). It is unclear whether this discrepancy is because of differences across studies in the cVEMP recording method (e.g., consideration of the effect of SCM muscle EMG on cVEMP amplitude) or in the amount of noise exposure before measurement of cVEMP, as there is evidence in guinea pigs that long-term or continuous noise exposure has more detrimental effects on cVEMP than short-term or intermittent noise exposure (Akdogan et al. 2009; Hsu et al. 2008).

An unexpected finding of this study was the absence of horizontal semicircular canal dysfunction on the SHA rotary chair test in all of the 43 NE participants. Most previous studies using caloric testing or rotational testing to examine the relationship between vestibular function and NIHL have reported subclinical vestibular findings associated with NIHL, although the vestibular findings vary among studies (Salmivalli et al. 1977; Man et al. 1980; Oosterveld et al. 1980; Shupak et al. 1994; Golz et al. 2001). For example, Shupak et al. (1994) reported lower VOR gain and decreased caloric responses in an NIHL group, whereas Oosterveld et al. (1980) reported spontaneous nystagmus, positional nystagmus, asymmetry on rotational testing, and a caloric weakness in airline mechanics with NIHL. Man et al. (1980) reported a higher incidence of positional nystagmus in acoustic trauma subjects than in a control group. Similarly, van Campen et al. (1999) found that 6 of 30 Oklahoma City bombing survivors tested had positional nystagmus and 2 had caloric weaknesses. In the present study, rotary chair testing was the primary method used to evaluate horizontal semicircular canal function. A limitation of this method is that rotary chair is less sensitive to unilateral vestibular lesions because both ears are stimulated during rotation. Without caloric testing, the rotary chair findings should be interpreted with caution. Using caloric testing, Golz et al. (2001) found that individuals with asymmetric NIHL were more likely to have abnormal findings (47%) compared with individuals with symmetrical NIHL (5%). In the present study, subclinical differences in horizontal semicircular canal function between groups could not be determined, because the control group did not undergo tests of rotary chair or caloric testing.

It is interesting that approximately half of the NE participants (49%) complained of dizziness. This finding is consistent with previous studies that have reported dizziness in some individuals exposed to noise (Alberti et al. 1979; Golz et al. 2001). A report of subjective dizziness was most common in the NE participants with abnormal cVEMPs because 57% of these participants reported dizziness compared with 48% of NE participants with normal cVEMPs. Thirty-four percent of both NE subgroups reported multiple quality of dizziness categories (i.e., vertigo, imbalance, or lightheadedness). Figure 8 shows the quality of dizziness categories for the NE participants with normal cVEMPs and the NE participants with abnormal cVEMPs. Lightheadedness was the most common symptom for both groups, and vertigo was the least common symptom. The low incidence of vertigo is consistent with the findings of normal horizontal semicircular canal function in all NE participants. Imbalance was reported more often in the NE participants with normal cVEMPs than in the NE participants with abnormal cVEMPs.

Sensory input to the vestibular sensory organs and nerves is processed by the central vestibular system, and the origin for dizziness can occur anywhere along these pathways. The central vestibular system includes the brainstem and cerebellum but pathways also project to higher centers in the midbrain and cerebral cortex. To our knowledge, the effect of noise on the central vestibular system has not been investigated previously. In this study, ocular motor testing was used as a screening measure for determining CNS function independent from peripheral vestibular system function. In addition, fixation suppression of vestibular nystagmus, which requires intact connections between the cerebellum and vestibular nuclei, was used as a clinical test for central vestibular involvement. All NE participants had normal ocular motor function and fixation suppression of vestibular nystagmus suggesting that noise exposure does not affect central vestibular function. Previous studies have reported prolonged cVEMP latency in patients with

central lesions in central vestibular pathways (Itoh et al. 2001; Pollack et al. 2006; Tseng & Young 2010). In this study, there was no difference in cVEMP latency between the NE and control participants. These findings suggest that noise exposure has a minimal effect on central vestibular pathways.

CONCLUSIONS

These findings provide further evidence of noise-induced vestibular loss in humans, and the results are consistent with previous studies in animals and humans that suggest the saccule may be particularly susceptible to noise-related damage. The cVEMP is a clinical test dependent on sacculocollic function that seems to be sensitive to the effects of noise exposure on the vestibular system. There is emerging evidence that the severity of NIHL is related to the presence or absence of cVEMPs. That is, cVEMPs are more likely to be absent in individuals with more severe NIHL than in individuals with less severe NIHL.

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