The Effects of High-Frequency Hearing Loss on Low-Frequency Components of the Click-Evoked Otoacoustic Emission

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Abstract
Click-evoked otoacoustic emission (CEOAE) input/output (I/O) functions were measured in ears with normal hearing and in ears with sensorineural hearing loss above 2000 Hz. The low- to midfrequency CEOAEs obtained from the ears with high-frequency hearing loss were significantly reduced in level compared to the CEOAEs obtained from the ears with normal hearing even though there were no significant group differences in the 250–2000 Hz pure-tone thresholds. The findings are discussed within the context of two hypotheses that explain the low- to midfrequency reduction in transient-evoked otoacoustic emission (TEOAE) magnitude: (1) subclinical damage to the more apical regions of the cochlea not detected by behavioral audiometry, or (2) trauma to the basal region of the cochlea that affects the generation of low-frequency emissions. It is proposed that localized damage at basal cochlear sites affects the generation of low- to midfrequency CEOAE energy.

Key Words: Click-evoked otoacoustic emission, high-frequency hearing loss, transient-evoked otoacoustic emission

Abbreviations: CEOAE = click-evoked otoacoustic emission; EOAE = evoked otoacoustic emission; I/O = input/output; OHCs = outer hair cells; TBOAE = tone-burst-evoked otoacoustic emission; TEOAE = transient-evoked otoacoustic emission; SSOAE = synchronized spontaneous otoacoustic emission

Sumario
Se midieron las funciones de input/output (I/O) para emisiones otoacústicas evocadas por clicks (CEOAE) en oídos con audición normal y en oídos con hipoacusias sensorineurales por encima de los 2000 Hz. Las CEOAE de baja a media frecuencia obtenidas a partir de los oídos con hipoacusia de alta frecuencia fueron significativamente reducidas en su nivel, comparadas con las CEOAE obtenidas de los oídos con audición normal, aunque no existieron diferencias significativas de grupo en los umbrales tonales de 250-2000 Hz. Los hallazgos se discuten dentro del contexto de dos hipótesis que explican la reducción en las frecuencias bajas a medias, en emisiones en la magnitud de las otoacústicas evocadas por transitorios (TEOAE): (1) daño sub-clínico a las regiones más apicales de la cóclea, no detectada por la audiometría conductual, o (2) trauma a la región basal de la cóclea que afecta la generación de emisiones de baja frecuencia. Se propone que el daño localizado a los sitios basales de la cóclea afecta la generación de la energía de las CEOAE de baja a media frecuencia.

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Evoked otoacoustic emissions (EOAEs), first detected by Kemp (1978), are low-level sounds generated within the cochlea in response to auditory stimuli that travel from the inner ear laterally through the middle ear into the ear canal where they can be recorded by a miniature microphone. It is generally accepted that the cochlear outer hair cells (OHCs) are an integral component in the production of EOAEs as OHCs and EOAEs share a similar physiological vulnerability to ototoxicity (Brown et al, 1989), noise exposure (Anderson and Kemp, 1979), hypoxia (Rebillard and Levigne-Rebillard, 1992), and genetic disorders (Horner et al, 1985). EOAEs, therefore, are associated with the same cochlear processes as those responsible for normal-hearing sensitivity and frequency selectivity. Although the clinical utility of EOAEs in the identification of hearing loss has been established (Kemp et al, 1990; Gorga et al, 1993, 1996, 1997, 1999; Prieve et al, 1993; Hurley and Musiek, 1994; Kimberley et al, 1994; Glattke et al, 1995; Stover et al, 1996; Hussain et al, 1998; Vinck et al, 1998; Dorn et al, 1999; Harrison and Norton, 1999), some issues concerning the processes involved in the generation of EOAEs remain unclear. One of these issues is related to the frequency specificity of transient-evoked otoacoustic emissions (TEOAEs) and the notion that TEOAEs are generated by independent frequency channels (Probst et al, 1986; Xu et al, 1994; Probst and colleagues, 1986) observed that the amplitude spectrum of summed TBOAEs was similar to the amplitude spectrum of the CEOAE. In a similar study, Xu et al (1994) measured TEOAEs obtained with individual tone bursts at 1000, 2000, and 3000 Hz and for a complex stimulus that consisted of a digital addition of the three single tone-burst stimuli. When the responses to the individual tone bursts were combined and compared with the response to the tone-burst complex, the amplitude spectra were similar. The apparent linear correspondence between stimulus and response spectra observed in these studies lends support to the notion that TEOAEs are evoked by a number of independent generators that are distributed along the cochlear partition.

The above hypothesis also has been tested using subjects with high-frequency hearing loss and normal low- to midfrequency thresholds. If TEOAE frequency channels are independent, then the low- to midfrequency TEOAEs obtained from subjects with high-frequency hearing loss (presumably partially damaged cochlea) would be similar to the low- to midfrequency TEOAEs obtained from subjects with normal hearing sensitivity across the entire range of audiometric frequencies (presumably intact cochlea). In this regard, somewhat conflicting results have been obtained. Prieve et al (1996) observed that TBOAE and CEOAE input-output functions were essentially identical in regions of normal hearing both for subjects with normal hearing and for subjects with normal low- to midfrequency hearing and high-frequency hearing loss. In addition, there was no significant difference in TEOAE spectrum of the TBOAE generally contains energy that corresponds to the stimulus spectrum (Elberling et al, 1985; Probst et al, 1986; Norton and Neely, 1987). When measured in the same ear, Probst and colleagues (1986) observed that the amplitude spectrum of summed TBOAEs was similar to the amplitude spectrum of the CEOAE.
group delay between the subjects with normal hearing and the subjects with hearing loss. Prieve et al concluded that the data supported the view that TEOAE frequency channels were independent.

In contrast to the Prieve et al data, several studies have shown a reduction in low- to midfrequency TEOAE level obtained from subjects with high-frequency hearing loss compared to subjects with normal hearing (Hauser et al, 1991; Avan et al, 1997; Kowalska and Sułkowski, 1997). Similarly, Arnold et al (1999) found that the 4 to 8 kHz DPOAE levels were significantly correlated with the average pure-tone thresholds from 11 to 20 kHz and that ultra-high-frequency hearing accounted for a significant proportion of the variance in DPOAE levels from 4 to 8 kHz. Avan et al (1991) and Avan and Bonfils (1993) demonstrated that TEOAE threshold and DPOAE amplitude were significantly correlated with the audiometric threshold at least one octave above the TEOAE frequency and one octave above the geometric mean of the DPOAE primary frequencies. The temporal characteristics of the TEOAE waveform also have been shown to be dependent on the status of the basal cochlea (Avan et al, 1993). Conflicting results also have been reported for guinea pig. Avan et al (1995) and Withnell et al (2000) observed significant reductions in low-frequency TEOAE magnitude following basal cochlear trauma, whereas the reductions observed by Ueda (1999) were negligible. Thus, it is not clear if localized damage at remote (basal) cochlear sites affects the generation of low- to midfrequency TEOAE energy in the human cochlea.

The purpose of this study was to reexamine the independence of TEOAE frequency channels by comparing the CEOAE levels obtained from a group of subjects having sensorineural hearing loss restricted to the higher audiometric frequencies with the CEOAE levels obtained from a group of subjects with normal hearing across the range of audiometric frequencies. In contrast to previous studies, the pure-tone thresholds of the subjects with high-frequency hearing loss were statistically matched (250–2000 Hz) with those from subjects with normal hearing, and CEOAEs were obtained for stimulus levels from 60 to 85 dB pSPL. In addition, attempts were made to balance the subject groups in terms of the following factors that have been shown to influence CEOAE level: (1) the number of right ears, (2) the number of males versus females, and (3) the number of ears having synchronized spontaneous otoacoustic emissions (SSOAEs) (Robinette, 1992; Prieve and Falter, 1995).

METHODS

Subjects

Click-evoked otoacoustic emissions (CEOAEs) were measured in one ear of two groups of adults. The normal hearing (NH) group consisted of 23 subjects (18 males and 5 females; 10 right ears and 13 left ears) between the ages of 28 and 62 years [Mean (M) = 47.4 yrs., standard deviation (SD) = 8.9] with pure-tone thresholds ≤30 dB HL (ANSI, 1996) at the octave frequencies from 250 to 8000 Hz as well as at the interoctave frequencies of 3000 and 6000 Hz. The hearing loss (HL) group consisted of 23 subjects (22 males and 1 female; 10 right ears and 13 left ears) between the ages of 40 and 76 years (M = 55.1 yrs., SD = 9.4) with pure-tone thresholds ≤30 dB HL at the octave frequencies from 250 to 2000 Hz and high-frequency sensorineural hearing loss. Two ears in the NH group had synchronized spontaneous otoacoustic emissions (SSOAEs) whereas SSOAEs were not observed in the HL group.

In addition to pure-tone threshold data, each subject met the following inclusion criteria: (1) no history of middle-ear disease, (2) no excessive cerumen in the ear canal, (3) normal middle-ear function as determined by 226 Hz acoustic admittance measures [tymanometric peak pressure ±100 daPa; static admittance within published norms (Holte, 1996)], and (4) presence of an ipsilateral 1000 Hz acoustic reflex in the test ear. The test ear was selected in order to minimize threshold differences at the octave frequencies 250 through 2000 Hz between the NH group and the HL group. Although etiology of the hearing loss was not considered as an inclusion criterion, the majority of the subjects in the HL group had a positive history of military noise exposure.

Procedures

CEOAEs were recorded using the ILO88/96 Otodynamic Analyser hardware and software version 5.60Y (Bray and Kemp,
The subjects were seated in a reclining chair in a double-walled sound booth. CEOAEs were measured using the nonlinear method of stimulus presentation and synchronous time-domain averaging. Two waveforms (A and B), each consisting of 1040 artifact-free responses, were obtained at six stimulus levels from 60 to 85 dB pSPL in 5 dB increments. The click stimuli were presented at a rate of 50/sec, and the default response window of 2.5 to 20.5 msec postclick was used. The artifact rejection level was set at a single value for each subject within the range of 40 to 45 dB pSPL in an attempt to keep noise levels constant within and between subjects.

The CEOAE levels were calculated using the ILO88/96 software by analyzing the correlated portions of the A and B waveforms into half-octave bands centered at 750, 1000, 1500, and 2000 Hz. The corresponding noise levels were calculated as the average difference between the A and B waveforms in the same half-octave bands. Unlike some otoacoustic emission measurement systems, the noise floor estimate is not determined by the background acoustical/electrical noise but by some other proprietary factor in the ILO88/96 software or hardware with a much lower effective level. Although the measurement method is valid, it prevents the computing of accurate means and standard deviations of CEOAE level when the CEOAE level is below the corresponding noise level. The calculations performed by the software often yield inaccurate and large negative CEOAE-level values. To limit the effects of this factor, the noise-level estimate was imposed as an effective “noise floor” by setting the value of any half-octave band CEOAE level that was below the corresponding noise level equal to the noise-level value.

Data Analysis

A mixed-model analysis of variance (ANOVA) with repeated measures was computed separately for audiometric thresholds and for CEOAE response levels (Abacus Concepts, SuperANOVA, version 1.11). Differences were considered to be statistically significant if $p < 0.05$. All $p$-values reported were adjusted using the Greenhouse-Geisser estimate of epsilon to correct for the inherent correlation of repeated measurements.

RESULTS

Mean hearing thresholds for each group are shown in Figure 1. There were no significant differences in mean thresholds between the NH group and the HL group for the octave frequencies from 250 to 2000 Hz based on a 2 x 4 (group x frequency) mixed-model ANOVA with repeated measures on the second factor. There was no significant difference in either a group effect or frequency-by-group interaction.

The mean half-octave band CEOAE response level is plotted as a function of stimulus level for each group in Figure 2. Each panel shows the data for a different half-octave band. The circles represent the data for the NH group, and the squares represent the data for the HL group. The solid line represents the corresponding mean residual noise level plus one standard deviation for the NH group, and the dashed line represents the corollary for the HL group. For each half-octave band, the input-output functions for both groups demonstrated an increase in CEOAE response level with an increase in stimulus level. The mean response levels for the NH group were higher than the response levels of the HL group at each half-octave band and at each stimulus level. The difference in mean CEOAE response levels between the NH group and the HL group increased as the half-octave band center frequency increased.
The lowest stimulus level for which the mean CEOAE response level was higher than the noise level for both groups at all four half-octave bands was 75 dB pSPL. Only the CEOAE response levels obtained at 75, 80, and 85 dB pSPL, therefore, were selected for statistical analysis. A 2 x 4 x 3 (group x half-octave band x stimulus level) mixed-model ANOVA was performed (the repeated measure was CEOAE response level for three stimulus levels and four half-octave bands). The results of the ANOVA are listed in Table 1. Although there were no significant differences in mean pure-tone thresholds between the two groups for the octave frequencies from 250 to 2000 Hz, ANOVA results indicated that the CEOAE response level for the NH group was significantly higher than the HL group (F (1,44) = 11.664, p = 0.0014). In addition, CEOAE response level was significantly different depending on stimulus level (F (2,132) = 96.049, p < 0.0001).

![Figure 2](image_url)

The lowest stimulus level for which the mean CEOAE response level was higher than the noise level for both groups at all four half-octave bands was 75 dB pSPL. Only the CEOAE response levels obtained at 75, 80, and 85 dB pSPL, therefore, were selected for statistical analysis. A 2 x 4 x 3 (group x half-octave band x stimulus level) mixed-model ANOVA was performed (the repeated measure was CEOAE response level for three stimulus levels and four half-octave bands). The results of the ANOVA are listed in Table 1. Although there were no significant differences in mean pure-tone thresholds between the two groups for the octave frequencies from 250 to 2000 Hz, ANOVA results indicated that the CEOAE response level for the NH group was significantly higher than the HL group (F (1,44) = 11.664, p = 0.0014). In addition, CEOAE response level was significantly different depending on stimulus level (F (2,132) = 96.049, p < 0.0001).

### Table 1 Summary Table for Mixed-Model ANOVA

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<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>p*</th>
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<td></td>
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<td>2.77</td>
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* *p*-values adjusted using the Greenhouse-Geisser estimate of epsilon.
and half-octave band center frequency ($F_{(3,132)} = 33.972, p < 0.0001$). Post-hoc means contrasts indicated that all pair-wise comparisons for stimulus level ($p < 0.0001$) and half-octave band center frequency ($p \leq 0.0210$) were significant.

The interaction between half-octave band center frequency and stimulus level was significant ($F_{(6,88)} = 3.470, p = 0.0114$), indicating that the change in CEOAE response level (averaged across the two groups) as a function of stimulus level was dependent on the half-octave band center frequency. The group and stimulus level interaction was not significant, indicating that the change in CEOAE response level (averaged across the four half-octave bands) as a function of stimulus level was of the same magnitude for both subject groups. In contrast to the group by stimulus level interaction, the interaction between group and half-octave band center frequency was significant ($F_{(6,88)} = 6.879, p = 0.0114$), and is illustrated in Figure 3 in which the mean CEOAE response level (averaged across the three stimulus levels) is plotted as a function of the half-octave band center frequency for each group. The mean CEOAE response level for the NH group was higher than the HL group at each half-octave band, and the magnitude of the difference in CEOAE response level between the two groups increased as a function of half-octave band center frequency.

**DISCUSSION**

The low- to midfrequency CEOAEs obtained from ears with high-frequency hearing loss were significantly reduced in level compared to those obtained from ears with normal-hearing thresholds across the range of audiometric frequencies. These findings are in agreement with similar studies using both animal and human subjects. Hauser and colleagues (1991) measured click-evoked and tone-burst-evoked OAEs in a group of subjects with normal hearing across the range of audiometric frequencies and in a group of hearing-impaired subjects with normal hearing through 2000 Hz and moderate-severe high-frequency sensorineural hearing loss. The subjects with hearing loss showed reduced emission amplitudes in regions of normal hearing compared to the subjects with normal hearing. Avan et al (1997) evaluated CEOAE amplitude in subjects with normal hearing through 4000 Hz and varying degrees of ultra-high-frequency (8000–16000 Hz) hearing loss. Results showed a significant negative correlation between CEOAE amplitude in the 1000–4000 Hz region and the average ultra-high-frequency hearing thresholds. In a study of workers exposed to high levels of industrial noise resulting in high-frequency hearing loss, Kowalska and Sulkowski (1997) found that the CEOAE amplitude in regions of normal hearing was reduced by 3 dB compared to that of a nonexposed control group with normal hearing.

Prieve et al (1996) concluded that TEOAE frequency channels were independent. They measured click-evoked and tone-burst-evoked OAE input/output (I/O) functions and group delays using subjects with normal hearing from 250–8000 Hz and subjects with normal hearing at low- to midfrequencies and hearing loss at high frequencies. The click-evoked and tone-burst-evoked OAE I/O functions were essentially identical in regions of normal hearing for the normal-hearing subjects as well as for subjects with high-frequency hearing loss. In addition, there was no significant difference in group delay between the subjects with normal hearing and the subjects with hearing loss. It should be noted that only within-group comparisons of click-evoked and tone-burst-evoked OAE I/O functions were made; no direct comparisons between I/O functions for the subjects with
normal hearing and the subjects with hearing loss were presented. On closer examination of their Figure 4, it is apparent that the I/O functions obtained from the subject with high-frequency hearing loss were reduced in level compared to those of the subject with normal hearing at each stimulus level and at each 1/3-octave band center frequency at which hearing sensitivity was normal (500, 1000, and 2000 Hz). In addition, the difference in CEOAE and TBOAE response levels between the subject with normal hearing and the subject with hearing loss increased as the 1/3-octave band center frequency increased. When viewed in this manner, the findings for these two subjects were similar to the results obtained in the present study. One important difference between the current study and the Prieve et al study concerns the audiometric thresholds of the subjects with hearing loss. In the present study, the pure-tone thresholds of the subjects with hearing loss were statistically matched with those of the normal-hearing subjects at the octave frequencies from 250–2000 Hz, and the cutoff frequency for the high-frequency hearing loss was 2000 Hz in all subjects. In contrast, the pure-tone thresholds of the hearing-impaired subjects in the Prieve et al study were not statistically matched with the normal-hearing subjects, and their cutoff frequencies varied from 500 to 4000 Hz.

Several hypotheses have been proposed to explain the reduction in the low-frequency emission components in ears with high-frequency hearing loss. Avan et al (1991, 1995) suggested that the low-frequency reduction may be due to either: (1) subtle or subclinical damage to the more apical regions of the cochlea not detected by behavioral audiometry, or (2) trauma to the basal region of the cochlea that affects the generation of low-frequency emissions. Supporting the hypothesis that subclinical damage to the cochlea is revealed earlier by OAE measures than by pure-tone audiometry are the findings that significant loss of OHCs in the apical cochlea went undetected by pure-tone audiometry (Prosen et al, 1990; Nicol et al, 1992) and the reduction in EOAE magnitude observed in noise-exposed, audiometrically normal ears (Attias et al, 1995; Lucertini et al, 2002). Consistent with the notion that remote changes in the basal region of the cochlea affect the generation of low-frequency emissions, Avan et al (1995) and Withnell et al (2000) observed a reduction in low-frequency TEOAE magnitude following acoustic trauma to the basal cochlea of the guinea pig.

Yates and Withnell (1999) recorded CEOAEs in normal-hearing guinea pigs with high-pass click stimuli and observed that the response spectrum was comprised of frequencies not present in the stimulus. Furthermore, following acoustic trauma to the base of the cochlea that resulted in high-frequency hearing loss, low-frequency CEOAE level was substantially reduced if the click stimulus contained energy within the range of frequencies affected by the trauma (Withnell et al, 2000). These findings suggest that the basal region of the cochlea contributes to the generation of low-frequency CEOAE components in the form of intermodulation distortion products and provide a reasonable basis for the reduction in low-frequency TEOAE magnitude observed in human subjects with high-frequency hearing loss.

Additional observations demonstrating the influence of the basal cochlea on low-frequency CEOAE components are evident in data concerning the utility of CEOAEs in the identification of hearing loss. The accuracy of classification of audiometric status (normal hearing versus impaired hearing) using CEOAE signal-to-noise ratio was improved using a multivariate approach to data analysis in which the signal-to-noise ratios at multiple CEOAE frequency bands were used to predict audiometric status at a single frequency (Hussain et al, 1998). Specifically, accuracy was improved when the CEOAE signal-to-noise ratios at higher frequencies (2000 and 4000 Hz) were used to predict the audiometric status at a lower frequency (1000 Hz) in comparison to a univariate approach in which the CEOAE signal-to-noise ratio at 1000 Hz was used to predict audiometric status at the same frequency. In contrast, there was little or no improvement in accuracy over the univariate approach when the CEOAE signal-to-noise ratios at lower frequencies (1000 and 2000 Hz) were used to predict the audiometric status at a lower frequency (1000 Hz) in comparison to a univariate approach in which the CEOAE signal-to-noise ratio at 1000 Hz was used to predict audiometric status at the same frequency. In contrast, there was little or no improvement in accuracy over the univariate approach when the CEOAE signal-to-noise ratios at lower frequencies (1000 and 2000 Hz) were used to predict the audiometric status at the same frequency. In contrast, there was little or no improvement in accuracy over the univariate approach when the CEOAE signal-to-noise ratios at lower frequencies (1000 and 2000 Hz) were used to predict the audiometric status at a lower frequency (1000 Hz). Hussain et al suggested that the observed correlation across frequency might reflect some influence of the basal cochlea on low-frequency emission components.

The results of the present study...
demonstrate that the low- to midfrequency CEOAEs obtained from ears with high-frequency hearing loss are significantly reduced in level compared to those obtained from ears with normal hearing even though there were no statistically significant group differences in pure-tone thresholds in the region of normal hearing (250–2000 Hz). These findings are not consistent with the notion that CEOAEs are evoked by independent generators within the cochlea and indicate that localized damage at basal cochlear sites affects the generation of low- to midfrequency CEOAE energy. It was assumed that the normal low- to midfrequency pure-tone thresholds for the hearing loss subjects in this study reflected normal OHC function in the corresponding regions of the cochlea since the factors that produce high-frequency hearing loss (e.g., noise) typically operate in a systematic manner from high to low frequencies (Hawkins and Johnsson, 1976). Additional factors (gender, number of right ears, and number of ears with SSOAEs) were considered in the subject selection process in order to minimize their influence on any observed group differences in CEOAE level. However, the presence of subclinical damage to the more apical regions of the cochlea cannot be ruled out in studies using human subjects. Therefore, it is not possible to determine unequivocally if the low-frequency reduction in CEOAE level is due to subclinical damage to the more apical regions of the cochlea or to remote changes in the basal cochlea. Moreover, it is conceivable that both mechanisms contribute to the reduction in low-frequency CEOAE magnitude observed in ears with high-frequency hearing loss.

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REFERENCES


