Application of a Stimulus Spectral Calibration Routine to Click Evoked Otoacoustic Emissions

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Abstract
This study examined the influence of a calibrated transient stimulus on click evoked otoacoustic emissions (CEOAEs). The calibration procedure produced a spectrally uniform stimulus (1-8 kHz) at the plane of the ear probe that was very similar among individuals. However, the calibrated signal reduced the overall level and repeatability of the CEOAE, probably due to the minimization of the energy peak at 2 kHz, which is enhanced in the uncalibrated signal. The amplitude of CEOAEs obtained with the calibrated signal was less variable among individuals compared to CEOAEs obtained with the uncalibrated signal.

Key Words: Calibration, click otoacoustic emission

Abbreviations: CEOAEs = click evoked otoacoustic emissions, FIR = finite impulse response, MANOVA = multivariate analysis of variance, p-p = peak-to-peak, Pa = Pascal

Transient signals such as clicks have been a popular stimulus for clinical assessment of auditory function using auditory brainstem responses (ABRs) and click evoked otoacoustic emissions (CEOAEs). The transient nature of the clicks results in a simultaneous response from a vast number of neurons essential for recording ABRs (Ferraro and Durrant, 1994), and their broadband frequency response results in a widespread stimulation of the cochlea, allowing for broad frequency information in CEOAEs (Norton and Stover, 1994).

A click is produced by delivering a rectangular electric pulse of short duration (T) to a transducer. The energy in a rectangular pulse decreases with an increase in frequency until it reaches a spectral zero at 1/T. The damping and the resonant characteristics of the transducer modify the spectrum of the click. Weber et al (1981) reported that most transducers used clinically have two major resonant frequencies. These frequencies vary between transducers, even among those from the same manufacturer. The click spectra produced by different transducers may produce different excitation patterns on the basilar membrane (Weber et al, 1981), leading to variations in physiologic measurements. Furthermore, insufficient information on the transducers used to obtain auditory measures limits our ability to compare results obtained from different transducers (Schwartz et al, 1985).

Recently, there has been a growing interest in the use of insert earphones for hearing assessment. Insert phones prevent ear canal collapse, increase interaural attenuation, are more comfortable than circumaural headphones, and reduce the stimulus artifacts in ABR recordings (Hall, 1992).

Historically, acoustic calibration of insert transducers is accomplished using couplers (ANSI, 1973). However, the signal in the ear canal can be quite different from the signal in the coupler. Ear canal length, shape, volume, and insertion depth modify the original signal. Moreover, these parameters vary depending upon the age of the patient (McLellan and Webb, 1957; Kruger, 1987). Johnson and Nelson (1991) reported that click spectra recorded in the ear
canal differ significantly between adult and infant ears and also vary among individuals in the same age group. Variations in the acoustic signal may be an important factor in the variability of auditory measures.

One auditory measure that might be affected by stimulus variation due to earphone characteristics and ear canal acoustics is CEOAEs. CEOAEs are sounds generated in the cochlea and recorded in the external auditory canal (Kemp, 1978) using an insert ear probe. The click level and spectrum are modified by the transducer in the insert earphone and by the ear canal acoustics. The changes in click level and spectrum differ between transducers and individual ears. Thus, comparing CEOAEs across patients is limited by the variation in the input signals.

Chertoff and Chen (1996) designed a signal processing algorithm to compensate for earphone transfer functions and ear canal acoustics. The procedure provides a signal with a flat spectrum from 1 to 8 kHz at the plane of the probe in the ear canal. With this procedure, it is possible to produce the same stimuli at the plane of the ear probe regardless of the variations in transducers and in the individual ear canals. Furthermore, the calibration procedure results in a stimulus with more high-frequency energy than used in present clinical equipment. Therefore, calibration of the signal spectrum may decrease between-subject variability and increase the high-frequency energy in the response.

The purpose of this study was to determine if ear canal calibration of stimulus spectrum results in CEOAEs that differ from the CEOAEs obtained using uncalibrated signals.

**METHOD**

**Subjects**

Thirteen subjects aged 18 to 30 years took part in this study. All had normal hearing in both ears (defined as pure-tone thresholds better than 20 dB HL [ANSI, 1973] from 250 Hz to 8 kHz). Tympanometric findings were within normal limits (static compliance of 0.5 to 1.5 ml, middle ear pressure between -50 to +50 mm H₂O). Acoustic reflex thresholds were between 70 and 90 dB HL at 0.5, 1, 2, and 4 kHz for both ipsi- and contralateral stimulation. Subjects had no history of otologic diseases, nor any significant exposure to noise or ototoxic drugs. They were not under the influence of medication, including aspirin, on the day of the experiment.

**Stimulus and Data Acquisition**

An impulse generated from a finite impulse response (FIR) filter was used to evoke CEOAEs. This signal was chosen because after calibration its spectrum contains more high-frequency energy than a rectangular pulse (click), thereby providing CEOAEs with more high frequencies. This FIR pulse was generated by an array processor (AP2 Tucker-Davis Technologies) housed in a personal computer (Gateway 2000 486/DX2/50). The digital signal was converted to analog (DA2 Tucker-Davis Technologies), attenuated (PA4 Tucker-Davis Technologies), and delivered to a hearing aid transducer (Knowles BP 1717) housed in a plastic ear speculum.

CEOAEs were recorded with a miniature microphone (Knowles EK 3133) enclosed in the speculum. Output from the microphone was high-pass filtered at 400 Hz (Krohn-Hite 3202) and low-pass filtered at 10 kHz (Stanford Research SR560). The responses were amplified 20,000 times and sampled at 65,536 kHz (AD2 Tucker-Davis Technologies) for 2204 points. Stimuli were delivered at a rate of 24/sec and one response was the average of 1000 signal presentations.

Recording of CEOAEs used a nonlinear differential method to eliminate stimulus artifact. Responses to three sets of FIR pulses with positive polarity (A, B, C) were recorded and stored. Two additional responses (D and E) were recorded in response to FIR pulses with opposite (negative) polarity and three times the amplitude of the positive polarity pulses (i.e., at 9.5 dB more than the positive polarity pulses). To detect a CEOAE, the initial 3 msec of the response were blanked to avoid stimulus artifacts. One CEOAE was obtained by adding the responses of A, B, C, and D and, similarly, a second CEOAE was obtained by adding A, B, C, and E. This procedure was done for both uncalibrated and ear canal calibrated conditions. Reproducibility of the CEOAE was determined from a correlation coefficient computed between the two responses.

**Procedure**

Each subject was seated in a recliner and asked to remain as still as possible throughout the experiment. The ear probe assembly was placed in the subject’s ear with the help of a
suitably sized, adapted tympanometric probe tip. CEOAEs were recorded to uncalibrated and calibrated FIR pulses presented at 90, 80, and 70 dB peak-to-peak (p-p) SPL (level refers to responses D and E). The calibrated and the uncalibrated conditions and the signal levels within a condition were randomized among subjects. The calibrated FIR pulse was obtained...
from the calibration routine described by Chertoff and Chen (1996) with a slight modification. That is, the impulse response from the ear canal and ear probe assembly was obtained at 85 dB p-p SPL and 90 dB p-p SPL. The two responses were subtracted and the calibration algorithm was applied to the difference waveform. The subtraction procedure was done to eliminate the emission during calibration so that the calibrated signal did not cancel any emissions. The calibrated signal has a uniform spectrum across the frequencies from 1 kHz to 8 kHz (Fig. 1).

**Statistics**

There were three dependent measures: overall amplitude, amplitude within four-frequency bands (1-2.8 kHz, 2.8-4.6 kHz, 4.6-6.4 kHz, and 6.4-8.2 kHz), and correlation between the two responses. Summary statistics (means, standard deviations) for amplitude were computed in Pascals (Pa). Significant differences between CEOAEs obtained with uncalibrated and calibrated signals were determined using repeated measure multivariate analysis of variance (MANOVA). Data analysis concerning correlations used scores transformed to z scores (McNemar, 1962). Hypothesis testing for significant differences between correlations used the normal distribution. A probability value of 0.05 was used as criteria for statistical significance.

**RESULTS**

**Stimulus**

The top left and right panels in Figure 1 show the FIR pulse in the ear canal at the plane of the ear probe in the time domain before and after spectral calibration for 12 subjects (the input signal from one subject was lost and could not be plotted). The interpeak latencies of the uncalibrated stimulus were longer in duration than those of the calibrated stimulus. Additionally, the ringing for the uncalibrated signal continued for at least 3 msec, whereas the calibrated signal exhibited ringing for approximately 1.5 msec. The lower two panels of Figure 1 show the uncalibrated and calibrated stimulus in the frequency domain. The uncalibrated signal showed two energy peaks centered around 2 kHz and 6 kHz. In many of the subjects, the energy at 2 kHz was much larger than the energy at 6 kHz. Furthermore, the energy at 6 kHz varied among individuals. In contrast to the uncalibrated signals, the spectra of the calibrated signal were flat and more uniform among individuals.

**CEOAES**

The top two panels in Figure 2 show an example of the CEOAEs in the time domain for the uncalibrated and calibrated FIR pulse in one subject. A reduction in CEOAE energy occurred with the calibrated signals, especially at the response latency of approximately 5 msec. The lower panels show the energy distribution of the CEOAEs in the frequency domain. The reduction in amplitude in the time domain appeared to be due to the reduction in amplitude for frequencies around 2 kHz.

The mean amplitude of the CEOAE obtained with the uncalibrated and calibrated stimuli presented at 70, 80, and 90 dB p-p SPL is illustrated in Figure 3. As signal level increased, the amplitude of the CEOAE increased. More importantly, however, the amplitude of the CEOAEs to the calibrated signal were smaller than the amplitude of the CEOAEs obtained from the uncalibrated signal. A two-way repeated measures MANOVA yielded a significant main effect for calibration (F_{1,12} = 11.572, p < .05), indicating that CEOAEs were significantly reduced when the spectrum of the stimulus was calibrated. The differences between the calibrated and uncalibrated conditions were, on average, 7 μPa at 70 dB, 14 μPa at 80 dB, and 29 μPa at 90 dB p-p SPL. The interaction between level and calibration, however, was not statistically significant.

Calibrating the spectrum of the signal did not affect the amplitude of the CEOAE equally across frequency bands. Figure 4 shows the mean amplitude of the CEOAE for the uncalibrated and calibrated stimuli for each of the four frequency bands and three signal levels. The effect of calibration on the amplitude of the CEOAE occurred for the lowest frequency band (1-2.8 kHz), whereas for the higher frequency bands, calibration did not influence the amplitude of the emission. A repeated measure MANOVA indicated significant main effects of level (F_{2,24} = 41.16, p < .05) and frequency band (F_{3,36} = 64.16, p < .05) and a significant interaction between level and band (F_{3,36} = 20.36, p < .05), but, for this study, the most important result was the significant calibration and band (F_{3,36} = 13, p < .05) interaction. The significant interaction is apparent in Figure 4, that is, calibration reduced the amplitude of the CEOAE in the low
Figure 2  CEOAEs from one subject. Top panels represent the emissions in the time domain for the uncalibrated signal (left panel) and calibrated signal (right panel). The bottom panels represent the emissions in the frequency domain. W1 and W2 refer to the initial emission and its replication. Time zero represents the time after blanking the stimulus artifact. Signal level was 90 dB p-p SPL.
frequencies and had no effect on the amplitude of the CEOAE in the high frequencies.

In addition to reducing the amplitude of the emission, the calibration procedure altered the between-subject variability. Table 1 illustrates the variation in amplitude among individuals. At 70 and 80 dB, the standard deviations were reduced by approximately 10 μPa, and at 90 dB the standard deviation was reduced by 17 μPa.

Figure 5 shows the mean correlation between the first and second CEOAE recording. As signal level increased, the correlation between the two responses increased. However, the correlation between responses for the calibrated signal were significantly lower than the uncalibrated signal at each signal level ($Z_{70} = 3.95$, $p < .05$; $Z_{80} = 4.51$, $p < .05$; $Z_{90} = 4.35$, $p < .05$).

DISCUSSION

Stimulus

Spectral calibration produced a signal with more high-frequency energy than the uncalibrated signal and decreased the variation in the acoustic signal among individuals. Interestingly, the reduced variability is not readily apparent when the acoustic signal is represented in the time domain. Both the uncalibrated and calibrated signal look remarkably similar among individuals. The difference, however, is easily seen in the spectrum of the signals.

The uncalibrated signal showed variation among individuals, especially in the high-frequency region, whereas calibrating the signal resulted in a signal more uniform among individuals. This suggests that examining the time domain representation of the signal may not be appropriate for determining the similarity of acoustic signals among individuals, and stresses the importance of examining the spectrum of the signal to determine subject variation.

An important concern with in-situ calibration is the location of the microphone in the ear canal. Although the spectral calibration algorithm equalized the energy in the signal and decreased the variability among individuals, this occurred at the plane of the probe in the ear canal. Correcting for sound pressure and flattening the spectrum at a location remote from the tympanic membrane does not guarantee that the corrections remain at the tympanic membrane. In fact, after calibration, the signal at the tympanic membrane could have excessive energy at particular frequency locations (Siegel, 1994). For example, the notch at 4 kHz (see Fig. 1) could be due to standing waves, a problem due to the location of the microphone in the ear canal. The calibration procedure would provide gain at the notch, which could lead to an increase in sound pressure at 4 kHz at the tympanic membrane. Future work with a microphone routed to the tympanic membrane will be necessary to examine the influence of the standing waves on the calibration routine and to determine the appropriate location for in-situ calibration for CEOAEs.

CEOAE

Calibration resulted in smaller CEOAEs when compared to the amplitude of the CEOAEs obtained with the uncalibrated signal. The reduction was largest for frequencies between 1 kHz and 2.8 kHz. One simple explanation involves the relation between the stimulus spectrum and response spectrum. The spectrum of the calibrated signal does not contain extra energy in the 1- to 2.8-kHz frequency region like the uncalibrated signal. The lack of energy in this frequency region in the stimulus resulted in smaller emissions in the same frequency region.

Interestingly, the relation between the change in energy in the stimulus and the corresponding change in the response suggests that the amplitude of the emission in the 1- to 2.8-kHz frequency region was not saturated. If one considers that toneburst emissions centered
at some frequency are similar to the frequency bands in CEOAEs (Stover and Norton, 1993), then the lack of saturation is conceivable. That is, the spectrum level of the FIR pulse at 90 dB p-p SPL was approximately 52 dB SPL. The input/output function of toneburst evoked emissions in this frequency region shows that saturation begins at approximately 50 dB SPL (Stover and Norton, 1993). Therefore, the signal level was at the border where the emission is saturated and a reduction in signal level due to spectral calibration could reduce the amplitude of the emission.

In addition to a frequency-specific reduction in CEOAE amplitude, the overall amplitude of the response was reduced when the spectrum of the acoustic signal was calibrated. This suggests that the 1- to 2.8-kHz frequency band of emissions contributes heavily to the overall amplitude of the response. This is consistent with a number of studies showing that emissions are more robust in lower frequencies than higher frequencies (Kemp et al, 1986; Norton and Neely, 1987; Kemp et al, 1990). The reason for this frequency emphasis is not known. It may be due to the inherent nature of the response generators or middle ear characteristics. However, the results from this study indicate a strong role for earphone and external ear acoustics on the spectrum of the emission.

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<th>Stimulus</th>
<th>70 dB</th>
<th>80 dB</th>
<th>90 dB</th>
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<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
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<tr>
<td>Uncalibrated</td>
<td>112</td>
<td>27</td>
<td>132</td>
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<tr>
<td>Calibrated</td>
<td>105</td>
<td>18</td>
<td>118</td>
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Clinical Implications

In clinical settings, it is desirable to obtain large CEOAE amplitudes and repeatable waveforms to help differentiate pathologic conditions. At stimulus intensities at or above 70 dB p-p SPL, calibration reduced the amplitude of the CEOAEs and correlation between responses. This could limit the clinical utility of the calibration procedure. The change in amplitude, however, is relatively small in dB, being approximately 0.6, 1.0, and 1.7 dB for the three signal levels. If this change is clinically relevant, it is possible that the smaller amplitudes and lower correlations may be remedied by using higher signal levels. Or, perhaps, a signal shaped to emphasize certain frequencies may provide larger responses and be more appropriate for CEOAEs. Future investigations will be necessary to validate these possibilities.

A favorable outcome of the calibration procedure is the reduction in variability in overall amplitude among individuals. A reduction in intersubject variability could be beneficial when differentiating results between normal and pathologic cases. However, the improvement in variability was small when considered in dB and must be interpreted with caution. Evaluating the variability for emissions with comparable response amplitudes (see Table 1) shows that the variability is similar between emissions obtained with the calibrated and uncalibrated signals. A second outcome of this study was the relation between the stimulus spectrum and the emission spectrum. The large emissions obtained with the uncalibrated signal were, most likely, due to the 2-kHz energy peak in the stimulus at the plane of the ear probe. Because the energy peaks can be influenced by factors such as subject age, characteristics of the ear canal, transducer, and probe placement, amplitude changes in the CEOAEs may simply reflect variations in the ear canal signal. Clinically, therefore, careful consideration of the acoustic signal must be taken into account when making clinical interpretations. The use of this calibration procedure to control for spectral content could minimize the effects of spectral variations in the acoustic signal, leading to improved clinical interpretation.

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References


