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2pPPa7. Demonstration of distributed distortion-product otoacoustic emission components using onset-latency techniques
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An oversimplified notion is that DPOAEs originate from a restricted region on the basilar membrane (BM). In actuality, DPOAEs are a distributed process involving the interaction of many wavelets, most likely generated over a broad region at, and basal to the overlap place of the primary tones. In the present study, DPOAEs were measured in rabbits as time waveforms by using phase rotation to cancel all components in the final average, except the 2f1-f2 DPOAE. At times, f2 was turned off for 6 ms producing a gap so that the DPOAE was no longer generated. These procedures allowed the DPOAE onset as well as the decay during the gap to be observed in the time domain. Results showed that complexities emerged near the onset and decay of the DPOAE time waveform as the f2/f1 ratio decreased, and at the beginning of the gap when f2 was turned off. Such complexities were unaffected by interference tones (ITs) near the DPOAE. However, these complexities were removed by ITs presented above f2, which can be explained by the interactions of distributed DPOAE components with different phase relationships.

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INTRODUCTION

Distortion-product otoacoustic emissions (DPOAEs) are commonly believed to consist of two distinct components (Shera & Guinan 1999). One element, referred to as the ‘overlap’ or ‘distortion’ component, is generated near f2 at a basilar-membrane (BM) locus where the f1 and f2 primary tones mix within the cochlea’s inherent nonlinearity. The f2 distortion component then travels to the DPOAE frequency place (f1p), and is reflected via a linear coherent reflection mechanism to give rise to the secondary or ‘reflection’ component. This point-source treatment then assumes that the two components emanate from very restricted regions on the BM. A contrasting view is that DPOAEs are generated via a distributed process involving the interaction of numerous wavelets, most likely emanating from a region at and basal to f2, where the f1 and f2 primary-tone traveling-wave (TW) envelopes overlap on the BM (Kemp 2002; Shera 2003).

One line of evidence supporting the existence of distributed DPOAE components used a suppression paradigm consisting of a third (f3) or interference tone (IT), placed at 1/3 octave (oct) above f2 (Martin et al. 2009, 2010, 2011). The vector differences between two conditions, i.e., IT present or absent during the collection of 2f1-f2 DPOAE level/phase maps and DP-grams, were then computed to reveal emission components that were removed by the IT. This series of IT studies showed that significant DPOAE components were generated at BM loci that were much more broadly distributed than had been generally supposed, i.e., up to at least two oct basal to the f2 primary-tone site.

A potential objection to this interpretation of findings based on the IT strategy is the possibility that the apparent basal components were generated by the complex interactions associated with the simultaneous presence of three pure tones (i.e., f1, f2, f3) in a nonlinear system due to a ‘catalyst mechanism’ (Fahey et al. 2000). It would be more convincing, then, if the actual existence of distributed DPOAE components could be demonstrated without relying on the complex IT paradigm. The present report relates the outcomes of experiments in which DPOAE time waveforms were obtained using a modification of an onset-latency method described by Whitehead et al. (1996), whereby the measured DPOAE is visualized in the time domain. With this approach, time-waveform constituents can be observed at the DPOAE onset and, depending on the latency value, information concerning their origins, i.e., whether they are generated from apical versus basal cochlear sites, would be obtained. In addition, the decay of the DPOAE induced by turning off the f1 primary tone would also provide other useful details concerning the generation loci of the emission.

METHODS

Subjects

Four adult female rabbits (3-4 kg) served as experimental subjects and, according to standard DP-gram measures, they exhibited normal cochlear function in both ears. Rabbits were tested in a sound-attenuated chamber while awake and comfortably restrained by a standard Plexiglas holder.

DPOAE Measures

The standard DP-gram methods used here are described in detail for rabbits in previous reports (Martin et al. 2010, 2011). Briefly, to establish normal baseline measures, DP-grams as a function of the f2 frequency (f2/f1=1.25) were measured in 0.1-oct steps, from f2=0.5-20.0 kHz, at various L1 and L2 levels (L1=L2=55, 65, 75 dB SPL, L1,L2=65, 55 dB SPL).

DPOAE Time Waveform Techniques

DPOAE time waveforms were obtained with L1=L2=65-dB SPL primary tones for f2/f1 ratios ranging from 1.01-1.25 in 0.02 steps. The procedures for obtaining DPOAE time waveforms were similar to those for obtaining their delays from the onset of the primary-tones until the DPOAE waveform could be detected in the ear canal (Whitehead et al. 1996). The emphasis here was to detect the emergence of multiple sources represented by perturbations in the emission’s time waveform. This capability was enhanced by turning f2 off at 5 ms, and then back on again at 11 ms after the initial onset of the primary tones, which allowed the visualization of complexities in the waveforms as various DPOAE components decayed during the 6-ms gap. The paradigm also provided for a replication of the onset process once f2 was turned back on. In some instances, the introduction of an IT during the onset/offset process brought about a deliberate suppression of discrete components of the generation activity.
The basic paradigm to obtain the DPOAE time waveforms was based on a phase-rotation technique in which the phases of the \( f_1 \) and \( f_2 \) primaries were systematically rotated between stimulus presentations, i.e., by 45° and 90°, respectively (Whitehead et al., 1996). Phase rotations were arranged to provide equal numbers of in-phase and 180° out-of-phase presentations, so that the primary tones and their harmonics, and all DPOAEs, except the 2\( f_1 \)-\( f_2 \) one of interest, were canceled in the final average. The phase-rotation process revealed the raw time waveform, which was then recovered by an inverse FFT (IFFT) operation, written to an output file, and transferred to a commercial spreadsheet, which was designed to subtract the acoustic delay, so that actual DPOAE latencies could be plotted on the time axis. The spreadsheet also synthesized a reference sinusoid that was in-phase and of equal magnitude to the DPOAE steady-state phase and level at 15-20 ms after primary-tone onset. The reference sinusoid was compared to that of the DPOAE at earlier regions of the time waveform.

**RESULTS**

**Effects of \( f_2/f_1 \) Ratio on DPOAE Time Waveforms**

Figure 1 for the right (R) ear of rabbit RV36 illustrates a portion of a typical \( f_2/f_1 \)-ratio series in which DPOAEs are shown as waveforms in the time domain. In panel A, the DPOAE waveform is depicted for the narrow

![DPOAE time waveforms](image)

**FIGURE 1.** DPOAE time waveforms for several narrow \( f_2/f_1 \)-ratio settings for a typical rabbit showing short-latency onset perturbations (black arrows) around 0.5-1.5 ms. At about 1.5 ms, the overall waveform magnitude decreased, which is consistent with the notion that longer-latency \( f_2 \) components were canceling the early onset of basal DPOAE components with shorter latencies. At the offset of \( f_2 \) (open arrowheads), as the canceling short-latency basal components decayed, a large component presumably generated nearer to \( f_2 \) became apparent. These more apical components required approximately 3.5 ms to decay (dashed arrow in panel a) representing a latency more consistent with the \( f_2=4 \) kHz cochlear location than the initial 0.5-ms irregularity observed as the primary tones were turned on. Vertical lines at 5 and 11 ms indicate the offset and onset of \( f_2 \), respectively. Values in parentheses just below the \( f_2/f_1 \)-ratio value at the top left of each plot indicate the level of the corresponding DPOAE at steady state in dB SPL.

\( f_2/f_1 \) ratio of 1.09 for the rabbit, where \( f_2 \) was turned off starting at 5 ms for a period of 6 ms. In this and the subsequent plot in panel B, note as the \( f_2/f_1 \) ratio decreased and the DPOAE level diminished (values in parentheses at top left), short-latency complexities were revealed at about 0.5-1 ms (solid black arrows in Figs 1a,b) near the onset of the DPOAE time waveforms. It should be emphasized that all rabbits showed similar complexities at such narrow \( f_2/f_1 \) ratios. The short latency of these waveform perturbations is consistent with them representing the more basal DPOAE components that theoretically should arrive back at the ear-canal microphone first. When \( f_2 \) was turned off, as indicated in the region demarcated by the two vertical black lines between 5 and 11 ms, large-
magnitude complexities became apparent in the time-domain waveforms (open arrowheads in Figs 1a,b). These offset perturbations are consistent with the emergence of longer-latency components generated closer to f₂ that were initially cancelled in the onset, and then revealed as more basal components responsible for the cancellation decayed, when f₂ was terminated. Note the long latencies of about 3.5 ms of some components (dashed arrow in Fig 1a) measured from the offset of f₂ (vertical black line at 5 ms). If DPOAE generation was restricted to a narrow region around f₂, rather than distributed, then, theoretically, these ‘offset’ latencies (dashed arrow) should be equal to their initial onset-latency counterparts (black arrow) at approximately 0.5-1 ms. Overall, these simple time-domain DPOAE waveforms provide compelling evidence for the interaction of distributed DPOAE components arising from distinct cochlear loci, with the complexity of the waveform depending upon the extent to which different components are either in-phase or out-of-phase, i.e., either adding to or cancelling one another.

Effects of ITs Basal to f₂ on DPOAE Time Waveform Complexities

To test the hypothesis that DPOAE time-waveform complexities were based upon the interaction of f₂ components with those generated more basally, ITs were introduced at various distances above a 4-kHz f₂ and the results are illustrated in Fig 2 for the R ear of rabbit RV36. In Fig 2a, an onset complexity is clearly visible (black arrow) as well as an offset complexity when f₂ was turned off (vertical line at 5 ms). When a 60-dB SPL IT was placed at 8 kHz, the onset-related component (black arrow in Fig 2B) was largely eliminated; the complexity produced by turning f₂ off was also substantially altered; and the DPOAE level increased from approximately 13 to 27 dB SPL. Together, these results suggest that DPOAE components in the 8-kHz range, which were approximately an oct above f₂, were interacting to produce the obvious waveform perturbations. In sum, as might be expected, distributed components from different cochlear locations contribute differentially to the overall time waveform, depending on their magnitude and phase.

FIGURE 2. DPOAE time waveform DPOAE perturbations originate from a restricted BM region above f₂. In panel a, DPOAE complexities at the onset of the primaries (black arrow at ~1 ms) and offset of f₂ (vertical line at 5 ms) are easily appreciated. In panel b, an 8-kHz IT presented an oct above the f₂=4 kHz frequency removed these complexities. These findings are consistent with the suggestion that at an oct above f₂, the IT removed DPOAE components originating basal to f₂ that were responsible for the short-latency irregularity. As these basal components diminished when f₂ was turned off at 5 ms, especially in panel a, DPOAE components generated nearer f₂ dominated to produce the large offset component.

DISCUSSION

The present report details the findings of a series of experiments in rabbits that utilized a modification of the DPOAE onset-latency method originally described by Whitehead et al. (1996) to demonstrate complexities in
DPOAE time waveforms that are consistent with the interaction of distributed DPOAE components along the BM. In these studies, at narrow f_2/f_1 ratios, short-latency onset perturbations were observed in the DPOAE time waveforms. When f_2 was turned off at 5 ms after the onset of primary tones, and on again at 11 ms, a large DPOAE component was revealed throughout a significant portion of the 6-ms ‘off’ period, which was consistent with the decay of basal components that previously were canceling the emission sources nearer to f_2. The onset complexities can not be the result of a catalyst mechanism, because only f_1 and f_2 were present in the absence of a third IT or f_3. In fact, for the offset complexities, only f_1 was present at a sufficient level to generate a DPOAE. Since only minimal stimulus ringing was observed after f_2 was turned off, the f_2 residual would not have sufficient magnitude to generate an emission, especially, one that was larger than when f_2 was present at its full level. When an IT was introduced at a frequency basal to f_2, both onset and offset complexities were eliminated as shown in Fig 2b. It would be extremely difficult to invent a scenario where the IT somehow generated a component that canceled these complexities. Consequently, the overall findings provide substantial time-domain evidence for the interaction of distributed DPOAE components, some of which could be removed by the IT. The present findings also have relevance to the results by other investigators that have proposed a role for distributed DPOAE components in contributing to a variety of DPOAE phenomena including the form of the commonly observed f_2/f_1 ratio function (e.g., Fahey et al., 2006), notches in DP-grams (e.g., Fahey et al., 2008), and the observation that under certain circumstances DPOAEs appear to be traveling, unexpectedly, in a reverse direction from the cochlear base to apex (e.g., Ren, 2004).

CONCLUSIONS

The present findings demonstrated complexities in DPOAE time waveforms as f_2/f_1 ratios were decreased both at the onset of the primaries and when f_2 was turned off. The most reasonable explanation for these time-domain complexities was that they were based upon the interaction of distributed DPOAE components. This interpretation is reinforced by the fact that such complexities could be removed by the presentation of ITs above f_2. However, the fact that they were apparent in the absence of a third tone (i.e., IT) supports a host of previous research (see Martin et al. 2010) demonstrating that catalyst effects of the IT are negligible, and that phenomena such as suppression/enhancement of DPOAE by ITs above f_2 (Martin et al. 1999, 2003) represent the removal of DPOAE components and not their creation by the third tone. For certain, this aspect of DPOAE generation deserves significant attention in future studies aimed at achieving a better relationship between the DP-gram and clinical audiogram. In addition,

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