Delay dependence for the origin of the nonlinear derived transient evoked otoacoustic emission

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In the guinea pig it has been shown that the nonlinear derived transient evoked otoacoustic emission (TEOAE_{nl}) is comprised of significant amounts of intermodulation distortion energy. It is expected that intermodulation distortion arising from a nonlinear distortion mechanism will contribute to the overall TEOAE in a stimulus-level-dependent manner, being greatest when basilar-membrane vibration in response to a click stimulus is greatest; with decay of vibration of the basilar membrane subsequent to stimulation by a click, nonlinear interaction along the cochlear partition should reduce and so provide for a linear mechanism to dominate TEOAE_{nl} generation, i.e., the contributions of each of these mechanisms should be delay dependent. To examine this delay dependence, TEOAE_{nl} evoked by acoustic clicks of varying bandwidth were time-domain windowed using a recursive exponential filter in an attempt to separate two components with amplitude and phase properties consistent with different mechanism. The latter part of the TEOAE_{nl} has an amplitude microstructure and a phase response more consistent with a place-fixed mechanism. © 2005 Acoustical Society of America. [DOI: 10.1121/1.1798352]

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I. INTRODUCTION

Studies of mammalian otoacoustic emissions (OAEs) over the past two decades have revealed notable differences between human and rodent OAEs. That is, rodents have (i) larger distortion product OAEs for the same level of stimulus, consistent with broader cochlear tuning (Shera et al., 2002); (ii) a much lower prevalence of spontaneous OAEs, in contrast to humans where such OAEs are commonly found (Strickland et al., 1985); and (iii) transient evoked OAEs (TEOAEs) with amplitude microstructure that is qualitatively similar to that found in humans. Studies in humans suggest a one-to-one correspondence between stimulus frequencies and TEOAE frequencies, i.e., each frequency contained within the TEOAE is evoked by only that frequency within the click stimulus (Kemp, 1986; Prieve et al., 1996), while in rodents (or in the guinea pig, at least) the TEOAE appears to be comprised of significant energy generated by intermodulation distortion (Yates and Withnell, 1999). These differences could be explained by describing the rodent cochlea as having broader cochlear filters and a less irregular cochlea and/or better impedance match between the middle and inner ears than that of humans.

TEOAEs in humans are thought to arise from an independent channel-generating mechanism such as a placefixed, linear reflection mechanism (Zweig and Shera, 1995). But, OAEs in general appear to arise from a composite of two mechanisms, a nonlinear distortion mechanism and a place-fixed mechanism (Talmadge *et al.*, 1998; Shera and Guinan, 1999; Goodman *et al.*, 2003). The contribution of each of these mechanisms is thought to be stimulus-level dependent and so with increasing stimulus level, intermodulation distortion energy arising from a nonlinear distortion mechanism should contribute more significantly to the TEOAE. In the guinea pig, it has been shown that the nonlinear derived TEOAE is comprised of significant amounts of intermodulation distortion energy, but the relative contribution of the two mechanisms has not been explicitly examined. This study examines the origin of the nonlinear derived TEOAE in guinea pig based on the hypothesis that the relative contributions of each of these mechanisms should be stimulus-level and delay dependent, some of the earliest part of the response of the basilar membrane to a high-level click stimulus involving the greatest excursion of the membrane from its resting position (Recio and Rhode, 2000) and so involving the largest nonlinear interaction. With decay of vibration of the basilar membrane, nonlinear interaction along the cochlear partition should reduce and so provide for a linear mechanism to dominate TEOAE_{nl} generation.

A. Origin of the TEOAE

OAEs are thought to have a complex generation involving two discrete mechanisms: place-fixed and wave-fixed (Kemp, 1986).

(i) Place-fixed: Zweig and Shera (1995) provided a theoretical framework for the generation of the placefixed mechanism, suggesting that place-fixed OAEs are an analog of Bragg scattering: the tall, broad peak of the traveling wave emphasizes a localized region from which energy is reflected from a distributed, place-fixed irregularity, i.e., the emission is thought to arise from the tip region of the traveling wave enve-

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lope. The defining signature of such emissions is a phase accumulation that will increase with frequency.

(ii) Wave-fixed (nonlinear distortion): Basilar-membrane impedance is not a linear function of stimulus level and the nonlinear change in impedance acts as a perturbation that may reflect the incident energy to become an otoacoustic emission (Talmadge *et al.*, 2000). This impedance perturbation is associated with the traveling wave envelope. The OAE arising from a nonlinear wave-related interaction has a phase accumulation that is constant, irrespective of stimulus frequency (Shera and Guinan, 1999).

The contribution of each of the above mechanisms appears to vary with stimulus level (Goodman et al., 2003), stimulus type, and species. For the TEOAE, examination of the origin is further complicated by the fact that the TEOAE can be extracted in more than one way, the differing methods not necessarily being equivalent in terms of the TEOAE obtained. Ear-canal sound pressure (P_{ec}) recorded in response to an acoustic stimulus is comprised of the stimulus, the OAE, and noise. Noise is reduced by synchronous averaging of the ear-canal sound-pressure recording. Extraction of the OAE from P_{ec} is dependent on the frequency and onset of the OAE relative to the acoustic stimulus. For OAEs with a frequency that differs from the stimulus frequency, the OAE can be distinguished from the stimulus using Fourier analysis or narrow-band windowing. For OAEs with a frequency that is the same as the stimulus, i.e., stimulus frequency OAEs, or in response to a short-duration stimulus with a spectrum that significantly overlaps that of the stimulus, i.e., TEOAEs, extraction of the OAE from $P_{\rm ec}$ is more problematic. The TEOAE can be extracted from $P_{\rm ec}$ in one of three ways, of which two are considered here:

- (i) Time-domain windowing,
- (ii) Nonlinear derived extraction.

and the third, noise suppression, is discussed in Sec. IV.

1. Time-domain windowing

TEOAEs result from using a very short-duration stimulus to evoke an OAE¹; ideally, there would be no overlap in time between the stimulus and the OAE. However, ringing of the loudspeaker or an acoustic stimulus that persists longer than the electrical stimulus that evoked it is typically due to resonance of the loudspeaker. It is, as a result, typical that the acoustic transient stimulus waveform will overlap in time with the TEOAE. As a result, it is not possible to isolate the *total* TEOAE from the stimulus using time-domain windowing. The degree of overlap between the stimulus and the TEOAE in time dictates how much of the total TEOAE can be extracted. Figure 1 provides an example of P_{ec} recorded from one animal. A time-domain window [F(t)] is also shown. Extraction of TEOAE_{win}, that part of P_{ec} that does not include the stimulus, using this example would be

 $\text{TEOAE}_{\text{win}} = P_{\text{ec}} - F(t) \cdot P_{\text{ec}}$

If there were no overlap in time between stimulus and TEOAE, TEOAE, would include all of the TEOAE. If



FIG. 1. Time-domain windowing: The ear-canal sound pressure (P_{ec}) when multiplied by a time-domain window F(t) can be used to try and isolate the stimulus from the OAE, i.e., TEOAE_{win} = $P_{ec} - F(t) \cdot P_{ec}$.

there were overlap between the stimulus and the TEOAE, TEOAE_{win} would only contain that part of the TEOAE that did not overlap with the stimulus and was not removed by time-domain windowing.

In humans, cochlear delay times are sufficiently long that some considerable part of the TEOAE is present beyond the duration of the stimulus (Kemp, 1978). Time-domain windowing of P_{ec} thus provides a means to extract a significant part of the TEOAE, although how much of the total TEOAE is extracted is not known, with stimulus ringing obscuring the onset and earliest part of the TEOAE. Rodents have much shorter cochlear delay times (Shera et al., 2002), and so the TEOAE appears to be considerably obscured by the stimulus, the result being that *time-domain windowing* cannot isolate some part of the TEOAE from the stimulus. Figure 2 provides an example of the P_{ec} recorded in a live animal and postmortem [panels (b) and (c)] and the $TEOAE_{nl}$ obtained from the live animal [panel (a)]. The y axis in panels (b) and (c) has been truncated to emphasize the amplitude of P_{ec} relative to TEOAE_{n1}, i.e., much of the early part of the stimulus has an amplitude that exceeds the upper bounds of the figures. It is evident that $TEOAE_{nl}$ in panel (a) enters the noise floor before the stimulus ceases in panel (b). Panel (c), where the stimulus level in the ear canal postmortem, was matched to the stimulus used with the live animal (stimulus levels differ by 0.5 dB), provides an estimate of cessation of the stimulus: comparison of panel (b) with panel (c) suggests that there is no significant TEOAE in panel (b) beyond cessation of the stimulus [determined by panel (c)]; i.e., the sound-pressure waveform in the ear canal beyond 0.004 s is noise only.² Because the TEOAE has an onset time that is delayed relative to the stimulus and a duration that is at least as long as the stimulus, it follows that some part of the TEOAE must persist beyond cessation of the stimulus. However, in rodents, this component has not been observed-presumably because it is obscured by noise and so, with sufficient averaging, it might be possible to identify this delayed, small part of the TEOAE. Clearly, though, it would not constitute a significant part of the overall TEOAE.

2. Nonlinear derived extraction

Synchronously averaged human-ear-canal soundpressure recordings in the time domain reveal that the part of



estimated cessation of stimulus

FIG. 2. An example of the ear-canal sound pressure ($P_{\rm ec}$) recorded in a live animal [panel (b)] and postmortem [panel (c)] and the TEOAE_{nl} obtained from the live animal [panel (a)]. The y axis in panels (b) and (c) has been truncated to emphasize the amplitude of $P_{\rm ec}$ relative to TEOAE_{nl}, i.e., much of the early part of the stimulus has an amplitude that exceeds the upper bounds of the figures. It is evident that TEOAE_{nl} in panel (a) enters the noise floor before the stimulus ceases in panel (b). Panel (c), where the stimulus level in the ear canal postmortem was matched to the stimulus used with the live animal (stimulus levels differ by 0.5 dB), provides an estimate of cessation of the stimulus: comparison of panel (b) with panel (c) suggests that there is no significant TEOAE in panel (b) beyond cessation of the stimulus.

the TEOAE that persists beyond the stimulus grows compressively nonlinearly (Kemp, 1978), and as a result Kemp suggested that the TEOAE could be extracted from the earcanal sound-pressure recording by making use of this nonlinear growth (see Kemp *et al.*, 1990), i.e., in response to a stimulus train consisting of *n* stimuli with a peak pressure *P* and one stimulus with a peak pressure *n*.*P*, the nonlinear derived TEOAE (TEOAE_{nl}) is given by

$$\text{TEOAE}_{\text{nl}} = \frac{n \cdot P_{\text{ecL}} - P_{\text{ecnL}}}{n - 1},$$

where P_{ecnL} is the ear-canal sound pressure recorded in response to the transient acoustic stimulus with a peak pressure n.P, P_{ecL} is the ear-canal sound pressure recorded in response to the transient acoustic stimulus with a peak pressure P. This is illustrated in Fig. 3 for n=2. Extraction of the total TEOAE is dependent on the stimulus level being sufficiently high that emission growth has saturated, otherwise the emission is underestimated.

Because the TEOAE in the guinea pig appears to be considerably obscured by the stimulus, i.e., most of the TEOAE is present within the duration of the stimulus, only TEOAE_{nl} can be reported as representative of the TEOAE.

3. TEOAE_{nl} versus TEOAE_{win}

In humans, studies of TEOAE origin have been reported for both TEOAE_{nl} and TEOAE_{win}. Prieve et al. (1996) reported findings for $TEOAE_{nl}^{3}$ that are consistent with a oneto-one correspondence between the stimulus frequency and the frequency of the OAE, and so would suggest $TEOAE_{n1}$ arises predominantly from a place-fixed mechanism. A oneto-one correspondence between the stimulus frequency and the frequency of the OAE would not occur for an OAE arising from a wave-fixed, nonlinear distortion mechanism due to the generation of intermodulation distortion products. A $TEOAE_{nl}$ that arises predominantly from a linear, place-fixed reflection mechanism is not at odds with the method of extraction of the OAE, i.e., a method of extraction of an OAE that utilizes cochlear nonlinearity will extract on OAE that arises from a linear mechanism if the growth of the OAE is affected by the compressively nonlinear growth of basilarmembrane vibration (Shera and Guinan, 1999; Kalluri and Shera, 2004).

Avan *et al.* (1997) reported findings for $\text{TEOAE}_{\text{win}}$, where the first 2.5 ms following stimulus onset of P_{ec} was



FIG. 3. An ear-canal sound-pressure measurement versus time in response to a stimulus train of three click stimuli, the first two clicks being of equal amplitude and the third twice the amplitude of the first two. The TEOAE is extracted from the ear-canal sound-pressure recordings by making use of the nonlinear growth of the TEOAE with stimulus level. In this example, the TEOAE = $(2.P_{ec} - P_{ecnL})$, where the stimulus for the recording of P_{ecnL} is twice as large as the stimulus for the recording of P_{ecl} .



FIG. 4. An example of a $P_{\rm ec}$ (click stimulus, 3–9-kHz bandwidth) versus time measured in the ear canal of a guinea pig, and the corresponding $\rm TEOAE_{nl}$.

removed by windowing. Damage to the basal turn of the cochlea was found to influence TEOAEwin energy at frequencies corresponding tonotopically to more apical cochlear locations. Such a finding is consistent with intermodulation distortion energy contributing to this TEOAE (Yates and Withnell, 1999; Withnell et al., 2000). Of course, it is also possible that damage to the basal turn introduces an additional source of reflection from which OAE energy could arise (Avan et al., 1997; Kakigi et al., 1998; Shera et al., 2004). Withnell et al. (2000) reported small changes to TEOAE_{n1} amplitude spectra in guinea pig with 1–5-kHz click stimuli following TTS of the basal turn, consistent with an additional reflection source, but also found that there was no change for an electrically evoked OAE (EEOAE) when the basal turn was damaged and the electrical current was injected into the third turn. No change to the EEOAE may not refute an additional reflection source associated with damage to the basal turn-modeling studies suggest such cochlear "perturbations" affect forward and reverse traveling waves differently with greater reflection of forward traveling waves (Shera, personal communication 2004).

Each method of extraction of the TEOAE has its limitations and neither is necessarily representative of the total TEOAE. TEOAE_{win} is contaminated by stimulus artifact and/or some of the TEOAE having been removed by windowing. TEOAE_{nl} will underestimate the TEOAE if the growth of the TEOAE is compressively nonlinear and the stimulus level is not sufficiently high that emission growth has saturated. Commensurate with this, Ravazzani *et al.* (1996) found TEOAE_{nl} in humans to be similar to TEOAE_{win} greater than 6 ms postsignal onset in response to "highlevel" stimuli.

In the guinea pig, studies of TEOAE origin have been confined to TEOAE_{nl}, with recent findings suggesting that TEOAE_{nl} appears to be comprised of significant energy generated by intermodulation distortion (Yates and Withnell, 1999; Withnell *et al.*, 2000). This is in stark contrast to the findings for TEOAE_{nl} in humans (Prieve *et al.*, 1996), and yet TEOAE_{nl} amplitude microstructure in guinea pig is qualitatively similar to that found in humans, i.e., both exhibit a quasiregular cyclical variation in the amplitude spectrum of the emission.

B. Origin of TEOAE_{nl} in guinea pig

Intermodulation distortion has been shown to contribute to the TEOAE_{nl} in guinea pig (Yates and Withnell, 1999). Energy arising from a nonlinear distortion mechanism, it has been suggested, should have an amplitude spectrum that is, for the most part, essentially devoid of microstructure (Talmadge *et al.*, 2000). Further, energy arising from a nonlinear distortion mechanism should be present in the earliest part of the TEOAE_{nl} response when the basilar-membrane response to the acoustic transient stimulus is greatest; as the response of the basilar membrane to the acoustic transient decays, nonlinear interaction will reduce and so provide for OAE arising from a place-fixed mechanism to contribute more significantly.

Amplitude microstructure for an OAE that arises solely from a linear place-fixed reflection mechanism would arise from the complex interaction of intracochlear standing waves with the initial apically reflected wave/s and variations in cochlear reflectance. This resonant behavior (intracochlear standing waves) will produce TEOAE_{nl}'s with a slow decay time (Talmadge et al., 1998) and spontaneous OAEs (Talmadge *et al.*, 1998; Shera, 2003). While such a TEOAE_{n1} will arise without nonlinear interaction, and so each frequency in the TEOAE_{nl} will correspond to its tonotopic cochlear location, intracochlear reflections will disassociate a simple temporal relationship for TEOAE_{nl} frequencies. In the absence of intracochlear reflections, the TEOAE_{nl} would be expected to have a fast decay time and a temporal relationship whereby the higher the OAE frequency, the shorter the delay time.

The amplitude microstructure in the TEOAE_{nl} in guinea pig, if it does not arise from intermodulation distortion energy, must arise from the interaction of OAE arising from a nonlinear distortion mechanism and OAE arising from a place-fixed mechanism. As such, it should be temporally distinguishable as being dominant in the latter part of the TEOAE_{nl} . To investigate this temporal relationship and amplitude microstructure in TEOAE_{nl} in guinea pig, the TEOAE_{nl} was time-domain windowed using a recursive exponential filter in an attempt to determine if two components with amplitude and phase properties consistent with different mechanisms of OAE generation could be distinguished.

II. METHOD

A. Animal surgery

Albino guinea pigs (300 to 550 grams) were anesthetized with Nembutal (35 mg/kg i.p.) and Atropine (0.06– 0.09 mg i.p.), followed approximately 15 minutes later by Hypnorm (0.1–0.15 ml i.m.). Neuroleptanaethesia was maintained with supplemental doses of Nembutal and Hypnorm. Guinea pigs were tracheostomized and mechanically ventilated on Carbogen (5% CO2 in O2) with body rectal temperature maintained at approximately 38.5 deg Celsius. The head was positioned using a custom-made head holder that could be rotated for access to the ear canal. Heart rate was monitored throughout each experiment. The bulla was opened dorso-laterally and a silver wire electrode placed on the round-window niche for the recording and monitoring of the compound action potential (CAP). In some cases, Pancuronium (0.15 ml i.m.) was administered to reduce physiological noise associated with muscle contractions. The number of animals examined for this study was 18, of which 10 had CAP thresholds that remained stable throughout the experiment and were within laboratory norms. The data presented in this paper represent a subset of the data obtained from the 10 animals with good CAP thresholds.

$$A(t) = \begin{bmatrix} \cos^2(\pi t/0.003)[\sin(2\pi f_c t)]/(2\pi f_c t) & -0.0015 < t < 0.0015 \\ 0 & \text{otherwise} \end{bmatrix}$$

B. Signal generation

The method for stimulus delivery has been described previously (Withnell *et al.*, 1998; Withnell and Yates, 1998). Briefly, the acoustic stimuli were delivered by a Beyer DT48 loudspeaker placed approximately 4 cm from the entrance to the ear canal. The stimulus waveform was calculated as a sinc function $(\sin(x)/x)$, appropriately time scaled according to the required low-pass frequency and windowed over 3 ms, using the equation

where f_c is the low-pass corner frequency.

Acoustic transient stimuli with varying bandwidths (e.g., 3–9, 7–14, 10–18 kHz) and stimulus levels were delivered to the ear without amplification, buffered by a Tucker-Davis HB6 amplifier. Stimulus spectra were relatively flat, achieved by compensating for the loudspeaker frequency response in the ear canal (see Yates and Withnell, 1999, for more details).

C. Data acquisition

A

Ear-canal sound-pressure recordings (P_{ec}) were made by a Sennheiser MKE 2–5 electrostatic microphone fitted with a metal probe tube (1.2 mm long, 1.3 mm i.d., 1500 Ω acoustic resistor) positioned approximately 2 mm into the ear canal. The microphone and probe tube combination was calibrated against a Bruel & Kjaer 4138 1/8-in. microphone. The output from the probe tube microphone was amplified 20 dB, high-pass filtered (0.64 kHz, 4-pole Butterworth), and transmitted as a balanced input to one of the analog input channels of the computer sound card (total gain=30 dB). It was subsequently digitized at a rate of 96 kHz.

This study examines TEOAE_{nl} obtained from the guinea pig, obtained using the nonlinear derived response technique (Kemp *et al.*, 1990) with a 6-dB stimulus level ratio, i.e., the stimulus train consisted of three acoustic transients with a 21.4-ms interstimulus interval, one of the stimuli being 6 dB higher than the other two. Each synchronously averaged recording represented a total of 21.38 s of data collection (21.4-ms epochs, 2048 points for the FFT). Data analysis was performed using Microsoft EXCEL and MATLAB.

D. Time-domain windowing of TEOAE_{nl}

Time-domain windowing to separate components with disparate amplitude structure was performed using a recursive exponential filter developed by Shera and Zweig (see Kalluri and Shera, 2001, Shera and Zweig, 1993), i.e.,

TEOAE_{nl} short latency component = $T(t) \cdot F(t)$, where T(t) is TEOAE_{nl} and $F(t) = 1/\Gamma_n(\tau)$ $\tau\!=\!t/\tau_{\rm cut},$ where t is time, $\tau_{\rm cut}$ is the length of the window

 $\Gamma_{\rm n}(\tau)$ is defined recursively as

$$\Gamma_{n+1}(\tau) = e^{\Gamma n(\tau) - 1}$$
, with $\Gamma_1(\tau) = e^{\tau^2}$.

The value of $\tau_{\rm cut}$ was chosen with the goal of minimizing the amplitude microstructure of the short latency component.

Filter order (*n*) was 14. This filter order was chosen based on the requirement of having a filter sufficiently steep that it meaningfully separates the TEOAE_{nl} in time while not being so steep that it introduces artifacts into the response. The late component of TEOAE_{nl} is given by

 TEOAE_{nl} late component = $T(t) - [T(t) \cdot F(t)]$.

III. RESULTS

A. Amplitude microstructure

Figure 4 shows a click stimulus (3–9-kHz bandwidth) and the corresponding TEOAE_{nl} versus time obtained from a guinea pig. Figure 5, panels (a) to (f), illustrates the effect of time-domain windowing the TEOAE_{nl} of Fig. 4 with six different values of $\tau_{\rm cut}$ (ranging from 0.00105 to 0.00315 seconds⁴), each panel having both amplitude and phase of the early OAE component, late OAE component, and the amplitude of the unwindowed TEOAE [except for panel (a), where no part of the TEOAE is within the window and so only the late component is present, this component being equivalent to the unwindowed TEOAE]. It is expected that applying a time-domain window to any signal in the time domain will separate the signal into two components-an early component and a late component. This is evident in panels (b) to (f). It is notable in panels (b) and (c) that the early OAE component has a relatively flat amplitude spectrum, with none of the amplitude microstructure that is evident in the unwindowed TEOAE or the late OAE component, and a phase slope that is not as steep as that of the late OAE component [panels (b) and (c) show the phase of $TEOAE_{nl}$, the phase of the early OAE component in both cases being similar to the phase of TEOAE_{nl}]. In subsequent figures, the choice of value of $au_{\rm cut}$ used was that which



FIG. 5. Amplitude and phase spectra illustrating time-domain windowing of the TEOAE_{nl} in Fig. 4 with six different cutoff values (τ_{cut}) for separating the TEOAE_{nl} into two components, designated the "early component" and "late component." In panel (a), the TEOAE_{nl} is not windowed at all and so the windowed component is negligible, while the remainder (the late component) is equal to the TEOAE_{nl}. Subsequent panels (b) to (f) show various degrees of separation of the TEOAE_{nl} into two components. Panel (c) shows the TEOAE_{nl} to be separated into two components, distinguishable based on amplitude and phase. See the text for further details.

- provided an early OAE component essentially devoid of amplitude microstructure and a late OAE component with amplitude microstructure;
- (ii) had a phase for the dominant or largest component with a similar slope to the phase of $TEOAE_{nl}$; and
- (iii) produced two components with distinguishable phase slopes, i.e., the slope of the early OAE component was less than that of the late OAE component.

Based on these criteria, a τ_{cut} of 0.0021 s, panel (c), would be chosen. Panel (b) with a τ_{cut} of 0.001 75 s provides for a late component with larger amplitude than the early component for a significant part of the frequency range of the OAE, but the phase of TEOAE_{nl} [right side of panel (b)] has a slope that is inconsistent with this—TEOAE_{nl} phase suggests a single component dominating above 4.5 kHz with the slope consistent with the dominant component being the early OAE component. To be consistent with the TEOAE_{nl} phase, the amplitude of the early component should be greater than the late component above 4.5 kHz.

Pursuant to the observation that the TEOAE_{nl} can be temporally windowed into two components with the amplitude microstructure confined to the late component, Fig. 6 shows examples from three animals of amplitude spectra and phase for TEOAE_{nl} windowed⁵ with the amplitude microstructure largely confined to the late component. In each case, the stimulus had a bandwidth of 4–10 kHz. The early OAE component spectral amplitude is predominantly greater than the late OAE component in panels (a) and (b), panel (c) revealing a different mix of the two components (the late OAE component is larger than the early OAE component below 5.8 kHz and from 9 to 9.8 kHz). The right side of panels (a) and (c) show TEOAE_{nl} phase to have a similar slope to the late OAE component at lower frequencies, becoming similar to the early OAE component phase slope above 5 to 6 kHz. Panel (b) shows $TEOAE_{nl}$ phase slope to be similar to the early component phase slope. A relatively slowly rotating phase for the TEOAE_{nl} emission is not commensurate with a round-trip physical cochlear delay to the 4-10-kHz region of the guinea pig cochlea. For a characteristic frequency of 7 kHz, the slope of the TEOAE_{nl} phase gives delays of approximately 350 μ s, too short for either a one-way or a round-trip cochlear delay, i.e., a round-trip cochlear delay of about 1600 μ s would be expected.⁶ The slope of the phase in panels (a) and (b) of $TEOAE_{nl}$ is consistent with the emission arising predominantly from a wave-fixed mechanism. The steeper slope for $TEOAE_{nl}$ in panel (a) below 5.2 kHz and in panel (c) below 5.8 kHz and from 9 to 9.8 kHz is commensurate with this part of TEOAE_{nl} arising from a place-fixed mechanism.

Figure 7 shows two examples of TEOAE_{nl} evoked by 10–18-kHz acoustic transients, i.e., a more basal cochlear stimulation than for Fig. 6. Amplitude microstructure is less pronounced in panel (a) than is found in panel (b) or in Fig. 6. Indeed, in panel (a), while it was possible to window the



FIG. 6. Examples from three animals of amplitude and phase spectra for TEOAE_{nl} windowed with the amplitude microstructure largely confined to the late component. In each case, the stimulus had a bandwidth of 4-10 kHz. The early OAE component spectral amplitude is predominantly greater than the late OAE component in panels (a) and (b), panel (c) revealing a different mix of the two components (the late OAE component is larger than the early OAE component below 5.8 kHz and from 9 to 9.8 kHz). Amplitude spectra are relatively devoid of microstructure for the early component, the late component having, in each case, pronounced microstructure. Phase responses show an early component with a shallow phase slope and a late component with a much steeper phase.

 $TEOAE_{nl}$ into two components with amplitude microstructure confined to the late component, the phases of the two components shown on the right side of panel (a) reveal similar slopes and the amplitude of the late OAE component on

the left side of panel (a) is more than 10 dB less than the early component versus frequency. That is, the phases of the slopes of the two components do not differ in panel (a)—it would seem in this case, commensurate with the small am-



FIG. 7. Two examples of amplitude and phase spectra for $\ensuremath{\text{TEOAE}}_{nl}$ evoked by 10–18-kHz acoustic clicks, i.e., a more basal cochlear stimulation than for Fig. 6. TEOAE_{nl} amplitude microstructure is less pronounced in panel (a) than in panel (b). Indeed, in panel (a), while it was possible to window the TEOAE_{nl} into two components with amplitude microstructure confined to the late component, the phases of the two components shown on the right side of panel (a) reveal similar slopes and the amplitude of the late OAE component on the left side of panel (a) is more than 10 dB less than the early component versus frequency. That is, the phases of the slopes of the two components do not differ in panel (a)-it would seem in this case, commensurate with the small amplitude microstructure, that this TEOAE_{nl} does not have two distinguishable OAE components. The TEOAE_{nl} in panel (b), in contrast, has greater amplitude microstructure and time-domain windowing isolates two components distinguishable based on amplitude and phase.



FIG. 8. Amplitude and phase spectra illustrating the stimulus-level dependence of the relative contribution of nonlinear distortion and place-fixed mechanisms to the generation of TEOAE_{nl}. Panel (a) is the OAE evoked by the highest level stimulus, panel (c), the lowest level stimulus, stimulus level decreasing in 5-dB steps from (a) to (c). In panel (a), TEOAE_{nl} is dominated by the early component. With decreasing stimulus level, the relative contribution of the early component reduces.

plitude microstructure, that this TEOAE_{nl} does not have two distinguishable OAE components. The TEOAE_{nl} in panel (b), in contrast, has greater amplitude microstructure, and time-domain windowing does isolate two components distinguishable based on amplitude and phase.

B. TEOAE_{nl} versus stimulus level

If the two components of the TEOAE_{nl} in guinea pig represent energy arising from a nonlinear distortion mechanism (the early component) and energy arising from a placefixed reflection mechanism (the late component), then the relative contribution of these two components should be stimulus-level dependent (Shera and Guinan, 1999; Talmadge et al., 2000; Goodman et al., 2003), i.e., the component arising from a nonlinear distortion or wave-fixed mechanism should become more significant relative to the place-fixed component with increasing stimulus level. This is examined in Fig. 8. Stimulus level decreases from panel (a) to panel (c) in 5-dB steps. The $TEOAE_{nl}$ in panel (a) is dominated by the early OAE component below 8 kHz and the late OAE component above 8 kHz-this is evident from the TEOAE_{nl} phase on the right side of panel (a), there being a shallow phase slope below 8 kHz and a steeper slope above 8 kHz. Panel (c) shows a TEOAE_{nl} with a steeper phase response up to 8 kHz than TEOAE_{nl} in panel (a), consistent with it being predominantly made up of the late OAE component (above 8 kHz the late component dominated the TEOAE_{nl} at all stimulus levels), i.e., this is consistent with the TEOAE_{nl} in panel (c) being dominated by a place-fixed generating mechanism. Windowing of TEOAE_{nl} to isolate an early OAE component with an amplitude spectrum relatively devoid of microstructure [left side of panel (c)] is consistent with this.

Figure 8 presents data consistent with the notion that the early and late OAE components represent OAE generated by a nonlinear distortion mechanism and a place-fixed mechanism, respectively, these two mechanisms being stimuluslevel dependent. OAE generated by a nonlinear distortion or wave-fixed mechanism presumably becomes increasingly significant as stimulus level increases.

C. Physiological verification of TEOAE_{nl}

The TEOAE was extracted using the nonlinear derived extraction paradigm. The setup for stimulus delivery and response acquisition was an open-field system (see Withnell *et al.*, 1998). An open-field system results in very little non-linear stimulus-related artifact being present in TEOAE_{nl} and so eliminates the need for time-domain windowing of the early part of the response as is done for human TEOAE recordings where the TEOAE is acquired using a closed system. Figure 9 illustrates the stimulus contamination of TEOAE_{nl} by contrasting the nonlinear derived component of the ear-canal sound-pressure recording extracted pre- and postmortem (stimulus level being matched pre- and postmor-



FIG. 9. Verification of the physiological nature of the TEOAE_{nl} response. Panel (a) shows the TEOAE_{nl} obtained from an alive animal, panel (b) the nonlinear derived residual postmortem.

tem), and the physiological nature of the TEOAE_{nl} obtained in this study. It is evident that there is very little contamination of TEOAE_{nl} by the stimulus.

IV. DISCUSSION

It would appear that the TEOAE_{n1} in the guinea pig can be time-domain filtered to separate two components with different amplitude and phase properties consistent with different mechanisms of OAE generation. It has previously been shown that intermodulation distortion energy contributes significantly to this TEOAE_{nl} (Yates and Withnell, 1999; Withnell et al., 2000). It is suggested that the earliest part of the TEOAE_{nl} separable by time-domain filtering that is essentially devoid of amplitude microstructure and has a phase slope that is shallow (associated with wave scaling⁷) arises from a nonlinear distortion mechanism. A click stimulus causes the basilar membrane to vibrate analogous to stimulating the cochlea with a range of stimulus levels-the displacement of the basilar membrane is largest soon after the onset of the cochlear response to a high-level click, providing for greatest nonlinear interaction, and then decays over time. The latter part of the TEOAE_{nl} with amplitude microstructure and a phase response that is steeper than the early component presumably arises from a place-fixed mechanism. The amplitude microstructure present in this latter component, in the absence of spontaneous OAEs and multiple intracochlear reflections (Withnell et al., 2003), could be due to variations in cochlear reflectance (Zweig and Shera, 1995; Goodman et al., 2003). The amplitude microstructure of $TEOAE_{nl}$ presumably arises from the complex interaction of the component arising from nonlinear distortion with the component arising from a place-fixed mechanism.

A stimulus-level dependence is at the heart of the perceived mechanisms by which OAEs are generated (Zweig and Shera, 1995; Talmadge *et al.*, 2000; Goodman *et al.*, 2003). Figure 8 suggests that the relative contributions of each of these mechanisms is stimulus-level dependent, the contribution of intermodulation distortion to the TEOAE_{nl} becoming more significant with increasing stimulus level. At



FIG. 10. Average group delay data (phase derivative) for the four animals from Figs. 5(c) and 6, a trend line fitted to this data, and the equation of best fit reported by Shera and Guinan (2003) to their data for SFOAEs evoked by 40-dB SPL stimuli.

the lowest stimulus level shown in Fig. 8 [panel (c)], the TEOAE_{nl} is predominantly comprised of energy arising from a place-fixed mechanism. TEOAE_{nl} phase in panel (c) is much steeper than in panel (a), commensurate with a shift in the mechanism dominating production of $TEOAE_{nl}$.

Time-domain windowing of the TEOAE_{nl} does not always appear to isolate two components distinguishable by their amplitude and phase. Figure 7(a) provides an example of a TEOAE_{nl} with very little amplitude microstructure that appears to arise almost totally from a nonlinear distortion mechanism—in this example, windowing does not isolate two components with different phase slopes.

For wideband inputs, group delay typically has no physical meaning, it being defined for narrow-band inputs (Papoulis, 1962). However, the cochlea separates a wideband input into a series of narrow-band inputs. A TEOAE generated by a linear, place-fixed reflection mechanism may be equivalent to an OAE that is a composite of SFOAEs generated by low-level stimuli with a frequency range encapsulated by the bandwidth of the click stimulus, i.e., it is a wideband response that is the sum of a series of narrow-band responses without nonlinear interaction. With no SOAEs and no multiple internal reflections (Withnell et al., 2003), group delay should then represent a round-trip delay for independentchannel generated TEOAE components. However, the phase response of an OAE arising from a place-fixed mechanism has been shown to be representative of round-trip cochlear delay subject to the effects of variation in cochlear reflectance (Shera and Guinan, 2003). Figure 10 provides the average group delay data (phase derivative) for the four animals from Figs. 5(c) and 6, a trend line fitted to this data, and the equation of best fit reported by Shera and Guinan (2003) to their data for SFOAEs evoked by 40-dB SPL stimuli. It is evident that the group-delay trend line for the latter part of the TEOAE_{nl} component presumed to arise from a placefixed origin underestimates SFOAE delay reported by Shera and Guinan (2003). The disagreement may be due to any of

- (i) the small sample size of our data set;
- (ii) the TEOAE_{nl} component described as having a placefixed origin may still have a component with a nonlinear origin confounding the phase;
- (iii) it is not meaningful to calculate group delay for wideband inputs; and
- (iv) stimulus-level differences.

Irrespective of the disagreement between the trend line for group-delay data from the latter part of the TEOAE_{nl} versus the findings of Shera and Guinan (2003), the notable differences in amplitude and phase for the two components of the TEOAE_{nl} isolated by time-domain windowing lead us to believe that the latter part of the TEOAE_{nl} does indeed arise from a place-fixed origin.

A. Choice of $\tau_{\rm cut}$

It is not contended nor implied that there is a value of τ_{cut} that perfectly separates TEOAE_{nl} arising from a nonlinear distortion mechanism versus a place-fixed mechanism. It is to be expected, based on a stimulus-level dependence underlying the generation of OAEs, that the TEOAE_{nl} over time represents a continuum of energy arising from both wave-fixed and place-fixed mechanisms. The degree to which each mechanism contributes to the TEOAE_{nl} at any point in time is presumably dependent on the displacement of the BM at that point in time in response to a click stimulus.

B. Noise suppression paradigm to extract the TEOAE from $P_{\rm ec}$

In Sec. I A, time-domain windowing and nonlinear derived extraction were considered in terms of isolating the TEOAE from the stimulus. A third method of extracting the TEOAE from the ear-canal sound pressure ($P_{\rm ec}$) is using a noise suppression paradigm. Molenaar *et al.* (2000) examined TEOAEs evoked by clicks with and without pseudorandom noise bursts of alternating phase in humans, the noise suppressing the TEOAE otherwise evoked by the click stimulus. This technique has the limitation that the TEOAE extracted will underestimate the total TEOAE if the noise suppressor does not completely suppress the generation of TEOAE. It does offer, though, an alternative means of extracting the TEOAE from $P_{\rm ec}$ that is worth investigating in the guinea pig.

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- ²The larger level of noise in panel (b) relative to panel (c) presumably is due to physiological noise.
- ³TEOAE_{nl} for this study was actually a combination of windowing and nonlinear extraction. The nonlinear extraction paradigm using a closed system [see Withnell *et al.* (1998) for discussion of closed versus open systems] in humans has a stimulus artifact that contaminates the early part of the ear-canal sound-pressure recording, and so the first 6 ms of the earcanal sound-pressure recording was zeroed (see Prieve *et al.*, 1996).

 $^4\tau_{\rm cut}$ is defined as that value in time where weighting value of window is 50%.

 ${}^{5}\tau_{cut}$ = 0.002 52 s for Fig. 6, panel (a) and 0.002 62 s for Fig. 6, panel (b). ⁶Estimate derived from auditory-nerve fiber threshold tuning data of Tsuji and Liberman (1997), based on a 400- μ s delay to the 18-kHz region, and assuming a phase change across filter bandwidth that is independent of bandwidth with a reciprocal relationship between cochlear group delay and filter bandwidth.

⁷The phase versus frequency response is relatively flat due to the impedance perturbation that reflects the incident energy shifting with the traveling wave envelope, i.e., the "traveling wave envelopes are locally 'shift-similar,' with the number of wavelengths in the traveling wave nearly independent of frequency" (Shera and Guinan, 1999, p. 786), with the reflection coming from the same region of the traveling wave, regardless of stimulus frequency. Wave scaling is a consequence of scaling symmetry (Shera and Guinan, 1999).

- Avan, P., Elbez, M., and Bonfils, P. (1997). "Click-evoked otoacoustic emissions and the influence of high-frequency hearing losses in humans," J. Acoust. Soc. Am. 101, 2771–2777.
- Goodman, S. S., Withnell, R. H., and Shera, C. A. (2003). "The origin of SFOAE microstructure in guinea pig," Hear. Res. 183, 7–17.
- Kakigi, A., Hirakawa, H., Harel, N., Mount, R. J., and Harrison, R. V. (1998). "Basal cochlear lesions result in increased amplitude of otoacoustic emissions," Audiol. Neuro-Otol. 3, 361–372.
- Kalluri, R., and Shera, C. A. (2001). "Distortion-product source unmixing: A test of the two mechanism model for DPOAE generation," J. Acoust. Soc. Am. 109, 622–637.
- Kalluri, R., and Shera, C. A. (2004). "The relationship between TEOAEs and SFOAEs at low stimulus levels," Association for Research in Otolaryngology Mid-Winter Meeting, Daytona Beach, Florida.
- Kemp, D. T. (1978). "Stimulated acoustic emissions from within the human auditory system," J. Acoust. Soc. Am. 64, 1386–1391.
- Kemp, D. T. (1986). "Otoacoustic emissions, traveling waves and cochlear mechanisms," Hear. Res. 22, 95–104.
- Kemp, D. T., Ryan, S., and Bray, P. (1990). "A guide to the effective use of otoacoustic emissions," Ear Hear. 11, 93–105.
- Molenaar, D. G., Shaw, G., and Eggermont, J. J. (2000). "Noise suppression of transient-evoked otoacoustic emissions. I. A comparison with the nonlinear method," Hear. Res. 143, 197–207.
- Papoulis (**1962**). *The Fourier Integral and its Applications*. (McGraw-Hill Book Company, New York).
- Prieve, B. A., Gorga, M. P., and Neely, S. T. (1996). "Click- and tone-burstevoked otoacoustic emissions in normal-hearing and hearing-impaired ears," J. Acoust. Soc. Am. 99, 3077–3086.
- Ravazzani, P., Tognola, G., and Grandori, F. (1996). "Derived nonlinear' versus 'linear' click-evoked otoacoustic emissions," Audiology 35, 73– 86.
- Recio, A., and Rhode, W. S. (2000). "Basilar membrane responses to broadband stimuli," J. Acoust. Soc. Am. 108, 2281–2298.
- Shera, C. A. (2003). "Wave interference in the generation of reflection- and distortion-source OAEs," in *Biophysics of the Cochlea: From Molecule to Model* (World Scientific, Singapore), pp. 439–449.
- Shera, C. A., and Guinan, J. J. (1999). "Evoked otoacoustic emissions arise by two fundamentally different mechanisms: A taxonomy for mammalian OAEs," J. Acoust. Soc. Am. 105, 782–798.
- Shera, C. A., and Guinan, J. J. (2003). "Stimulus-frequency-emission group delay: A test of coherent reflection filtering and a window on cochlear tuning," J. Acoust. Soc. Am. 113, 2762–2772.
- Shera, C. A., Guinan, J. J., and Oxenham, A. J. (2002). "Revised estimates of human cochlear tuning from otoacoustic and behavioral measurements," Proc. Natl. Acad. Sci. U.S.A. 99, 3318–3323.
- Shera, C. A., Tubis, A. Talmadge, C. L., and Guinan, J. J. (2004). "The dual effect of 'suppressor' tones on stimulus-frequency otoacoustic emissions," Association for Research in Otolaryngology Mid-Winter Meeting, Daytona Beach, Florida.

¹In response to a transient acoustic stimulus that has a relatively flat stimulus spectrum, the cochlea distributes the energy along the cochlear partition in a frequency-dependent manner with a delay that is reciprocally related to stimulus frequency. The TEOAE generated will reflect such cochlear delay and the mechanisms underlying generation.

- Shera, C. A., and Zweig, G. (1993). "Noninvasive measurement of the cochlear traveling wave ratio," J. Acoust. Soc. Am. 93, 3333–3352.
- Strickland, E. A., Burns, E. M., and Tubis, A. (1985). "Incidence of spontaneous otoacoustic emissions in children and infants," J. Acoust. Soc. Am. 78, 931–935.
- Talmadge, C. L., Tubis, A., Long, G. R., and Piskorski, P. (1998). "Modeling otoacoustic emission and hearing threshold fine structures," J. Acoust. Soc. Am. 104, 1517–1543.
- Talmadge, C. L., Tubis, A., Long, G. R., and Tong, C. (2000). "Modeling the combined effects of basilar membrane nonlinearity and roughness on stimulus frequency otoacoustic emission fine structure," J. Acoust. Soc. Am. 108, 2911–2932
- Tsuji, J. and Liberman, M. C. (1997). "Intracellular labeling of auditory nerve fibers in guinea pig: Central and peripheral projections," J. Comp. Neurol. 381, 188–202.
- Withnell, R. H., Kirk, D. L., and Yates, G. K. (1998). "Otoacoustic emis-

sions measured with a physically open recording system," J. Acoust. Soc. Am. 104, 350–355.

- Withnell, R. H., and Yates, G. K. (1998). "Onset of basilar membrane nonlinearity reflected in cubic distortion tone input–output functions," Hear. Res. 123, 87–96.
- Withnell, R. H., Yates, G. K., and Kirk, D. L. (2000). "Changes to low-frequency components of the TEOAE following acoustic trauma to the base of the cochlea," Hear. Res. 139, 1–12.
- Withnell, R. H., Shaffer, L. A., and Talmadge, C. L. (2003). "Generation of DPOAEs in the guinea pig," Hear. Res. 178, 106–117.
- Yates, G. K., and Withnell, R. H. (**1999**). "The role of intermodulation distortion in transient-evoked otoacoustic emissions," Hear. Res. **136**, 49–64.
- Zweig, G., and Shera, C. A. (1995). "The origin of periodicity in the spectrum of evoked otoacoustic emissions," J. Acoust. Soc. Am. 98, 2018– 2047.