Enhancement of the transient-evoked otoacoustic emission produced by the addition of a pure tone in the guinea pig

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This study examined the transient-evoked otoacoustic emission obtained in response to a click stimulus presented in combination with a pure tone in the guinea pig. Low-pass filtered click waveforms were digitally generated using a $\sin(t)/t$ function windowed over 3 ms with an elevated cosine envelope. Transient-evoked otoacoustic emissions were obtained using the nonlinear derived response technique. Phase locked pure tones of various frequencies at $\sim$70 dB SPL were electrically mixed with electrical clicks, with the pure tone present only for the three lower level stimuli in the train of four stimuli. Enhancement in the amplitude of the response spectrum at frequencies which corresponded to regions of the basilar membrane apical to the tone was observed with the addition of the tone. This finding is inconsistent with the transient-evoked otoacoustic emission being the result of independent generators. It suggests that intermodulation distortion energy may contribute to the transient-evoked otoacoustic emission, the enhancement in the emission response spectrum at frequencies below the pure tone being a result of a complex interaction on the basilar membrane of intermodulation distortion products. © *1998 Acoustical Society of America.* [S0001-4966(98)06107-4]

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INTRODUCTION

Otoacoustic emissions are evidence of an active process or mechanical injection of energy in the cochlea (Patuzzi *et al.*, 1989; Dallos, 1992). The active process is a positive feedback mechanism which enhances the vibration of the basilar membrane with negative damping or mechanical amplification (Patuzzi and Rajan, 1992; Dallos, 1992; Davis, 1983). An understanding of the physiological origin of the various types of otoacoustic emission is essential if otoacoustic emissions are to be meaningfully applied as an investigative tool of auditory function. Of the two most commonly measured types of emission, distortion product otoacoustic emissions are now felt to be reasonably well understood, being due to a nonlinearity inherent in the mechano-electrical transduction process (Jaramillo *et al.*, 1993; Howard and Hudspeth, 1988). The origin of the transient-evoked otoacoustic emission (TEOAE), it has been suggested, involves reflections from impedance discontinuities, either anatomical or as a result of a wave related mechanical interaction (Kemp, 1978, 1986; Guelke and Bunn, 1985; Strube, 1989).

A transient acoustical stimulus has a broad spectral profile which stimulates a wide region of the cochlear partition and produces an otoacoustic emission with a similarly broad power spectrum. TEOAEs have been presumed to involve a linear correspondence between the stimulus spectrum and the response spectrum (Wit *et al.*, 1981; Kemp *et al.*, 1990; Xu *et al.*, 1994), the frequency spectrum of the TEOAE being correlated with the presence or absence of auditory dysfunction at the corresponding cochleotopic location on the basilar membrane (Kemp *et al.*, 1990; Prieve *et al.*, 1993, 1996; Ueda *et al.*, 1997). However, this simple relationship may not be valid, since the assumption of a cochleotopic correlation between the acoustic stimulating energy and the resultant emission for TEOAEs does not satisfactorily explain certain experimental and histopathological findings (Wit and Ritsma, 1983; Avan *et al.*, 1993, 1995, 1997; Hilger *et al.*, 1995).

Sutton (1985) examined suppression of a TEOAE by pure tones, considering the spectral energy of the emission in the 1–3 kHz region with suppressor tones at 1010, 1290, 1760, and 2000 Hz on one human ear. He reported that the emission spectrum was reduced by a pure-tone suppressor, but in a complex manner and not confined to the region of the suppressor frequency. Sutton suggested that ''the emission generator does not behave as a simple localized source at the place for that frequency, but rather that the activity is distributed over a considerable length of the basilar membrane.'' Kemp and Chum (1980) and Tarvartkiladze *et al.* (1994) have also examined suppression of a TEOAE by pure tones. In contrast to Sutton (1985), Kemp and Chum (1980) reported suppression of a TEOAE in the human to be confined to the region of the suppressor tone frequency; however, for the example given in that paper the frequency of the suppressor tone was the ''particular frequency [that] resulted in the greatest and most tuned suppression'' (Kemp and Chum, 1980). Tarvartkiladze *et al.* (1994) reported iso-suppression tuning curves and also found a degree of ''tuning,'' but this tuning was associated with the spectrum of the response.

To explore further the question of origin of the TEOAE, we have extended the work of Sutton to consider the effect

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of a pure-tone suppressor on the TEOAE with a variety of stimulus spectra and pure-tone suppressors, but in this case in the guinea pig. Pure tones of ~70 dB SPL were used in this study: a 70 dB SPL pure tone presumably suppresses a region on the basilar membrane that roughly matches the suppressor excitation pattern on the basilar membrane with maximum suppression occurring in the region of the frequency of the pure tone. If TEOAE generators act independently, then with the addition of a 70 dB SPL tone we expect a reduction in the amplitude of the TEOAE power spectrum over a range of frequencies but with the predominant effect being a maximal reduction of the emission near the frequency of the tone. Enhancement of the emission is not to be expected at any frequency.

I. METHOD

Pigmented guinea pigs approximately 600 grams in weight were anaesthetized with Nembutal (30–35 mg/kg i.p.) and Atropine (0.06 mg i.p.), followed approximately 15 min later by Leptan or Hypnorm (0.15–0.2 ml i.m.). Neuroleptanaesthesia (Evans, 1979) was maintained using supplementary doses of Nembutal and Leptan or Hypnorm. The guinea pigs were tracheostomised and artificially respired on Carbogen (5% CO_2 in O_2), with body (rectal) temperature maintained at 37 °C. The head was positioned using a headholder which could then be rotated for access to the ear canal. Alloferin or Pancuronium (0.15 ml i.m.) was administered to reduce stapedius muscle contractions. During paralysis potentially noxious stimuli produced no change in heart rate. The bulla was opened post-auricularly and a silver wire electrode placed on the round window niche for recording of the compound action potential to monitor the condition of the cochlea. A plastic tube was positioned in the bulla opening to ensure that the bulla was always adequately ventilated, although no attempt was made to seal the bulla. This could have resulted in some variation in bulla resonance from animal to animal (in the range 300–1000 Hz), but OAEs were only examined above 1000 Hz.

This experiment was computer controlled with custom software and a sound-card (Crystal Semiconductor Corporation CS4231A). Electrical mono-polar click stimuli were acoustically delivered open-field by a Foster dynamic earphone type T016H01A0000. Ear canal sound pressures were measured with a Sennheiser MKE 2–5 electrostatic microphone coupled to a metal probe tube 9 mm in length and 1.3-mm internal diameter. A 1500-Ω acoustic filter was used as an acoustic damper in the probe tube. The probe tube was placed approximately 2 mm into the external auditory meatus and the earphone positioned near the pinna with the position of the earphone adjusted to obtain a relatively flat ear canal sound pressure spectrum. This arrangement provides a viable alternative to conventional acoustic probe systems for the measurement of otoacoustic emissions, the position of the earphone close to the pinna but not coupled to the meatus providing a much wider stimulus spectrum than that typically obtained with conventional acoustic probe designs (Withnell *et al.*, 1998).

The output from the probe tube microphone was amplified 20 dB, bandpass filtered (0.3–100 kHz) using two-pole Butterworth filters, and then digitized in 26-ms epochs at a rate of 44.1 kHz. Stimulus repetition rate was approximately 38.5 Hz. A cosine-ended window was applied to the first and last 2 ms of the averaged response *post hoc* to avoid frequency splatter associated with the Fourier transform calculation; no windowing of the TEOAE was performed though as stimulus onset in each epoch occurred at 2 ms. Corrections for the probe tube response have been made.

Low-pass filtered click waveforms were digitally generated using a $\sin(t)/t$ function windowed over 3 ms with an elevated-cosine envelope. TEOAEs were obtained using the nonlinear derived response technique (Kemp *et al.,* 1990). The stimulus train consisted of three click stimuli followed by a single similar click stimulus at three times the intensity. The three ear canal sound pressure measurements to the lower level click were added together, and the fourth ear canal measurement to the higher level click subtracted. The result was then divided by three to form the derived response. Each stimulus train was repeated 250 or 1000 times and the responses averaged. Pure tones of various frequencies at ~70 dB SPL and phase locked to the click repetition were electrically mixed with electrical clicks, with the pure tone present only for the three lower level stimuli in the train of four stimuli but not for the higher level reference stimulus. The phase of the tone was inverted on each alternate stimulus train so as to cancel it in the averaging process. Thus we were able to examine the effect of the pure tones on the response to the lower level click stimulus only. The choice of pure-tone frequency was restricted by the requirement of the tone having a phase relationship with respect to the signal averaging process such that the tone was cancelled from the averaged response. Fourier transforms for TEOAEs were calculated with 1024 data points. Data analysis was performed using Microsoft Excel.

The care and use of animals reported on in this study were approved by the Animal Experimentation Ethics Committee of the University of Western Australia and all procedures conformed with the Code of Practice of the National Health and Medical Research Council of Australia.

II. RESULTS

Figure 1 shows examples of the effect of a pure tone presented in combination with a click stimulus when the frequency of the pure tone was within the passband of the stimulus. TEOAEs are shown in response to an 80 dB pSPL (peak sound pressure level) click stimulus with the addition of a 66–72 dB SPL pure tone with a frequency of 3, 4, 5, or 6 kHz, the click stimulus having a relatively flat spectrum from 0 to 7 kHz. The 3-kHz tone enhanced the emission from 1 to 2 kHz and caused some reduction in the emission spectrum centered around 2.5 kHz. A 4-kHz tone produced enhancement of the TEOAE from 1.5 to 3 kHz, a 5-kHz tone from 2.5 to 5 kHz, and a 6-kHz tone from 4 to 6 kHz. Some reduction in the emission is seen around 2 and 5 kHz for the 5-kHz tone and from 2 to 4 kHz and from 6 to 7 kHz for the 6-kHz tone. In complete contrast to our expectation, but consistent with the findings of Sutton (1985) in a human ear, there was effectively no suppression centered on the suppressor frequency. Furthermore, there was evidence of enhance-

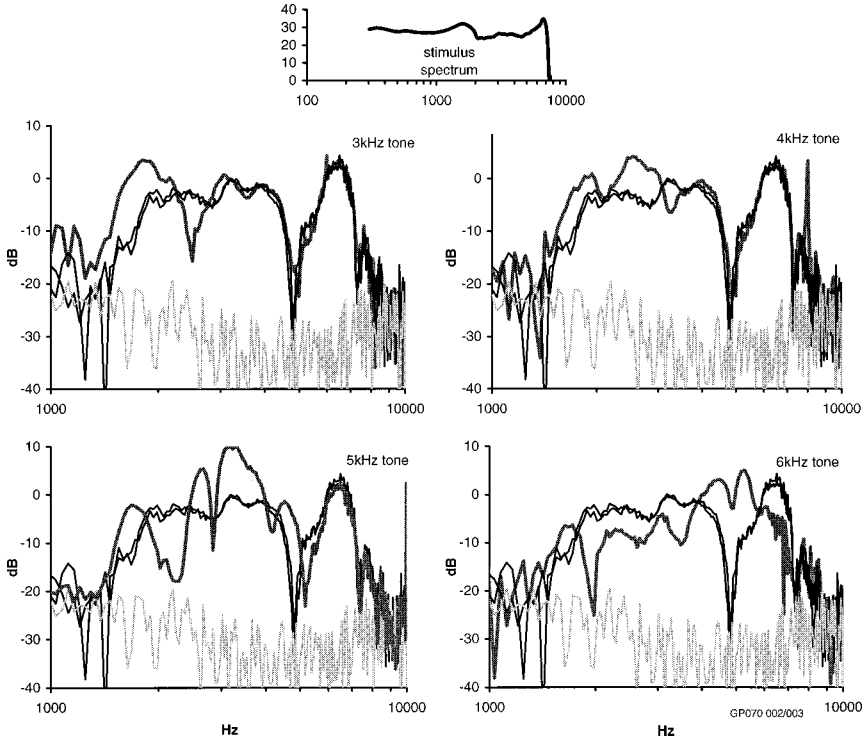

FIG. 1. TEOAEs in response to a 0–7 kHz bandwidth transient acoustic stimulus demonstrating enhancement and suppression of the emission spectrum when combined with an ~70 dB SPL 3-, 4-, 5-, or 6-kHz pure tone. Response repeatability is illustrated by two separate measures of the response without a pure tone added (dark curves). The lighter shading curve represents the TEOAE with the addition of a pure tone. A low level second harmonic of the pure tone that has not been cancelled is evident. Also shown is the noise floor. Decibels on ordinate scale is calculated *re*: 0.000 01 Pa/$\sqrt{\text{Hz}}$.

ment in all cases at frequencies corresponding to regions on the basilar membrane apical to the tone. Only for the case of the 6-kHz tone is there evidence of suppression in the region of the tone frequency.

We extended Sutton's work by considering the effect of a tone above the stimulus frequency range. Figure 2 shows power density spectra for TEOAEs obtained from a 75 dB pSPL click stimulus with a relatively flat signal spectrum from 0 to 5 kHz. The addition of a 66–70 dB SPL tone with a frequency of 10, 12, or 15 kHz clearly enhances the amplitude of the response spectrum below 5 kHz, the degree of enhancement being related to the frequency of the tone. The higher the frequency of the tone, the smaller the effect on the amplitude of the response spectrum. For the 10-kHz tone there is enhancement of the response spectrum from 1 to 5 kHz; the 12-kHz tone produces enhancement from 1.5 to 5 kHz; the 15-kHz tone enhancement is predominantly from 3.5 to 5 kHz. In all three cases the frequency of the tone is above the uppermost frequency of the stimulus spectrum.

Figures 1 and 2 provide results obtained from different animals. In Figs. 3 and 4, the effect on the TEOAE with the addition of a 68–69 dB SPL pure tone with a frequency both within and above the stimulus passband is shown for the one animal. Figure 3 is for an 80 dB pSPL click stimulus with a

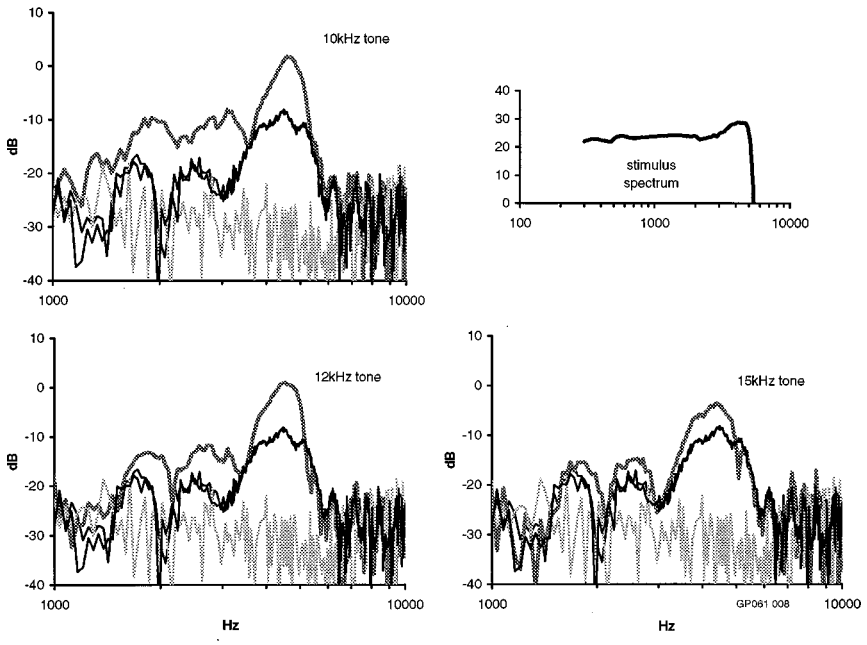

FIG. 2. TEOAEs in response to a 0–5 kHz bandwidth transient acoustic stimulus showing predominantly enhancement of the emission when combined with an ~70 dB SPL 10-, 12-, or 15-kHz pure tone. The lighter shading curve represents the TEOAE with the addition of a pure tone, the dark curves without a pure tone. Also shown is the noise floor.

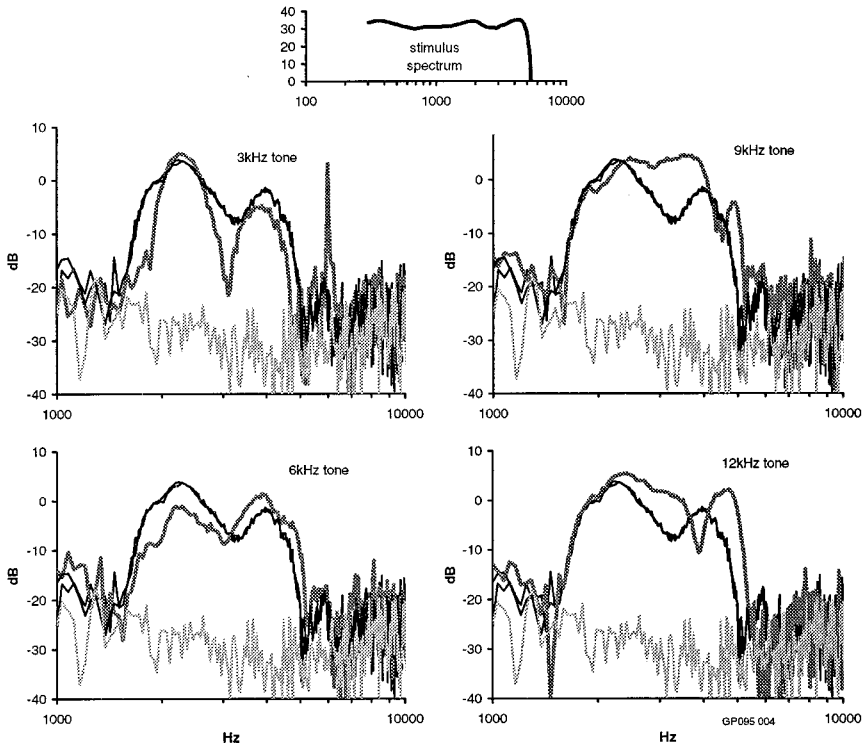

FIG. 3. TEOAEs in response to a 0–5 kHz bandwidth stimulus demonstrating the effect on the emission of adding an ~70 dB SPL 3-, 6-, 9-, or 12-kHz pure tone. The lighter shading curve represents the TEOAE with the addition of a pure tone, the dark curves without a pure tone. A low level second harmonic of the pure tone that has not been cancelled is evident. Also shown is the noise floor.

5-kHz bandwidth; Fig. 4 for an 89 dB pSPL stimulus with a 10-kHz bandwidth. In Fig. 3, enhancement of the TEOAE is evident from 3 to 5-kHz with the addition of the 6-kHz tone, from 2.5 to 5 kHz with a 9-kHz tone, and from 2.5 to 5.5 kHz with a 12-kHz tone. The TEOAE is reduced essentially from 1.5 to 5 kHz with the addition of a 3-kHz tone, from 1.5 to 3 kHz for a 6-kHz tone, from 2 to 2.5 kHz for a 9-kHz tone, and centered around 4 kHz with a 12-kHz tone.

Figure 4 shows changes to the TEOAE in response to a 10-kHz click stimulus with the addition of a 6-, 9-, 12-, or 15-kHz tone. Enhancement of the TEOAE is seen essentially from 3 to 6 kHz with the addition of a 6-kHz tone, and around 7 kHz for a 15-kHz tone. Note that in all cases there is TEOAE evident above the stimulus passband. The TEOAE is reduced from 2 to 3 kHz for a 6-kHz tone, from 6 to 8 kHz for a 12-kHz tone, and from 10 to 13 kHz and possibly around 3 and 5 kHz with a 15-kHz tone. The changes to the TEOAE in all cases are not as pronounced as

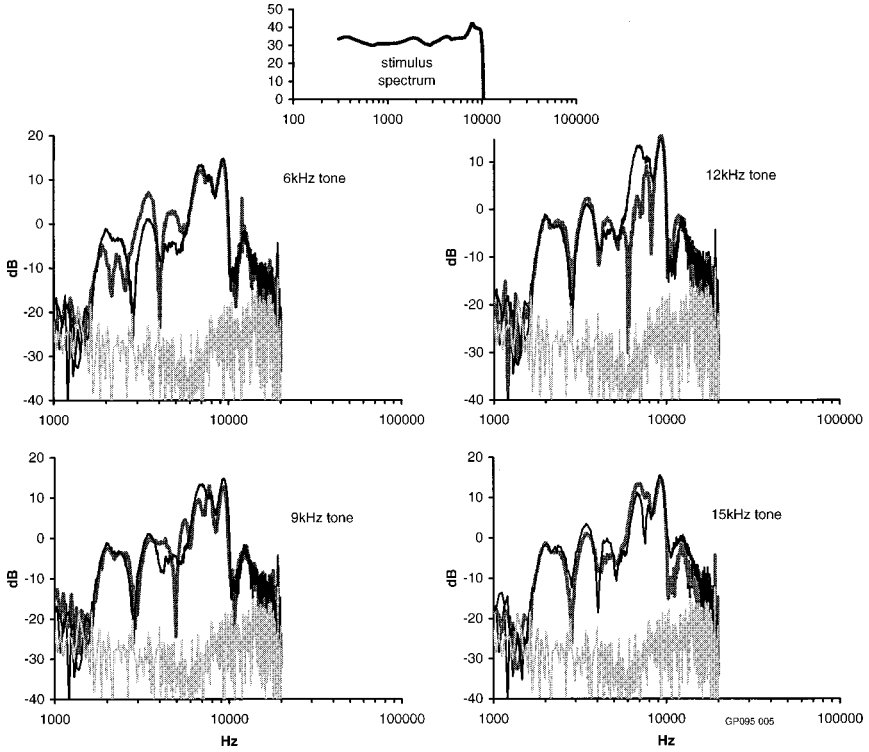

FIG. 4. TEOAEs in response to a 0–10 kHz stimulus demonstrating the effect on the emission of adding an ~70 dB SPL 6-, 9-, 12-, or 15-kHz pure tone. The lighter shading curve represents the TEOAE with the addition of a pure tone, the dark curves without a pure tone. Also shown is the noise floor.

for Figs. 1–3, with indeed very little change to the TEOAE with the addition of a 9-kHz tone.

Thus as seen in Figs. 1 and 2, changes to the TEOAE in Figs. 3 and 4 are seen at frequencies remote from the pure-tone suppressor, with enhancement of the TEOAE present in a number of cases for some part of the emission spectrum.

In these experiments, the contralateral ear was not occluded; measurement of the sound pressure level in the contralateral ear of animal GP061 revealed the level to be 20–60 dB less than the ipsilateral ear in the range 0.3–20 kHz. Destruction of the ossicular chain in the contralateral ear of animal GP061 did not alter the emission measured. The physiological nature of the results presented in this study have been verified with post mortem measurements: the magnitude of the response measured post mortem was significantly reduced, the responses post mortem with and without the suppressor tone being similar.

III. DISCUSSION

If the TEOAE truly represents a one-to-one frequency response to component frequencies of the stimulus, then simultaneous presentation of a pure tone with a click should suppress those TEOAE components which are close to the pure-tone frequency. We therefore expected the TEOAE spectra to be attenuated in amplitude close to the tone frequency. Instead, we found enhancement of the TEOAE spectra at frequencies below the pure tone, with suppression predominantly occurring in the case of tones within the passband of the stimulus, and usually not at the frequency of the tone. Thus the observed pattern of interference does not agree with the simple concept of local suppression of the TEOAE and is therefore at odds with existing theories of TEOAE production (Kemp and Chum, 1980; Kemp, 1986; Probst *et al.*, 1986; Prieve *et al.*, 1996).

There is other evidence that the existing model of TEOAE production may be incomplete. While Prieve *et al.* (1996) found that basal cochlear pathology in humans did not affect TEOAEs evoked from apical regions of the cochlea, Avan *et al.* (1993, 1995, 1997) have reported changes in low-frequency components of the TEOAE when basal-region damage was produced in guinea pigs or had been acquired in humans. Our results are more consistent with those of Avan *et al.* in that we observed changes in low-frequency components of the emission when the basal region of the cochlea was stimulated by high-frequency tones.

We considered two possible explanations for these results. First, the pure tone could have ''phase entrained'' certain components of the response, leading to enhancement of the spectrum due to phase locking (Neumann *et al.*, 1997). This is unlikely, however, because the tone was cancelled by phase inversion on alternate presentations of the click stimulus and so any component of the TEOAE which phase locked to the tone would also have been removed. Second, the tone may have modified the efficacy of transmission of the response from the site of generation back to the middle ear. There is some evidence of an enhancement effect in electrically evoked emissions, but only for acoustic frequencies below or close to the characteristic frequency of the stimulation site (Kirk and Yates, 1996; Mountain and Hubbard, 1989). We are left with no reasonable explanation in terms of existing models of emissions.

We can think of only one explanation for the unexpected effects of an interfering tone on the TEOAE. If a tone which stimulates only the basal region of the cochlea produces enhancement of the low-frequency energy in the emission, then it follows that at least a part of that low-frequency energy must have had its origin in the basal region. But the amplitude of vibration of the basilar membrane due to low-frequency components of the click stimulus will be small in the basal turn, so it seems likely that the modified emission is not a result of stimulation by those low-frequency components. On the other hand, it is well established that two high-frequency tones stimulating the basal region of the cochlea can produce significant amounts of intermodulation energy at much lower frequencies, so it is plausible that a broadband stimulus might also produce, in such a nonlinear system, quantities of intermodulation distortion. Thus we postulate that the TEOAE is actually comprised of significant quantities of intermodulation energy generated all along the cochlea, and that the introduction of an interferring tone suppresses local production of intermodulation energy over a range of frequencies.

We propose that each component frequency of the click stimulus interacts with every other component frequency to produce a range of intermodulation products. Thus each frequency in the emission may be the weighted vector sum of intermodulation products generated all along the basilar membrane; however, the predominant contribution to each frequency component would arise from more basal regions of the cochlea as emissions arising at sites below their own characteristic frequency do not propagate well back to the middle ear (Kirk and Yates, 1994) due to the low-pass filtering characteristics of the basilar membrane.[1] A pure tone will reduce the basilar membrane vibration at and basal of its characteristic place, thereby reducing the amplitude of intermodulation energy produced at that place. Significant amounts of intermodulation energy at similar frequencies will still be present due to activity at other, more apical, unsuppressed sites on the basilar membrane. The net result would depend on the precise way in which the remaining intermodulation products sum, but any outcome presumably is possible from suppression to enhancement. Furthermore, our proposal also provides a satisfactory explanation for the results of Avan *et al.* in that a reduction in mechanical vibration of the basilar membrane in basal regions of the cochlea will reduce the amount of intermodulation energy generated there and so influence the spectrum of the TEOAE, even at low emission frequencies.

The TEOAE being a composite of intermodulation distortion products with each frequency component having its origin in more basal regions of the cochlea provides a satisfactory explanation for the results seen in Figs. 1–4. Only in Fig. 1 with the addition of a 5- or 6-kHz tone and in Fig. 3 with the addition of a 3-kHz tone is there reduction in the amplitude of the TEOAE in the region of the tone frequency. In Fig. 1, the 6-kHz tone frequency is near the upper limit of the stimulus spectrum and so the energy present in the

TEOAE in this region might be dominated by more local contributions, since there is no stimulus energy at higher frequencies to contribute to the emission energy in this region. For the 5-kHz tone in Fig. 1, the dip in the emission spectrum near 5 kHz would appear to be a shifting of the dip present in the TEOAE without the addition of a suppressor tone rather than a localized suppressive effect. In Fig. 3 there is widespread reduction of the TEOAE subsequent to the addition of a 3-kHz tone. In Figs. 2 and 3, enhancement of the TEOAE is present over much of the response spectrum for tone frequencies significantly above the frequency range of the click stimulus. At face value this may appear to suggest a different mechanism for the enhancement of the TEOAE in comparison to that observed in Fig. 1. However, neural suppression tuning curves have been found to be much broader than threshold tuning curves (Schmiedt, 1982; Prijs, 1989; Delgutte, 1990) and so tones with frequencies considerably above the frequency range of the click stimulus presumably suppress some part of the basilar membrane response to the click. Figure 4 shows evidence for TEOAE present outside of the stimulus passband (above 10 kHz), this being consistent with intermodulation distortion energy contributing to the TEOAE.

It remains that there exists evidence to suggest that TEOAE energy at specific frequencies is not produced over a large extent of the cochlea (Prieve *et al.*, 1996), and so resolution of this question must await further studies.

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[1]Electrically evoked otoacoustic emissions show a low-pass filtering effect above the electrode location characteristic frequency (Mountain and Hubbard, 1989; Kirk and Yates, 1994, 1996), demonstrating that emissions that arise at a place with a characteristic frequency below the emission frequency do not propagate well back to the stapes.

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