DISTORTION-PRODUCT OTOACOUSTIC EMISSIONS: COMPUTER MODELLING AND EXPERIMENTAL DATA

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INTRODUCTION

This study concerns the computational modelling of auditory distortion products elicited by two closely-spaced stimulus tones f1 and f2. These distortion products can be perceived subjectively in psychoacoustic experiments as combination tones (CTs) and recorded objectively in the ear canal as distortion-product otoacoustic emissions (DPOAEs). Both phenomena are widely believed to have a common cochlear origin and appear to be closely related to the functioning of the outer hair cells (OHCs) [1].

In a recent article [2], we presented a computational model reproducing the essential properties of auditory distortion products. In particular, the model predicted a significant difference in the growth of the 2f1-f2 and 2f2-f1 DPOAEs with increasing level (L1=L2) of the stimulus tones. The current paper presents further computer simulations, and compares model results to corresponding experimental data. The purpose of this work is to help establish a stronger theoretical base from which to interpret clinical data.

MODEL

The model is described in detail in [2,3]. The input stimulus is equivalent to an acoustic pressure wave incident upon the head. It is processed through a series of cross-coupled concha/ear canal, middle ear and cochlear stages. The latter is a nonlinear 1-D transmission line approximation of basilar membrane (BM) motion, discretized into N=320 channels from base to apex. In each channel, a source $V_n^{ohc}(t)$ produces a nonlinear and saturating pressure, assumed to originate from the OHCs, as follows:

$$V_n^{ohc}(t) = G * (1 + |I_n(t)|/I_o)^{-0.5} * R_n * I_n(t) \qquad (1)$$

where R_n is the passive acoustic resistance of the cochlear partition, $I_n(t)$ is the BM volume velocity, I_o is a scaling constant (equivalent to a BM velocity of 3.6×10^{-3} cm/s), and G is the gain. Functionally, $V_n^{ohc}(t)$ reduces the damping of the BM at low levels and leads to level-dependent BM tuning curves. For G > 1.0, the damping of the BM can become negative (active case). The model is implemented numerically in the time domain and the sampling rate of operation is 71680 Hz. The DPOAE amplitudes are obtained from spectral analyses of the model output at the eardrum position.

Figure 1 shows the growth of the 2f1-f2 and 2f2-f1 DPOAEs in the model for increasing stimulus level (L1=L2) for f1 = 1400 Hz and f2/f1 = 1.20. The 2f1-f2 DPOAE levels increase monotonically with a slope of about 0.7 dB/dB. The levels are typically about 55-75 dB below that of the stimulus components f1 and f2. Increasing the gain G of the OHC pressure source from 0.99 (passive nonlinear case) to 1.01 and 1.02 (active nonlinear case) significantly increases the amplitude of the 2f1-f2 DPOAEs at low stimulus levels. An increase of about 15 dB is seen for L1 = L2 = 20 dB SPL. At higher levels, the saturation function in Equation (1) reduces the effective gain and prevents the BM from becoming active.

In contrast, the 2f2-f1 DPOAE levels are very small at low stimulus levels but they grow with a much steeper slope of about 2.0 dB/dB until they reach the size of the 2f1-f2 levels around L1 = L2 = 80 dB SPL. Increasing the gain G has no observable effect on the 2f2-f1 DPOAEs.

EXPERIMENTAL DATA

The measurements were collected in a quiet laboratory using a prototype distortion-product emission system under development at the Institute of Biomedical Engineering of the University of Toronto. The system consists of a virtual instrument installed on a PC equipped with a dual-channel signal generation and acquisition hardware, and an acoustic probe containing two independent miniature speakers and one miniature microphone. The system employs a special synchronous time-domain signal averaging to reduce the deleterious effect of noise on DPOAE measurements as described in [4]. The levels reported below are the average of three fit/refit of the acoustic probe. The standard deviation of the results is typically 0.5-1.0 dB. The subject (CG) was a 33-year old male with normal audiometric thresholds, tympanograms and acoustic reflexes.

Figure 2a shows the growth of the 2f1-f2 and 2f2-f1 DPOAEs for increasing stimulus level (L1=L2) in the left ear of the subject for f1 = 1378 Hz and f2/f1 = 1.25. The 2f1-f2 DPOAE levels increase monotonically with a slope of about 0.75 dB/dB, and are typically about 55-70 dB below that of the stimulus components f1 and f2. The 2f2-f1 DPOAE levels are much smaller than the 2f1-f2 levels at low stimulus levels. The gap decreases gradually with increasing stimulus level.

Figure 2b presents data for the right ear of the subject for the same stimulus parameters as in Figure 2a. Additionally, Figure 2c presents data for the left ear of the subject but for slightly different stimulus parameters f1 = 1470 Hz and

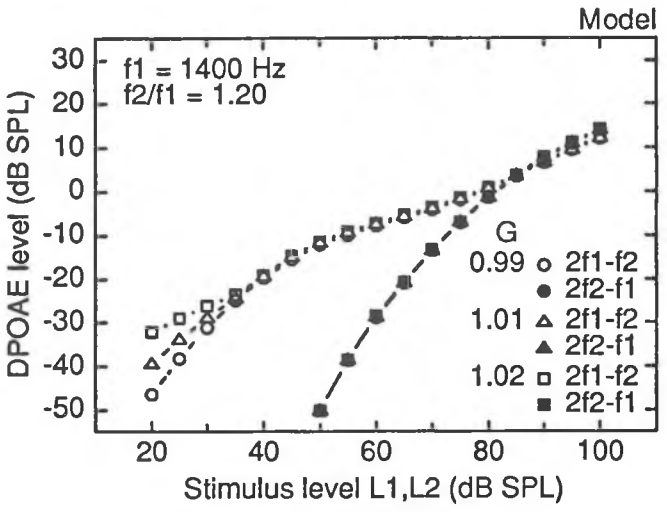

Figure 1: Model results

f2/f1 =1.20. Although the shapes and slopes of the curves in Figures 2a, 2b and 2c differ somewhat from one another, the main effect is that the difference between the 2f1-f2 and 2f2-f1 DPOAE levels decreases with stimulus level and approaches 0 dB around L1 = L2 = 75 dB SPL.

CONCLUSIONS

The model successfully predicts the general pattern of response observed for the 2f1-f2 and 2f2-f1 DPOAE amplitude versus stimulus levels (L1=L2). The best quantitative agreement between model and experimental data is obtained when the gain G of the OHC pressure source is sufficiently high to make the nonlinear cochlear model active. However, a non-active, but nonlinear, cochlear model seems sufficient to account for the experimental data at moderate and high stimulus levels.

REFERENCES

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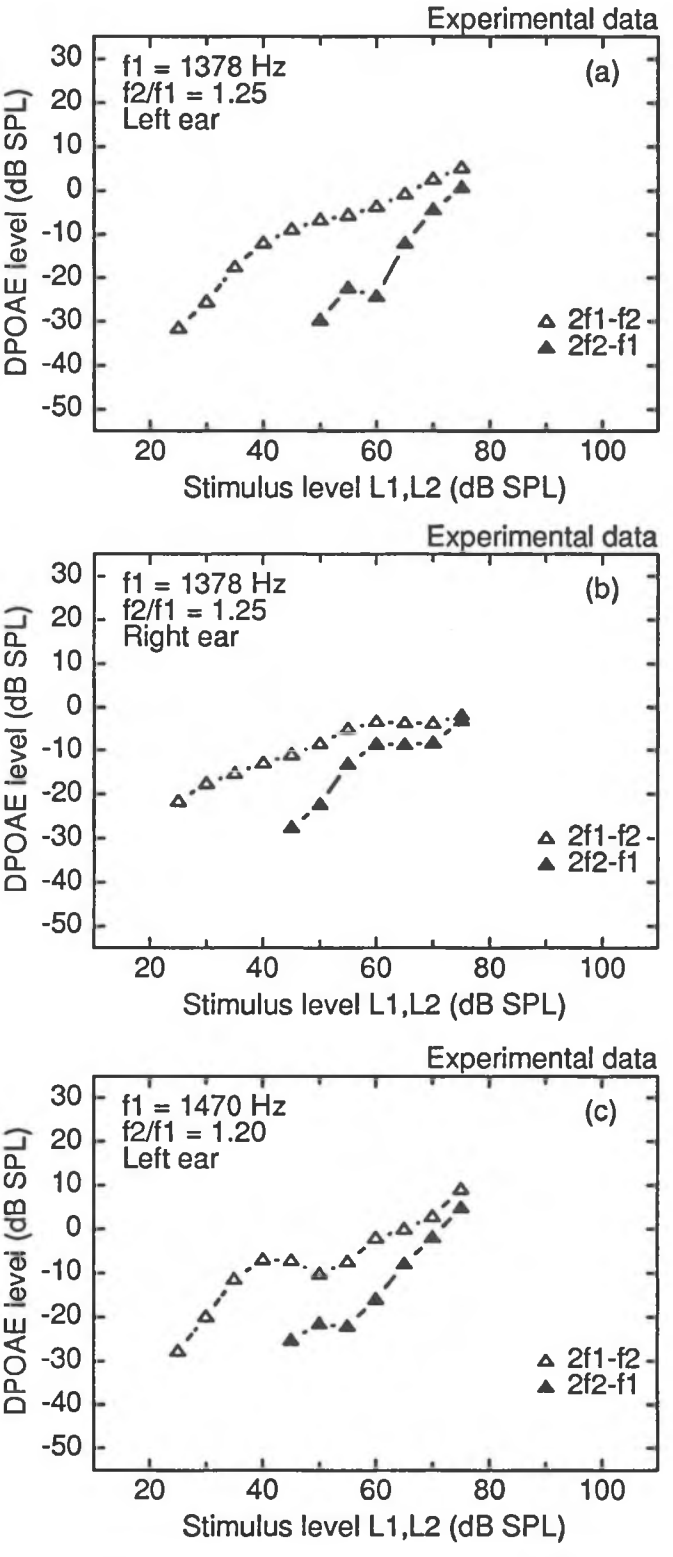

Figure 2: Experimental data for one subject