DISTORTION PRODUCT AND LOUDNESS GROWTH IN AN ACTIVE, NONLINEAR MODEL OF COCHLEAR MECHANICS

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A one-dimensional cochlear model is made active by incorporating feedback forces from outer hair cells (OHC). The mechanics of this model are nonlinear because the OHC force is limited by a saturating mechano-electric transduction stage. Distortion products in this model, measured in the acoustic pressure at the eardrum, demonstrate compressive growth at moderate levels and more rapid growth at the lower and higher levels. The compressive growth is primarily due to saturation of the OHC force. Loudness is estimated in this model from the simulated neural spike-discharge rate, which is integrated across all inner hair cells and across time. Log-loudness shows more rapid growth at low-levels and compressive growth at moderate levels, due to saturation of the OHC feedback force. Results from this cochlear model are compared with measurements of DPOAE and loudness growth in humans.

1 Introduction

The compressive growth of basilar membrane (BM) vibrations with increasing level is evident in the growth of distortion-product otoacoustic emissions (DPOAE) and in the growth of perceived loudness. In this study, we simulated both DPOAEs and loudness in an active, nonlinear model of cochlear mechanics. We then compared the growth of the simulated responses produced by this model with measurements of DPOAE amplitude and loudness in humans. By comparing the model with human data, we are able to infer characteristics of BM growth in humans, where direct measurements are not possible.

2 Model

A one-dimensional cochlear model is made active by incorporating a feedback force from outer hair cells (OHC). The micromechanics of the cochlear partition (CP) include two mechanical degrees-of-freedom at each radial cross-section (RCS). If we let ξ_b represent BM displacement and ξ_r represents the relative displacement between BM and the reticular lamina (RL), then the equations of motion can be written as

$$M_b\ddot{\xi}_b + R_b\dot{\xi}_b + K_b\xi_b + g_c R_c\dot{\xi}_c + g_c K_c\xi_c = -A_p P_f \tag{1}$$

$$M_r\ddot{\xi}_r + R_r\dot{\xi}_r + K_r\xi_r - g_c R_c\dot{\xi}_c - g_c K_c\xi_c = -g_r V_m \tag{2}$$

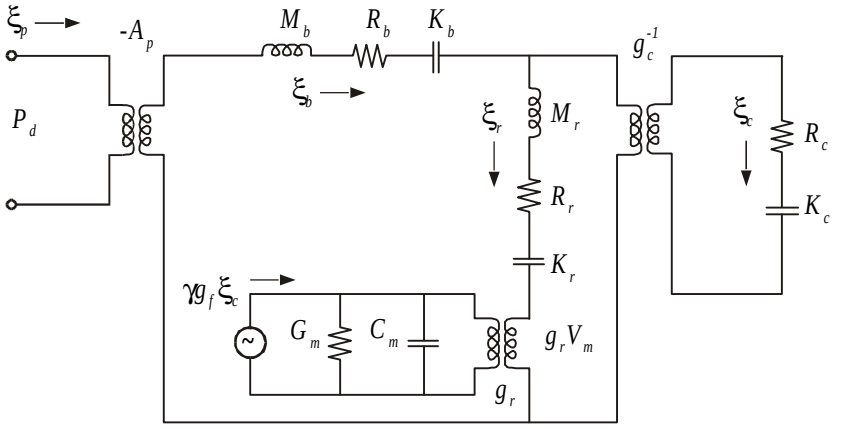

Figure 1. Electrical-analog circuit representing cochlear micromechanics, which includes two mechanical resonances and an OHC feedback force. The electrical current through the OHC is proportional to the shear displacement at low levels, but saturates at high levels. The OHC feedback force is proportional to voltage across the membrane.

where $\xi_c = g_c(\xi_b - \xi_r)$ represents the shear displacement between the RL and tectorial membrane; M_b, R_b, and K_b are the mass, damping, and stiffness associated with basilar membrane (BM) displacement; M_r, R_r, and K_r are the mass, damping, and stiffness associated with RL displacement, R_c, and K_c are the stiffness and damping associated with RL-TM shear displacement; P_d is the fluid pressure difference across the CP; and A_p is the area of the CP in a single RCS.

In equation (2), $g_r V_m$ represents a force exerted by the OHC between the RL and BM, which is proportional to the voltage across the outer hair cell membrane. The current passing through the OHC is controlled by the displacement of the hair bundle at the top of the cell. The OHC membrane voltage is governed by

$$C_m \dot{V}_m + G_m V_m = \gamma g_f \xi_c - g_r \dot{\xi}_r \tag{3}$$

where G_m and C_m are the conductance and capacitance of the OHC membrane. The second term on the RHS of equation (3) represents an electrical "displacement" current, which introduced by the reciprocal nature of the electro-mechanic transducer. An analog circuit corresponding to these equations is shown in Figure 1. This representation of cochlear micromechanics is similar to the one suggested by Neely and Stover [3]. The tuning that this model delivers to inner hair cells (IHC) is shown in the left panel of Figure 2.

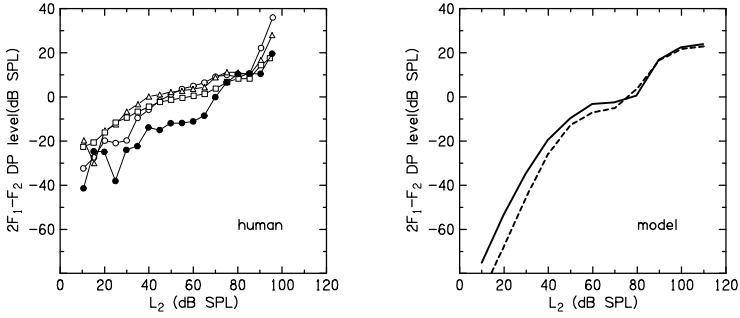

Figure 2. Input-output curves for the $2F_1$-F_2 DPOAE as a function of L_2 (the level of the higher frequency primary tone). L_1 was always 10 dB higher than L_2. In the left panel, the open symbols represent measurements in normal hearing human subjects, while the closed symbols represent measurements in a subject with a mild hearing loss at the F_2 frequency, which was 4 kHz for all measurements.

The mechanics of this model become nonlinear when a saturating mechano-electric transducer limits the OHC feedback. This nonlinearity is implemented in the model by making γ a function of ξ_c

$$\gamma(\xi) = \gamma_0 \left(1 + \left| \frac{\xi_c}{d_o} \right|^p \right)^{-1/p} \tag{4}$$

where p determines the maximum amount of compression and d_o is the displacement at which compression begins. Note that the product of γ and ξ_c is a constant (γ_0 / d_o) for large displacements. When $p = 3$, the value used for the model results presented in this paper, the minimum slope of the BM input-output curve becomes 1/3, as shown in the right panel of Figure 2. This type of saturating nonlinearity was suggested for the OHC feedback force by Yates [4].

3 Results

3.1 Distortion product otoacoustic emissions

DPOAEs (measured as acoustic pressure in the ear canal) demonstrate compressive growth at moderate levels and more rapid growth at both lower and higher levels. The left panel of Figure (3) illustrates these features. The open

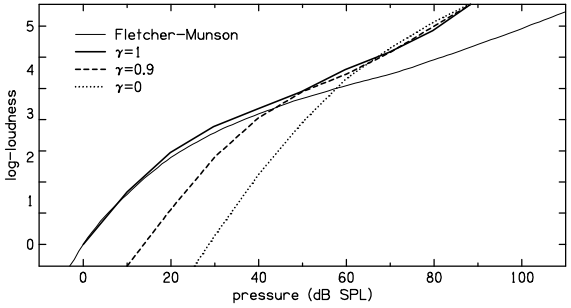

Figure 4. Loudness as a function of stimulus level. The thin line shows loudness growth for a 1-kHz tone based on the measurements of Fletcher and Munson [1]. The heavy lines show loudness growth in the model for the normal case and for two hearing-impaired cases with threshold shifts of 15 and 30 dB. The loudness values are normalized to one at 0 dB SPL for the normal ear. A base-10 logarithm was used to compute the log-loudness values.

symbols in this figure represent measurements in three, normal-hearing subjects. The filled symbols are measurements from a subject with moderate hearing loss at the F_2 frequency (4 kHz). Below 20 and above 90 dB SPL the slope of DPOAE growth in the hearing impaired ear is about the same as the slope for the normal ears. DPOAE amplitude is close to normal above 90 dB, but is smaller at lower stimulus levels

Many of the features observed in the DPOAE data are reproduced qualitatively in the model results, which are shown in the right panel of Figure (4). A very mild hearing loss (15 dB HL) was simulated in the model by reducing the OHC feedback force slightly ($\gamma_0 = 0.9$). The more rapid growth at lower and higher levels that was observed in the data is also observed in the model DPOAE. The normal and impaired DPOAE amplitudes are nearly the same in the model at the highest levels, just as the are in the human data.

The are some notable differences between the model and the measurements. The DPOAE amplitude tends to be lower in the model that in the measurements across all stimulus levels. Also, the *range* of compressive growth (with shallow slope) seems to be significantly smaller in the model (from 50 to 80 dB) than it is in the human ears (from 35 to 85 dB). Both of these differences seem to indicate that the model has insufficient gain at lower levels.

3.2 Loudness

Loudness is simulated in this model as the expected neural spike-discharge rate from the whole auditory nerve. This *whole-nerve rate* is derived from a simple estimate of the DC receptor potential of the IHC, which is then integrated across all

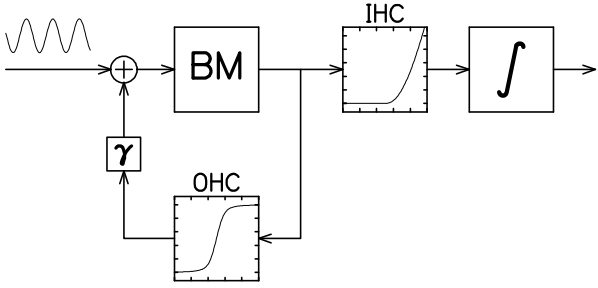

Figure 5. Block diagram of cochlear micromechanics illustrating OHC feedback force with a saturating nonlinearity and soft, half wave rectification at the inner hair cell. The nonlinear feedback onto BM generates DPOAEs in addition to providing dynamic range compression. The output of the IHC, integrated across time and place, provides an estimate of loudness.

IHCs. The IHC mechano-electric transduction is represented as a soft, half-wave rectifier. At very low levels, the voltage grows as the cube of the shear displacement. At higher levels, this transformation becomes nearly linear. A leaky integrator is applied to this voltage to the influence of the IHC membrane.

Results from model were compared with loudness data from Fletcher and Munson [1]. Log-loudness shows compressive growth at moderate levels and more rapid growth at low-levels due to the saturating OHC feedback force. At higher levels, the Fletcher and Munson data show less increase in slope, than would be suggested by either the measured DPOAE or simulated BM responses. The lack of a steeper slope in the log-loudness data at high levels may be due to saturation of spike discharge rate, although more recent loudness measurements by Florentine, Buus, and Poulsen [2] show a steeper slope at high levels than the measurements of Fletcher and Munson.

Loudness results were also obtained from the model for two different simulated hearing losses, which exhibited 15 and 30 dB of threshold shift at 4 kHz. These results are also shown in Figure 4. The two hearing-impaired loudness functions differ most from normal at threshold and are nearly the same as the normal loudness function at high levels.

4 Discussion

The active, nonlinear model of cochlear micromechanics described by equations (1) to (4) above, appears to simulate many response growth features seen in the DPOAE and loudness data. The important features of the model are illustrated in Figure (5). These features are (1) a positive feedback force onto the BM due to

OHC motility, (2) gradual saturation of the feedback force to achieve dynamic range compression at moderate levels, and (3) soft, half-wave rectification at the IHC which results in dynamic range expansion at very low levels.

4.1 DPOAEs

The steeper DPOAE growth at higher levels is clearly present in the human data above 85 dB SPL and has been previously observed in other species. In the model, the transition to a steeper DPOAE slope coincides with the upper limit of the OHC compression region.

The OHC compression range appears to be about 20 dB wider in the human data (35 to 85 dB) than in the model (50 to 80 dB). Assuming that the slope of the input-output curve is about 1/3, the model would require about 7 dB of additional gain at threshold to extend the OHC compression region by 20 dB. Additional gain at threshold would also help to improve the agreement in DPOAE amplitude between the model and the measurements.

4.2 Loudness

The excellent agreement of the model loudness and the Fletcher-Munson loudness function supports the use of cubic dependence of spike-rate on shear displacement at threshold. Above 90 dB SPL, the model loudness shown in Figure (4) grows too fast. Evidently, OHCs are unable to provide compression at these high levels, so the observed compression might be due to rate saturation at the IHCs. One interpretation of these results is that the IHCs provide a transformation to neural rate that grows as the third power of displacement at very low levels, and as the two-thirds power (Steven's law) of displacement at very high levels.

The model loudness simulates the more rapid growth of loudness for moderate levels in the impaired case. This feature is known as loudness recruitment and is due to the loss of OHC compression. Note that, at least in this model, the growth of growth of loudness will never become steeper than the expansive nonlinearity of the IHCs (i.e., displacement to the third power).

5 Conclusions

The model of OHC feedback force with saturating nonlinearity described in this paper provides a reasonable simulation of the essential features of DPOAE and loudness response growth. Comparison with the human DPOAE data suggests that the model should have about 7-dB additional gain at low levels or about 46-dB gain for a 4kHz tone at 0 dB SPL. Comparison with human loudness data suggests that the transformation from IHC displacement to neural spike rate needs to be expansive at low levels (a power of 3 at threshold) and compressive at high levels (a

power of 2/3 above 90 dB SPL). Further comparisons between the model and measurements will be required to substantiate these conclusions.

Acknowledgement

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