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# A MODEL FOR BIDIRECTIONAL TRANSDUCTION IN OUTER HAIR CELLS

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## Introduction

There is considerable evidence for the existence of a cochlear amplifier which serves to increase the sensitivity and frequency selectivity of the cochlea to low intensity sounds (Davis, 1983). Earlier models of traveling-wave amplification in the cochlea used negative damping components to supply the additional energy used to power the cochlear amplifier (Kim, *et al.*, 1980; de Boer, 1983; Koshigoe and Tubis, 1983). The negative damping models are now being replaced by more realistic feedback force models in which the fast motile response of the outer hair cell is implicated as the driving force for the cochlear amplifier (Geisler, 1986; Zwicker, 1986; Neely and Kim, 1987).

## Outer Hair Cell Motility

Cochlear outer hair cells (OHC) vary the length of their cell body in response to electrical stimulation (Brownell *et al.*, 1985). The motile property of OHCs is apparently linked to the presence of laminated cisternae adjacent to the plasma membrane. Because the OHCs are securely attached to Dieter's cells at their base and to the reticular lamina at their apex, a change in OHC length will change the separation between the reticular lamina and the basilar membrane (Brownell and Kachar, 1986).

In vitro studies of guinea pig OHCs indicate that length change is graded over a range of 1-2 $\mu$m (approximately 4% of the length) with a sensitivity of about 20 nm/mV of membrane polarization (Ashmore, 1987). Hyperpolarizing currents cause the cell to elongate and depolarizing currents cause the cell to contract. Although the sensitivity of the motile response decreases with frequency of sinusoidal electrical stimulation, length change has been observed at frequencies as high as 30 kHz (Gitter and Zenner, 1988).

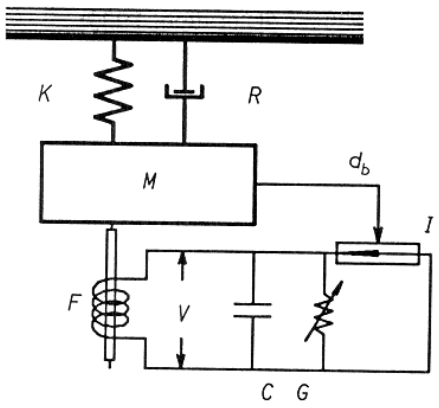


FIGURE 1 Schematic diagram of a model for an oscillating outer hair cell. The electrical components and transducers represent the motile outer hair cell and the mechanical components represent an isolated point on the basilar membrane. The transducer current $I$ is assumed to be proportional to basilar membrane displacement $d_b$ and the motile force $F$ proportional to membrane potential $V$. The model is nonlinear since the transducer current saturates for large displacements.

It is becoming increasingly clear that bidirectional transduction in OHCs involves separate and distinct transducers for *mechanical-to-electrical* and *electrical-to-mechanical* transduction. The site of *mechanical-to-electrical* transduction is at the hair bundle (HB) at the apex of the cell where electrical current entering the cell (possibly through the tips of the stereocilia) is modulated by displacement of the HB (Hudspeth and Corey, 1977). The mechanical-to-electrical transducer is essentially the same for inner hair cells (IHC) as it is for OHCs. (One difference between IHCs and OHCs, however, is that the tips of the stereocilia are imbedded in the tectorial membrane for OHCs and not for IHCs.)

The site of *electrical-to-mechanical* transduction is at the plasma membrane of the OHC along the sides of the cell body (Ashmore, 1987). This motile response is not due to actin-myosin interaction (since it occurs in the absence of ATP) and is most likely driven by transmembrane voltage along the sides of the OHC (Ashmore, 1988). It is possible that the elongation of the OHC is due to constrictive forces along the sides of the cell with no net change in cell volume.

## Oscillating Outer Hair Cell

Spontaneous otoacoustic emissions (SOAE) are low intensity narrow-band sounds which are present in about 25% of normal human ears. They are thought to be the result of continuous oscillations within the cochlea driven by one or more motile OHCs.

Fig. 1 shows a diagram of a simple model of an OHC coupled to an isolated place on the basilar membrane (BM). The mass, spring, and dashpot ($M$, $K$, and $R$) represent the mechanical properties of the cochlear partition. The current source $I$ is controlled by displacement of the BM and represents mechanical-to-electrical transduction at the OHC. The capacitor and resistor ($C$ and $G$) represent the electrical properties of the OHC membrane. Finally, the forcing function $F$ acting on the mass of the BM represents electrical-to-mechanical transduction. This motile force is modeled as being proportional to the transmembrane voltage $V$.

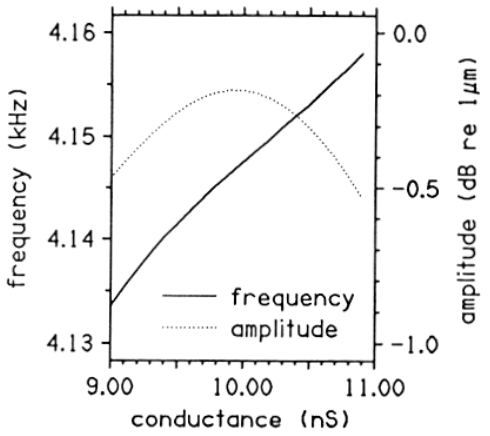


FIGURE 2 Amplitude and frequency of oscillation in the OHC model as a function of membrane conductance $G$. The curves indicate the results from numerical solution of eqs. (1) and (2) with the following parameter values:
$K$ = 62.5 dyn/cm
$R$ = 2.5 dyn·s/cm
$M$ = 0.1 gm
$G$ = 9-11 nS
$C$ = 5 pF
$I_o$ = 1 nA
$d_o$ = 1 $\mu$m
$\alpha$ = -10 dyn/V
These parameter values provide a mechanical resonance of 3.98 kHz and an electrical time constant of 0.5 ms.

If the $d_b$, $v_b$, and $a_b$ represent the displacement, velocity and acceleration of the basilar membrane, then the equations of motion can be written as

$$F = \alpha V = Ma_b + Rv_b + Kd_b \tag{1}$$

$$I = I_o \tanh(d_b/d_o) = C\, dV/dt + GV \tag{2}$$

where $\alpha$, $I_o$, and $d_o$ are transduction parameters. The mechanical-to-electrical transduction in eq. (2) is nearly linear for small displacements and saturates for large displacements (Hudspeth and Corey, 1977). The electrical-to-mechanical transduction in eq. (1) is modeled as a linear process, but has been observed to saturate for larger signals in a manner similar to the mechanical-to-electrical transduction (Ashmore, 1987).

If appropriate values are chosen for the parameters (see Fig. 2 caption), the OHC model becomes an oscillator. Numerical solutions to the equations show nearly sinusoidal oscillations with displacement amplitudes determined by the saturating nonlinearity of the mechanical-to-electrical transducer. The frequency of oscillation is determined primarily by the mechanical components and is slightly above the natural resonant frequency = $(K/M)^{1/2}$. Thus, the model suggests that SOAEs do not require the OHC to possess any intrinsic tuning (beyond the low-pass response of the cell membrane) as long as the electrical energy available to the OHC is bidirectionally coupled to the BM.

Another interesting observation with this oscillating OHC model is the effect of efferent stimulation on the frequency of oscillation. Efferent synapses at the base of the outer hair cells are thought to enhance the membrane conductance to ions when activated (Desmedt and Robertson, 1975). Fig. 2 shows that when the conductance in the OHC model is increased, the frequency of oscillation increases and the amplitude does not change significantly.

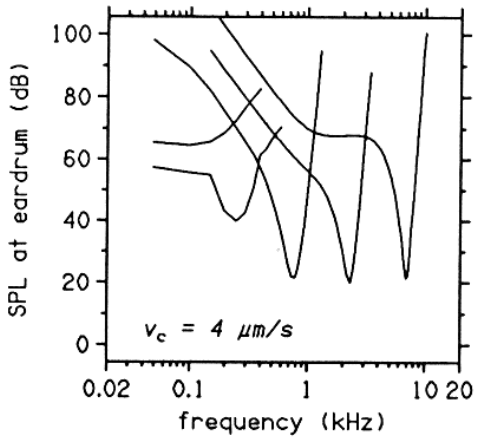


FIGURE 3 Isovelocity tuning curves for the active cochlear model without the transduction nonlinearity. The curves represent the sound pressure at the eardrum necessary to produce $v_c$ = 4 μm/s in the model at 5 places along the cochlear partition. The frequency to place map is similar to that of a human cochlea. The following model parameter values (cgs units) were used:

$K_b = 4 \cdot 10^8 \, e^{-3x}$
$R_b = 960 \, e^{-1.4x}$
$M_b = 0.004$
$K_t = 2 \cdot 10^8 \, e^{-3.2x}$
$R_t = 2000 \, e^{-1.4x}$
$M_t = 0.037$
$K_c = 2.25 \cdot 10^8 \, e^{-3.2x}$
$R_c = 0$
$K_a = 4.5 \cdot 10^8 \, e^{-3.1x}$

The frequency of the SOAE has been observed to increase in human subjects when an external tone one-half octave lower in frequency than the SOAE is presented to the opposite ear (Mott *et al.*, 1988). The contralateral acoustic stimulation is thought to stimulate efferents to the OHCs via the uncrossed olivocochlear bundle (Warr and Guinan, 1979).

## Active Nonlinear Cochlear Model

In this section, a representation of outer hair cell motility similar to the one presented in the previous section, will be used as the active element in a complete model of cochlear mechanics.

As before, let $d_b$, $v_b$, and $a_b$ represent the displacement, velocity and acceleration of the basilar membrane (BM) perpendicular to its resting plane. Let $d_t$, $v_t$, and $a_t$ represent the radial displacement, velocity and acceleration of the tectorial membrane (TM). The relevant stimulus to the HBs of the IHC and OHC will be $d_c = gd_b - d_t$ and $v_c = gv_b - v_t$, where $g$ is the lever ratio which describes the radial motion of the reticular lamina (RL) due to basilar membrane displacement.

Let $P_d$ be the fluid pressure difference across the BM and let $P_a$ represent the additional force on the basilar membrane due to OHC motility (the active element). The equations of motion can be written as

$$M_b a_b + R_b v_b + K_b d_b + (R_c v_c + K_c d_c)/g = P_d - P_a \tag{3}$$

$$M_t a_t + R_t v_t + K_t d_t - R_c v_c - K_c d_c = 0 \tag{4}$$

The active pressure $P_a$ is controlled by $d_c$ in the following way:

$$\begin{aligned} P_a &= -K_a d_c \text{ , for } d_c > 0 \\ &= 0 \text{ , for } d_c < 0 \end{aligned} \tag{5}$$

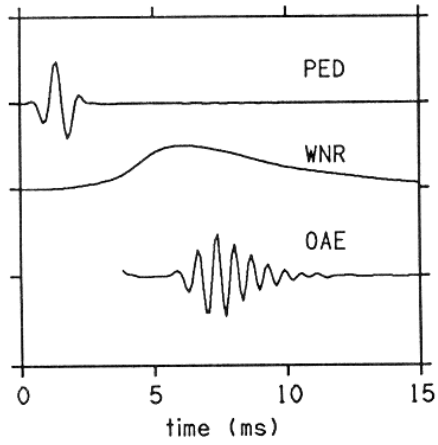


FIGURE 4 Transient response of the active, nonlinear cochlear model to a 1 kHz tone-burst stimulus. The model parameter values were the same as in Fig. 3. PED is the pressure at the eardrum showing the tone-burst stimulus. WNR is a measure of whole-nerve response computed by summing a rectified, low-pass filtered version of $v_c$ over the entire length of the cochlear partition. OAE is the same as PED except magnified to show the presence of an evoked otoacoustic emission.

The active element represented by eq. (5) is equivalent to the OHC model of the previous section except that the hyperbolic tangent is replaced by a rectifier function and the membrane capacitance is neglected. In terms of previous parameters, if $C = 0$, then $K_a = \alpha I_o/(d_o G)$. Any delay in the motile response (Ashmore, 1987) is also neglected in this model.

There are only two mechanical degrees of freedom represented by eqs. (3) and (4). Since the OHC are oriented perpendicular to the radial displacement of the TM, the active force $P_a$ is applied only to the BM in eq. (3).

Isovelocity tuning curves for a linearized version of the model are shown in Fig. 3 to demonstrate the amount of sharpening and increased sensitivity provided by the active elements. The shape of these tuning curves is similar to that of neural tuning curves in a cat (e.g., Kiang and Moxon, 1972).

The response of the model (with the nonlinearity) to a 1 kHz tone-burst is shown in Fig 4. Because of the nonlinearity, the latency of the whole nerve response (WNR), shown in the second trace in Fig 4, varies with intensity in a manner similar to the variation observed in human auditory brainstem responses (Neely, 1988). The evoked otoacoustic emission (EOAE), shown in the third trace in Fig 4, is not present in the linear version of the model.

## Discussion

Recent studies of OHC motility suggest that the OHC is capable of playing the role of the cochlear amplifier. The model of an oscillating OHC described above fits well with both the observations of SOAEs and the properties of OHCs. The dependence of the force $F$ on membrane potential introduces a -90° phase shift (lag) at high frequencies relative to displacement $d_b$ due to the membrane capacitance $C$. This makes the feedback force the equivalent of a *negative-damping* force.

In the complete (active, nonlinear) cochlear model, the fact that the nonlinearity facilitates the generation of the EOAE and makes the WNR dependent on stimulus intensity is encouraging. However, the fact that the membrane capacitance of the OHC was neglected to produce these results is contrary to the oscillating OHC model. If membrane capacitance were included (without altering the remainder of the model) then the effect of the active elements is substantially reduced for all but the lowest frequencies. It may be that the motile mechanism in the OHC is more sensitive to transducer current than membrane potential at high frequencies (Ashmore, 1987).

In the cochlear model the -90° phase shift is provided by the TM-HB resonance at $((K_t+K_c)/M_t)^{1/2}$ and, therefore, restricted to a narrow range of frequencies. Below this frequency the active force will add stiffness to the BM and above this frequency it will remove stiffness from the BM. The tuning of the *negative-damping* force in the cochlear model creates the separation between the *tip* and *tail* of the tuning curve.

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Comments and Discussion

**NEELY**

If a component of a cochlear model adds energy to the mechanical system, we call it an *active element*. The influence of the active element on the mechanical system we call an *active process*. If this active process works on a "cycle-by-cycle" basis at acoustic frequencies and increases cochlear sensitivity, we call it a *cochlear amplifier*. In the Neely and Kim (1986) model, the cochlear amplifier adds acoustic energy to the cochlear (amplifier) and, thereby, acts as a *traveling-wave amplifer*.

**KOLSTON, VIERGEVER and DIEPENDAAL**

This paper attempts to integrate several aspects of cochlear function into one cochlear model. The basic ideas are interesting and it is worthwhile to pursue them further. However, two points should be noted.

1. The results presented do not relate to a single cochlear model. Figs. 3 and 4 actually show calculations from two quite different models. The nonlinear model produces otoacoustic emissions (Fig. 4), whilst the linear model produces realistic neural tuning (Fig. 3). The nonlinearity saturates at a very low level (dm = 1nm) and so the nonlinear model will not exhibit realistic tuning. Furthermore, the fact that the linear model does not produce emissions is remarkable.
2. In Fig. 3 the cilia velocity is presented. 2-DOF cochlear models can easily produce this type of cilia motion, but usually do not succeed in coupling back the sharp tuning to the BM. It would have been interesting, therefore, if BM velocity ($V_b$) had been presented as well.

**NEELY**

Your points are well taken and I would caution the reader not to infer too much from the model results presented in this paper. I've attempted here only to present some ideas about the influence of OHCs on cochlear mechanics. I am still working on resolving the inconsistencies between the OHC model and the cochlear model.

**WIT**

If SOAE's in human ears and in frog ears are generated by the same mechanism, then the model for an oscillating outer hair cell presented in Fig. 1 in the paper by Neely should also describe frog SOAE's.

The consequence is that the model has to account for a more than 20% change of SOAE frequency, caused by a $10^0$C change of body temperature (Whitehead et al., 1986).

Furthermore, the model has to explain equally large frequency changes caused by direct current through the frogs' inner ear as shown in

Fig. 1. These frequency changes are much larger than the 0.6% change in Neely's Fig. 2.

The frequency spectra in Fig. 1 of a frog SOAE were recorded during DC flow between a wire electrode in the inner ear and a skin needle electrode. The permanent inner ear electrode was placed to record electrical correlates of SOAE's (Wit et al., this volume).

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**WILSON**

I would like to point out that OAEs, with the appropriate long delay, have been obtained in a linear model without recourse to a reverse travelling wave. Such a model would appear to be adaptable to explaining OAEs in frogs.

Sutton, G.J and Wilson, J.P. (1983) Modelling cochlear echoes: the influence of irregularities in frequency mapping on summed cochlear activity. In: Mechanics of Hearing, Eds: E. de Boer and M.A. Viergever, Delft Univ. Press, Delft, pp. 83-90.

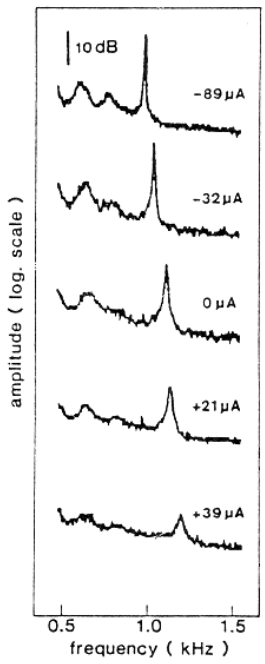


Figure 1. Frequency spectra of a frog SOAE for different values of DC through the inner ear. (Current is positive if the inner ear electrode is positive with respect to the skin electrode.)