

MICROMECHANICAL MODELS OF THE COCHLEA

We know that the inner ear separates the frequency components of complex sounds by mechanical means, but exactly how is this feat accomplished?

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Understanding human perception is at the forefront of scientific research goals today. The development of robots, virtual reality, speech coders and speech recognition devices depends on a good understanding of how we hear. The cochlea is the part of the inner ear that converts acoustic signals to the neural code that conveys auditory information to the brain. Modeling the function of the cochlea has been an active area of research since the development of the digital computer, yet several mysteries remain.¹

First is the problem of tuning. How does one explain, for example, the frequency selectivity seen in auditory nerve fibers? That is, how does the cochlea deliver to each nerve fiber only a specific, narrow range of frequencies? We now know from direct measurements within the cochlea that the tuning of auditory nerve fibers is entirely mechanical. However, as we will see, there is disagreement on the details of how this mechanical tuning is accomplished. Another aspect of the tuning problem is determining the physical properties of the structures within the cochlea that tune it over the wide range of audible frequencies.

The second major modeling mystery is the question of cochlear nonlinearities. Somehow, the cochlea compresses the large dynamic range of acoustic pressure variations that enter the ear into the much smaller dynamic range that can be processed by the sensory hair cells that detect these signals within the cochlea. (We define dynamic range as the ratio of the largest to the smallest signal amplitude that can be processed by a system.) We estimate the dynamic range of the sensory

hair cells (which is bracketed by thermal noise and signal saturation) to be about 10^3 , whereas the range of audible sound pressure levels is about 10^5 . The cochlea is a nonlinear signal processing system that is able to compress the dynamic range of input signals without significant degradation of the signal content.

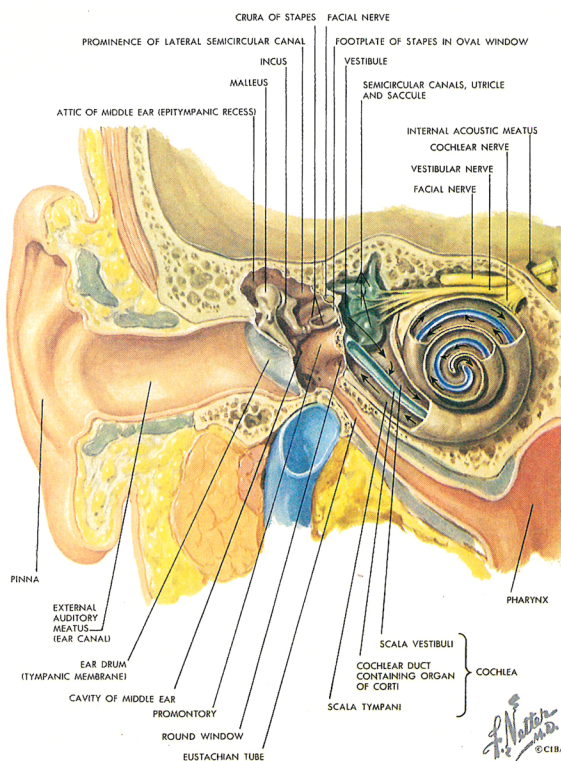
The ultimate goal of micromechanical models of the cochlea is to explain its tuning and nonlinear compression from physical principles. Only with such physical models in hand will we be able to develop more sophisticated models of sound perception, such as models of loudness. It is hoped that realizations of these models will be able to serve as the input signal processors for speech and music coders and speech recognition machines. We also hope that a full understanding of the cochlea will somehow help the hearing-impaired through the development of machine recognition of speech, improved cochlear-implant signal processing and improved hearing-aid design.

Because we are only beginning to learn how to characterize the nonlinear aspects of cochlear mechanics, linear models are still used to investigate questions of tuning. This article focuses on two competing linear cochlear models: the cochlear amplifier model and the resonant tectorial membrane model. The two models differ in their interpretations of how mechanical tuning is accomplished within the cochlea. Before we describe the models, we will look at some of the basics of cochlear structure and function.

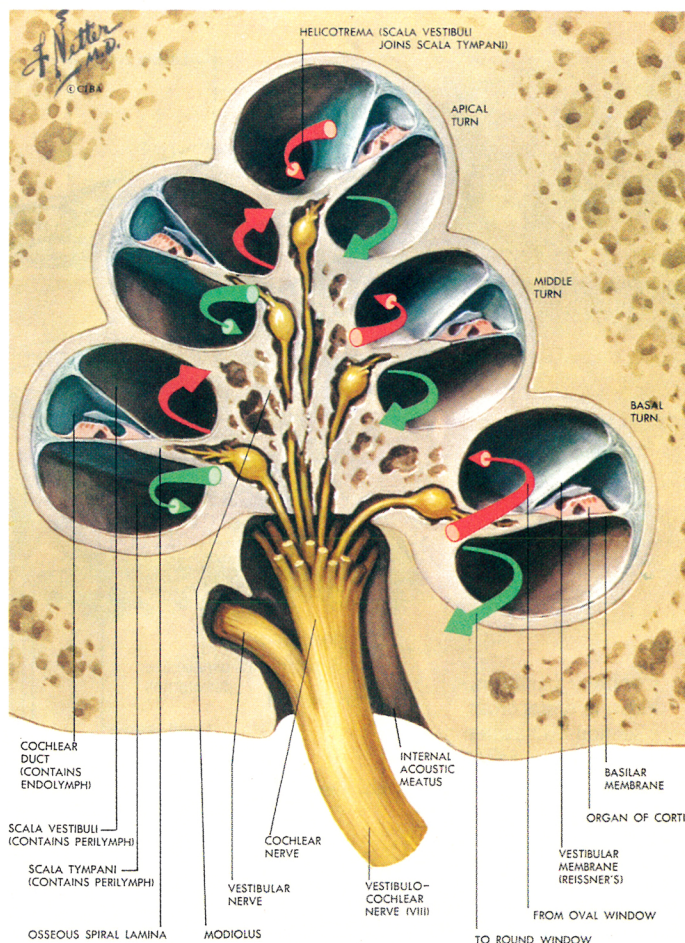
The physical cochlea

The cochlea is a spiraling, fluid-filled tunnel where fluid-borne mechanical signals are converted into the neural code carried by the auditory nerve. When the air pressure in front of the eardrum increases, the eardrum is pushed inward, moving the three small bones of the middle ear: the malleus, incus and stapes (see figure 1). The footplate of the stapes covers the oval window of the cochlea, and the movement of the stapes initiates in the cochlear fluid a

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Pathway of sound reception in the human ear (above) and cross section of the cochlea (right). The cochlea is a transducer that converts acoustic signals to neural code. (Drawings by Frank Netter, © CIBA, used by permission.) **Figure 1**



pressure wave, which propagates in a dispersive manner along the cochlear partition. This partition, which spans the width and length of the cochlea, consists of the basilar membrane, tectorial membrane and organ of Corti.

The organ of Corti is a collection of cells, including the sensory hair cells, that sit on the basilar membrane (see figure 2). Along the upper surface of the organ of Corti, called the reticular lamina, hair bundles protrude from the tops (or apexes) of the hair cells. Each hair bundle is composed of two to four rows of hairlike structures called stereocilia. Connected to the bottom (or base) of each hair cell are nerve fibers from the auditory nerve. There are two types of hair cells in the cochlea. The inner hair cells are primarily innervated (that is, connected to the auditory nerve) by afferent fibers, which deliver neural signals to the brain. The outer hair cells, on the other hand, are innervated primarily by efferent nerve fibers, which receive neural signals from the brain.

The human cochlea is believed to contain approximately 4000 inner hair cells and 12 000 outer hair cells. Each of these cell types is organized into rows of four cells standing abreast in an arc. The rows are spaced every 10 microns along the basilar membrane's length. There are typically one row of inner hair cells and three rows of outer hair cells.

The tectorial membrane lies on top of the organ of Corti and is attached, at its inner edge, to the bony spiral limbus. A thin fluid space 4-6 microns wide lies between these two surfaces, which shear as the basilar membrane

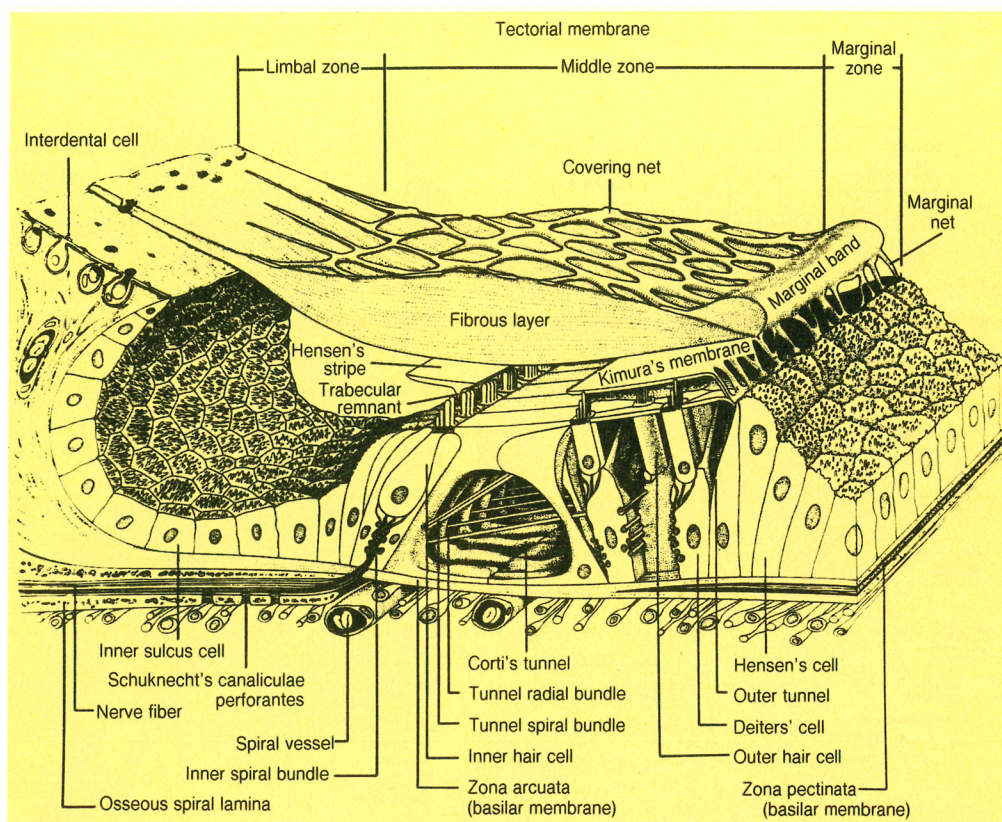
moves up and down.

Hair cells are primarily mechanoelectric transducers that convert displacement of the hair bundle (due to shearing between the tectorial membrane and the reticular lamina) into a change in the receptor current flowing through the cell. This is done by mechanical gating of ion channels that must be located in the hair bundle, probably near the top of each stereocilium. Outer hair cells also act as electromechanic transducers by converting voltages across their cell membranes into length changes. This capability is important to cochlear micromechanics, as we will describe.

Each point on the basilar membrane is mechanically tuned to a different frequency. In humans the spatial gradient is about 0.2 octaves/mm; in cats, about 0.32 octaves/mm. Roughly speaking, the cochlea acts like a bank of filters. The filtering allows the separation, with a good signal-to-noise ratio, of various frequency components of a signal. However, each filter has dynamic range compression built into its mechanical response. This nonlinearity makes the frequency response of each filter dependent on the amplitude of the acoustic signal.

All mammalian cochleas appear to function according to the same basic principles; however, the effective frequency range differs among species. For example, the range of audible frequencies is about 20 Hz to 16 kHz in the human cochlea and about 100 Hz to 40 kHz in the cat cochlea. The human basilar membrane is about 35 mm long, while the cat's is about 25 mm.

Cochlear partition,
drawn in radial cross
section. (Drawing
by Nancy Sally;
courtesy of David J.
Lim.) **Figure 2**



Measuring cochlear responses

As measurement technologies have evolved, cochlear measurements, and therefore models, have improved. The measurement of basilar membrane vibrations in animals is difficult because of the extremely small amplitude of the vibrations—less than 0.35 nanometers at the threshold of neural response—and the inaccessibility of the cochlea, which is deeply embedded in dense temporal bone in many species. To measure cochlear responses that have normal characteristics, one must keep the cochlea in extremely good condition—with its normal blood supply, for example. The sharp tuning that is typical of the healthy cochlea is lost when the cochlea is even slightly damaged. For ethical reasons, direct measurements of the motion of the basilar membrane and the responses of single nerve fibers are not possible in a living human cochlea.

The lowest sound level that the human ear can detect at 2 kHz has a diffuse-field sound pressure of about 20 micropascals. It is customary to use this sound pressure as a reference when describing the decibel levels of acoustic signals: The notation “dB SPL” is used to indicate a sound pressure level in decibels relative to 20 micropascals rms. Under normal diffuse-field conditions, this sound pressure level has an acoustic power density close to 1 picowatt per square meter. The acoustic power that enters the ear through the eardrum at the threshold of hearing is very small, about 8×10^{-17} watts. The loudest tolerable sounds are about 120 dB SPL.

Measurements from single auditory nerve fibers provide only indirect information about cochlear mechanics but have the distinct advantage that the cochlea need not be opened. Neural threshold-response tuning curves are an abundant and reliable source of data. Such tuning

curves show the sound pressure level at the eardrum sufficient to elicit a defined increase in the firing rate of an auditory nerve fiber as a function of the frequency of a pure tone stimulus. After recording the response from a nerve fiber, it is possible to mark it chemically so that the path of the fiber can be traced to the place within the cochlea where it synapses (connects) with an inner hair cell. In this way, one can make reliable frequency-to-place maps.

Typical neural threshold-response tuning curves in cats show a minimum ear canal pressure of about 14 dB SPL at the auditory nerve fibers’ most sensitive frequency and demonstrate that the fibers are sensitive to only a narrow band of frequencies. The characteristic frequency is defined as the frequency with the best sensitivity or maximum response for a given place or single neuron. Tuning curves for high characteristic frequencies tend to have narrower relative bandwidths, with steeper slopes, than low-characteristic-frequency tuning curves. The bandwidths in humans are commonly characterized as being about one-third of an octave.

The first technique that was used successfully to obtain nonlinear frequency response functions from the basilar membrane of a living (and apparently undamaged) cochlea was the Mössbauer method. This technique measures the Doppler shift in gamma radiation from a small radioactive source placed directly on the basilar membrane. Using the Mössbauer method, William S. Rhode of the University of Wisconsin at Madison observed that the amplitude of basilar membrane vibrations grew nonlinearly with increasing stimulus level.² Figure 3a shows, for example, the level dependence of the ratio of basilar membrane vibration to malleus vibration. If the mechanics of the cochlea were linear, this ratio would be independent of level. Note that the response ratio near the characteristic frequency is largest when the signal

level is smallest. This type of input-output relationship is referred to as a compressive nonlinearity. When the animal dies, or when outer hair cells are damaged, the basilar membrane response becomes smaller near the characteristic frequency, and the growth of the response with stimulus level becomes linear.

The degree of similarity between basilar membrane tuning and neural tuning is important because of its relevance to the debate between the resonant tectorial membrane and cochlear amplifier models. Figure 3b compares basilar membrane and inner hair cell tuning.³ It is generally agreed that the tuning measured at the inner hair cell is the same as that measured in an auditory nerve fiber. The inner hair cell tuning curve, however, is not identical to either the isodisplacement or the iso-velocity tuning curves for the basilar membrane, which are superimposed on it in figure 3b.

The cochlear amplifier models stress the similarities between the basilar membrane and inner hair cell tuning curves and try to account for the observed basilar membrane tuning. The resonant tectorial membrane models stress the differences between these two curves and try to account for the ratio of inner hair cell to basilar membrane frequency responses. The significance of the differences between basilar membrane and inner hair cell tuning is still a controversial issue.

The ability of outer hair cells to change their length in response to intracellular voltage changes provides clear evidence of electromechanical transduction within the cochlea and is believed to be the source of the basilar membrane compressive nonlinearity described above. This outer hair cell motility is proportional to the voltage across the cell membrane. The maximum length change observed in isolated outer hair cells is about 10% of the total length of the cell.

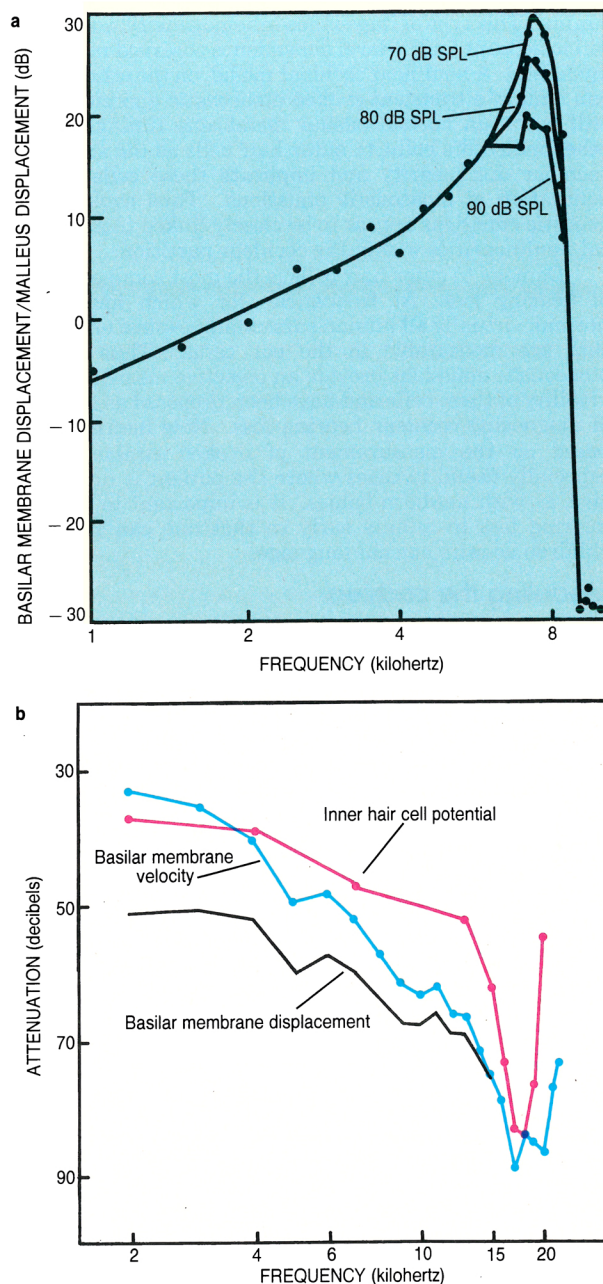
Otoacoustic emissions

The healthy ear not only detects sounds but also emits low-level sounds. Otoacoustic emissions originate in the cochlea and travel back through the middle ear to the ear canal. There are two categories of otoacoustic emissions: spontaneous emissions, which are low-level tones that persist without any external stimulus, and evoked emissions, which are generated in response to an external acoustic stimulus.

Population studies indicate that about 50% of normal human ears emit low-level tones. Since these spontaneous emissions are not present in all normal ears, their importance to hearing remains unclear.

Evoked emissions stimulated by a click or short tone burst are sometimes called cochlear echoes.⁴ These transient-evoked emissions can be recorded in the ear canal several milliseconds after the stimulus. Tones evoked in response to two external tones called primaries are known as acoustic distortion products. Distortion products are at different frequencies than the primaries, and so are easily distinguished by filtering. Stimulus-frequency otoacoustic emissions are evoked in response to a single external tone.

Two possible sources for the generation of evoked emissions are microreflections due to inhomogeneity in basilar membrane impedance along the length of the cochlea and scattering due to nonlinearity in basilar membrane mechanics. One can stimulate a transient-evoked otoacoustic emission in a cochlear model by introducing an abrupt change in the stiffness of the basilar membrane at one point; however, this model is unsatisfactory, because a small stiffness change will not evoke an emission of sufficient magnitude, and a large stiffness change will interfere with normal basilar membrane



Animal hearing data. **a:** Ratio of basilar membrane velocity at one place in a squirrel monkey cochlea to malleus velocity, as a function of frequency. This ratio depends on sound amplitude at frequencies near the characteristic frequency for that location. The level dependence indicates the existence of nonlinearity in the mechanics of the cochlea. (Adapted from ref. 2.) **b:** Stimulus levels required for a basilar membrane displacement of 350 picometers, a basilar membrane velocity of 40 microns per second measured near the stapes, and an inner hair cell dc receptor potential of 0.9 millivolts in a guinea pig. Note the similarities and differences between basilar membrane and inner hair cell tuning. (Adapted from ref. 3.) **Figure 3**

tuning. This type of impedance inhomogeneity would also fail to account for the level dependence observed in evoked emissions. A nonlinear cochlear model, on the other hand, can simulate transient-evoked otoacoustic emissions and still maintain normal basilar membrane tuning. Many other indicators point to outer hair cells as the source of cochlear nonlinearity and implicate these cells in the generation of otoacoustic emissions. Thus evoked otoacoustic emissions appear to be closely linked to mechanical nonlinearities within the cochlear partition.

Damage to outer hair cells is the most common cause of hearing loss. At frequencies for which hearing is normal, virtually all human ears exhibit evoked emissions that are measurable in the ear canal. Thus evoked otoacoustic emissions provide an objective measure of the viability of these cells and have been proposed as a means of diagnosing cochlear hearing loss. New hearing tests based on the measurement of evoked emissions are especially useful in cases where the patient is unresponsive, as with newborn babies. It is important to identify hearing loss in infants early so that one can help the children acquire normal language.

Modeling the cochlea

Models of cochlear mechanics help us make sense of the various experimental measurements and provide a framework to guide further exploration of the hearing process.⁵⁻⁷ Cochlear macromechanics is the study of the fluid mechanics of the cochlea, including its interaction with the flexible cochlear partition. Cochlear micromechanics focuses on the interaction of the structures within the cochlear partition: the basilar membrane, the tectorial membrane and the organ of Corti.

The first formal macromechanical models of the cochlea used a short-wave approximation. This approximation assumes that the wavelength of the wave that travels along the cochlear partition in response to a sinusoidal stimulus is much shorter than the cross-section diameter of the cochlea. Later models using a long-wave approximation, where the fluid mechanics problem reduces to that of a nonuniform transmission line, were more successful.

In macromechanical models of the cochlea it is

common to assume that the system is linear and that all physical variables have a harmonic time dependence $e^{i\omega t}$. A simple form of the driving-point impedance Z_p of the cochlear partition is

$$Z_p(x, \omega) = \frac{K_p(x)}{i\omega} + R_p(x) + i\omega M_p(x) \quad (1)$$

The variable x indicates distance along the length of the cochlea. The term K_p is the partition stiffness, R_p is the partition damping, and M_p is the mass. The driving-point impedance describes the ratio of the pressure difference across the basilar membrane to the "volume velocity" of the basilar membrane, as a function of place and frequency. Volume velocity is defined as the rate of change of the volume of fluid displaced by movement of the basilar membrane.

The resonant frequency $f_r(x)$ is determined by the ratio of the basilar membrane stiffness to the basilar membrane mass:

$$f_r(x) = \frac{1}{2\pi} \sqrt{\frac{K_p(x)}{M_p(x)}}$$

Waves of a given frequency do not travel beyond the place where $f_r(x)$ matches the frequency of excitation; the resonant frequency for a given place determines the traveling-wave cutoff frequency for that place.

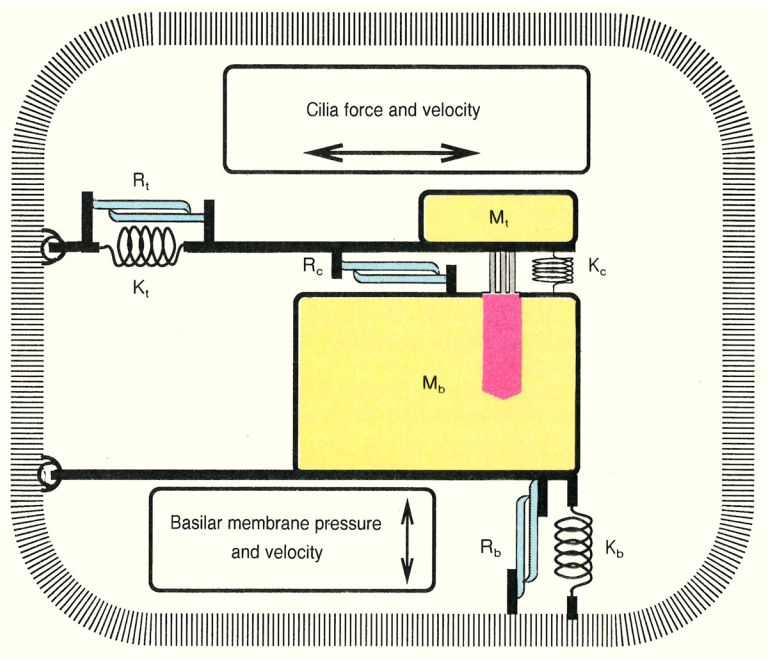
Methods have also been worked out for obtaining solutions from cochlear models with two- and three-dimensional representations of the fluid dynamics. In current modeling efforts the focus has shifted to the micromechanics of the cochlea. These efforts typically use one-dimensional (transmission line) models to simplify the process of obtaining solutions.

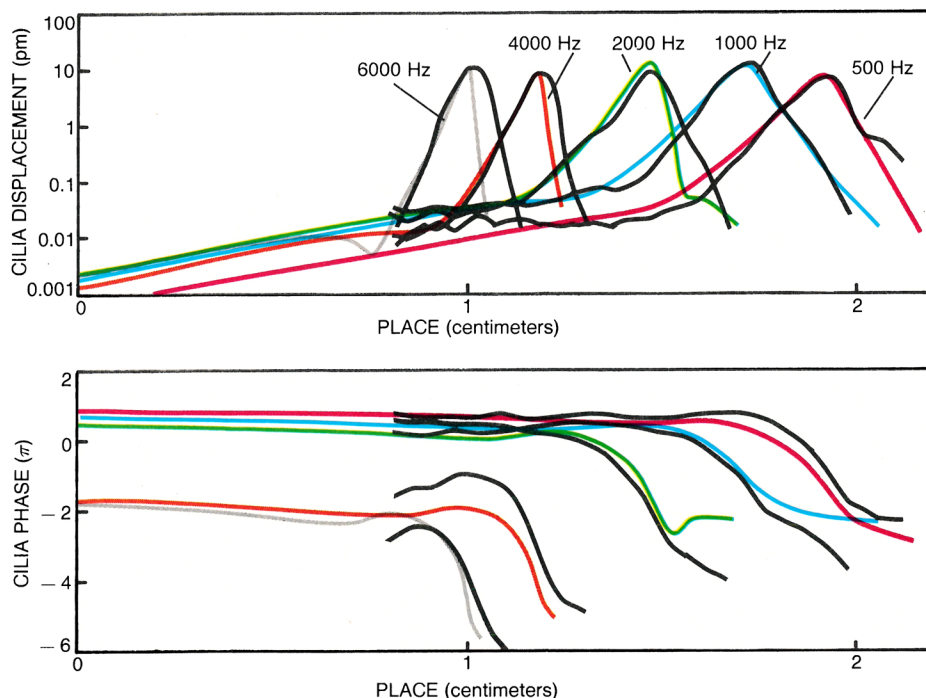
Micromechanics

Unlike the macromechanical models, each of the many micromechanical models that have been formulated is very different in its purpose and in its physics. This variation is due in part to a lack of direct experimental data from which to determine physical parameters, such as the stiffnesses of the hair bundle and tectorial

Basic model of the radial cross section of the cochlear partition. The mass of the entire organ of Corti is lumped into M_b , and that of the tectorial membrane is represented by M_t .

The two masses are coupled by the compliance K_c and damping R_c . The tectorial membrane is attached to bone by K_t and R_t ; the basilar membrane, by K_b and R_b . This representation is a common starting point for modeling cochlear micromechanics. **Figure 4**





Passive-model predictions versus measurements. Black curves represent neural data; a linear transformation has been applied that assumes a 10-picometer displacement of the inner hair cell's hair bundle (cilia) at the threshold of change in the neural discharge rate. Colored curves, labeled by characteristic frequency, represent excitation patterns computed from Allen's passive model.⁸ **Figure 5**

membrane, or the viscous drag on the hair bundle.

To organize our discussion of cochlear micromechanics, we represent each radial cross section through the cochlear partition as a linear mechanical network with two ports—that is, two points where pressure and velocity are defined. A general formalization of the relation between the basilar membrane pressure $P(x, \omega)$ and velocity $V(x, \omega)$, on the one hand, and the shear force $f(x, \omega)$ on the hair bundle of the outer hair cell and shear velocity $v(x, \omega)$ between the tectorial membrane and the reticular lamina, on the other, may be written in transmission matrix form:

$$\begin{bmatrix} f \\ v \end{bmatrix} = \begin{pmatrix} A & B \\ C & D \end{pmatrix} \begin{bmatrix} P \\ V \end{bmatrix} \quad (2)$$

A , B , C and D are complex functions of x and ω . If we knew the functions A , B , C and D , many current questions about modeling cochlear micromechanics would be answered.

The complex ratio of the force $f(x, \omega)$ on the hair bundle to the hair bundle velocity $v(x, \omega)$ defines the hair bundle load impedance Z_c . (The subscript stands for "cilia.") That impedance results from the assumed hair bundle stiffness K_c and the viscous drag R_c of the fluid in the narrow space between the tectorial membrane and the reticular lamina. It is generally assumed that

$$Z_c(x, \omega) = R_c + \frac{K_c}{i\omega} \quad (3)$$

One very important question in cochlear mechanics is the extent to which the tuning of the basilar membrane (at a given place) determines the tuning of the hair bundles of the inner and outer hair cells. We will define H_o , the transduction filter, to be the complex ratio of the hair bundle displacement of the outer hair cells to the basilar membrane displacement, as a function of frequency. In terms of the transmission matrix (and load impedance),

$$H_o(x, \omega) = \frac{1}{CZ_c + D} \quad (4)$$

The amount of additional tuning (filtering) provided by H_o is an open experimental question of long standing. Micromechanical models explore the heart of this uncertainty.

In terms of the general transmission matrix parameters, the partition impedance used in the macromechanical models (equation 1) is given by

$$\begin{aligned} Z_p(x, \omega) &= \frac{AZ_c + B}{CZ_c + D} \\ &= H_o(AZ_c + B) \end{aligned} \quad (5)$$

Some of the assumptions of macromechanical models that can be studied using micromechanical models are:

- ▷ the effect of geometrical factors, such as the length of the outer hair cells, on the cochlear partition impedance and cilia motions
- ▷ the effect of parameter relationships, such as the ratio of the tectorial membrane impedance K_t to the hair bundle impedance K_c , on the cochlear partition impedance and cilia motions
- ▷ the effect of nonlinear elements, such as the length of the outer hair cell as a function of its voltage
- ▷ the effect of adding mechanical degrees of freedom.

Figure 4 is a simplified model version of the radial cross-section geometry of figure 2. If we assume that the tectorial membrane is elastic between the place where it contacts the hair cells and its attachment to the bony spiral limbus, a significant enhancement of tuning between the basilar membrane and the inner hair cell necessarily follows (see equation 4). This example of *passive* enhancement of cochlear tuning is the basis of the resonant tectorial membrane model. If we assume that the motility of outer hair cells can be modeled as a controlled source of mechanical energy between the basilar membrane and the reticular lamina, we then have the ingredients for *active* enhancement of cochlear tuning, as in the cochlear amplifier model.

Active and passive models represent competing explanations of cochlear mechanics. We would like to define "active" and "passive," but it is difficult to agree on a

formal definition. Certainly, though, active models contain sources of mechanical energy at the stimulating frequency, and passive models do not.

Passive models

The resonant tectorial membrane model is the most successful passive model of cochlear tuning.⁸ This model assumes that the cochlear partition transforms the broadly tuned frequency response of the basilar membrane into a sharply tuned frequency response at the hair bundle of the outer hair cell. This transformation is achieved by resonant tuning of the tectorial membrane to a frequency about half an octave below the resonant frequency of the basilar membrane, which introduces a spectral zero, or antiresonance, into the transduction filter H_o , given by equation 4. The matrix elements of equation 2 are given by

$$\begin{aligned} A &= G + Z_b / GZ_t \\ B &= Z_b / G \\ C &= 1 / GZ_t \\ D &= 1 / G \end{aligned} \quad (6)$$

Here G is the ratio between the hair bundle displacement of the outer hair cells and the basilar membrane displacement (G is defined by geometry when the hair bundle load impedance Z_c is zero); the basilar membrane impedance Z_b is given by $i\omega M_b + R_b + K_b / i\omega$; and the tectorial membrane impedance Z_t is given by $i\omega M_t + R_t + K_t / i\omega$. Figure 4 illustrates the element definitions.

In this model, the outer hair cell transduction filter (equation 4),

$$H_o(x, \omega) = G \frac{Z_t}{Z_c + Z_t}$$

is a high-pass filter with a low-frequency attenuation given by $GK_t / (K_t + K_c)$. A key issue here is the magnitude of the impedance ratio, $|Z_t / Z_c|$, which in practice depends on the stiffness ratio K_t / K_c . If this magnitude is greater than 1, then the basilar membrane and outer hair cell tunings will be similar—they will differ only by G , which does not vary with frequency. If the magnitude is less than 1, then the tunings may differ.

Of equal importance is the partition input impedance $Z_p(x, \omega)$, given by equation 5,

$$Z_p(x, \omega) = Z_b + G^2 \frac{Z_c Z_t}{Z_c + Z_t}$$

which determines the tuning of the basilar membrane.

The response ratio of the displacement of the inner hair cell's hair bundle to the displacement of the basilar membrane is

$$H_i(x, \omega) = \frac{i\omega}{i\omega + 2\pi f_0} H_o(x, \omega)$$

where f_0 is the frequency above which the hair bundle of the inner hair cell follows fluid displacement rather than fluid velocity; f_0 is determined by the inner hair cell drag and hair bundle stiffness. The parameters of the resonant tectorial membrane model may be chosen so that results for mechanical inputs to the inner hair cells fit the

experimental neural threshold tuning curves closely, as shown in figure 5. As the figure demonstrates, it is important that the phase in both the model and experimental data reverse several millimeters before the peak. This 180° phase reversal is never seen in basilar membrane data, and so is strong experimental evidence in favor of a resonant tectorial membrane.

Active models

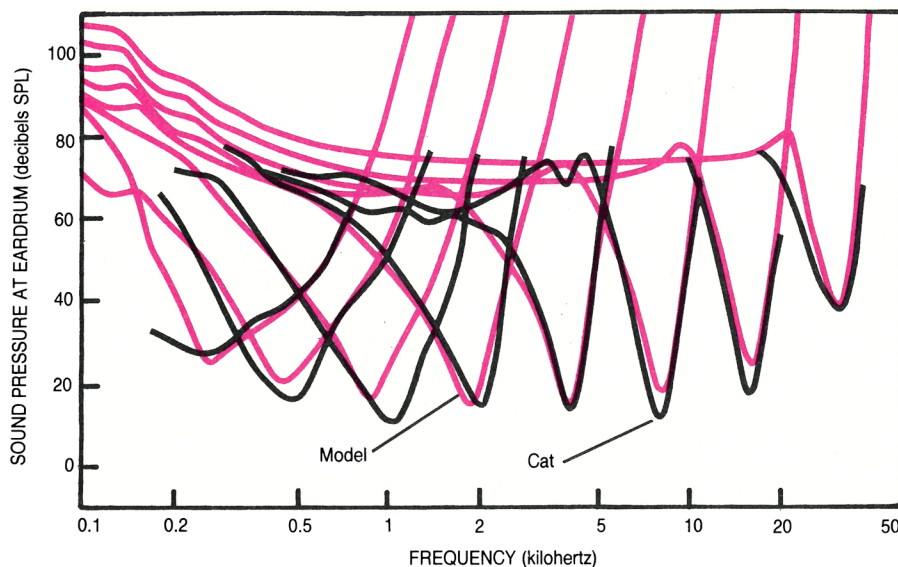
One obvious question about active cochlear models is, Are they really necessary? At least two attempts to answer this question based on detailed comparisons of measured basilar membrane responses with both active and passive models have concluded that a passive cochlear model could not account for the responses, even if the damping due to fluid viscosity were negligible.^{9,10} On the other hand, one study concluded that cochlear amplifier models are inconsistent with experimental distortion-product data measured in the ear canal.¹¹

The term "cochlear amplifier" refers to a hypothetical mechanism within the cochlear partition that increases the sensitivity of basilar membrane vibrations to low-level sounds and, at the same time, improves the frequency selectivity of these vibrations.¹² The cochlear amplifier adds mechanical energy to the cochlear partition at acoustic frequencies by drawing upon the electrical and mechanical energy available from the outer hair cells. In response to a tone, the amplifier adds mechanical energy to the cochlear traveling wave near the place of maximum response. This energy is reabsorbed at other places along the cochlear partition. The improvement in the ear's sensitivity due to the cochlear amplifier is thought to be 40 dB or more under certain conditions; however, the details of how this amplification might be accomplished are still unknown.

Cochlear amplifier models generally include a region where the real part of the impedance of the cochlear partition becomes negative. When the traveling wave generated by a sinusoidal stimulus encounters this region of negative damping, the power of the traveling wave increases abruptly. The pressure across the basilar membrane also increases in the negative-damping regions, relative to the passive case. George Zweig of Los Alamos National Laboratory has developed an ingenious negative-damping model,¹⁰ perhaps the most detailed of such models, based on Rhode's experimental data.

Hybrid cochlear amplifier models can include both negative-damping elements in the cochlear partition and a resonant tectorial membrane. For example, in Neely and Duck Kim's active model,¹³ the matrix element B of equation 6 is replaced by $Z_b / G - \gamma Z_4$, where γZ_4 represents a source of mechanical energy. This model is active for $\gamma > 0$. The model uses negative-damping elements to increase the sharpness of tuning of the basilar membrane responses, both in relative frequency response and in amplitude sensitivity at the most sensitive frequency, and to provide larger hair bundle displacements to the inner hair cell,¹⁴ as shown in figure 6. Models of this sort can achieve sharp tuning while maintaining stability, in the sense that transient responses decay with time.

Another view of active elements is that they provide the gain for a mechanical feedback loop. For example,



Active-model predictions versus measurements at eight positions along the basilar membrane. Black curves represent averages of single-nerve-fiber responses from six cats; they were obtained by M. Charles Liberman and Bertrand Delgutte at MIT. Colored curves represent tuning curves computed from Neely's active model.¹⁴ This cochlear amplifier model assumes a 300-picometer displacement of the inner hair cell's hair bundle at the neural rate threshold. **Figure 6**

David Mountain and Allen Hubbard of Boston University assume that $G = 1$, $Z_t \gg Z_c$ and the hair bundle stiffness K_c (equation 3) depends on the outer hair cell voltage.¹⁵ Based on the 90° phase shift resulting from the outer hair cell membrane capacitance, they developed a theory in which the effective hair bundle resistance R_c (equation 3) becomes negative for frequencies greater than the membrane cutoff frequency f_m but remains positive for lower frequencies.

C. Daniel Geisler proposed a feedback model where the cell body of the outer hair cell is modeled as a spring having an impedance $Z_r = K_r / i\omega$ in series with an active force generator.⁷ However, this model leads to a physically unrealizable system of equations.

Future work

Both active and passive models are reasonably successful at simulating the neural threshold-response tuning curves. Thus we need to look elsewhere to compare the two approaches. Differences between *nonlinear* resonant tectorial membrane and cochlear amplifier models have yet to be investigated.

The cochlear amplifier and resonant tectorial membrane models differ in their interpretations of the responses of damaged cochleas. In cochlear amplifier models, the loss of sensitivity of the cochlea with damage is interpreted as a loss of amplifier gain. In passive models, the loss of sensitivity has been interpreted as resulting from a change in the stiffness of the basilar membrane.⁷

The discovery of outer hair cell motility demonstrates the existence within the cochlear partition of a potential source of mechanical energy that is suitably positioned to influence vibrations of the basilar membrane. It is still an open question whether this source of energy is sufficient to power a cochlear amplifier at high frequencies.

One possible advantage of the cochlear amplifier models is that they may improve the signal-to-noise ratio in front of the inner hair cell. A weakness of the cochlear amplifier models has been their lack of specificity about the physical realization of the active elements. Until we have a detailed physical representation for the cochlear amplifier, resonant tectorial membrane models will have the advantage of being simpler and more explicit.

The resonant tectorial membrane model has been in disfavor because many feel it does not account for basilar membrane tuning. This widely held belief is due largely to

the experimental results of physiologists who have measured the basilar membrane-ear canal transfer function and found the tuning of basilar membrane velocity to be similar to neural threshold-response data. The experimental basilar membrane data, however, are largely unconvincing on this point. The question of whether one needs an active model to simulate measured basilar membrane responses is still being debated.

Further development of models of cochlear micromechanics would benefit from better estimates of the amplitude of hair bundle displacement at a given sound pressure level at the inner hair cell. Better estimates also are needed of the ratio of the basilar membrane frequency response to the inner hair cell frequency response, at both high and low frequencies.

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