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Cochlear Signal Processing

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This chapter describes the mechanical function of the cochlea, or inner ear, the organ that converts acoustical signals into neural signals. Models of the cochlea are important and useful because they succinctly describe the principles of the operation of the preneural portion of the hearing system. Many cochlear hearing disorders are still not well understood, and if systematic progress is to be made in improved diagnostics and treatment of these disorders, a clear understanding of basic principles is essential. The literature is full of speculations about various aspects of cochlear function and dysfunction. Unfortunately, we still do not have all the facts about many important issues, including how the cochlea attains its frequency selectivity. However, the experimental body of data has been growing at an accelerating pace as greater attention has been focused on this and other important and related issues.

Several topics will be covered here. First, the history and concepts behind the early cochlear models will be described, including extensions that have taken place in recent years. Next, recent modeling efforts in cochlear micromechanics are described. These models are intended to describe the mechanics of the tectorial membrane and the hair cells in greater detail. This leads to a discussion of the difference between basilar membrane, hair cell, and neural tuning. Finally the success of several of the micromechanical models is discussed.

FUNCTION OF THE INNER EAR

The purpose of modeling the cochlea is to help us understand how auditory signal processing is performed. The signals from 30,000 neurons represent the output of the human cochlea. These neurons encode 3,500 cochlear inner hair cell signals, which are filtered versions of the sound pressure at the tympanic membrane. In other words, each hair-cell signal has a limited frequency content, with a frequency spectrum that depends on the hair-cell location along the basilar membrane. In the cat, approximately 20 neurons encode each of these narrow-band hair-cell signals using a neural timing code, whereby the time between neural pulses carries the information being signaled into the auditory central nervous system.

We describe the cochlear signal processing that ensues by two separate means. First, we describe the signal representation at various points in the system. Second, we refer to models of the auditory system. These models are our most succinct means of conveying the results of years of detailed and difficult experimental work on cochlear function. An alternative way of describing our knowledge of the cochlear function (which we try not to use) would be to describe the multitude of experimental

results. This body of experimental knowledge has been very efficiently represented (to the extent that it is understood) in the form of mathematical models. When the experimental results are at variance with the model or when no model exists, the model is not a useful description, and the more basic description, using the experimental data base, is necessary. Several good books and review papers are available that make excellent supplemental reading (3,29,36,40,41,46).

For pedagogical purposes the inner ear may be functionally divided into several subcomponents. From Figs. 1 to 3, three major divisions may be defined and are classified here as: (a) macromechanics, (b) micromechanics, and (c) transduction.

Macromechanics describes the fluid motions of the scalae and assumes for analysis purposes that the basilar membrane is frequently treated as a dynamic system having mass, stiffness, and damping. Micromechanics describes the details of the motion of the organ of Corti, the inner and outer hair cells, the tectorial membrane, pillar cells, and the motion of the fluid in the space between the reticular lamina and the tectorial membrane. Transduction describes the electrochemical response of the inner hair cell to basilar membrane motions. This topic, however, is beyond the scope of this chapter.

There is a great deal of diverse opinion in the literature about several critical issues because of experimental uncertainty. For example, it is very difficult to observe experimentally the motion of the basilar membrane in a functionally undamaged cochlea. Furthermore, questions regarding the relative motion of the tectorial membrane to other adjacent structures are largely a matter of conjecture. Such questions are therefore at present best investigated by theoretical means. As a result, a variety of opinions exist as to the detailed function of the various structures.

On the other hand, firm and widely accepted indirect evidence exists on how these structures work. Since this indirect evidence takes on many forms, such as morphological, electrochemical, mechanical, acoustical, biophysical, these data are probably best related via a model.

COCHLEAR MACROMECHANICS

The first widely recognized model of the cochlea, attributed to Helmholtz (14), is described in an appendix of his book *On the Sensations of Tone*, which was first published in 1862. Helmholtz likened the cochlea to a bank of highly tuned resonators that are selective to different frequencies, much like a piano, where each string represents a different place on the basilar membrane. In fact, the model he proposed was unsatisfactory because it omitted many important features, the most important of which is the cochlear fluid that couples the mechanical resonators together.

It was not until the experimental observations of von Békésy in 1928 on human cadaver cochleas that the nature of the basilar membrane traveling wave behavior was unveiled. Typical fluid motions in the cochlea are shown by the arrows in Fig. 1. Figures 2 and 3 show an expanded view with details of the cochlear duct. Von Békésy found that the cochlea is like a linear "dispersive" transmission system in which different frequency components that make up the input signal travel at different speeds along the basilar membrane, thereby isolating those various frequency components at different places along the basilar membrane. He described this dispersive wave as a "traveling wave," which he observed using stroboscopic light in

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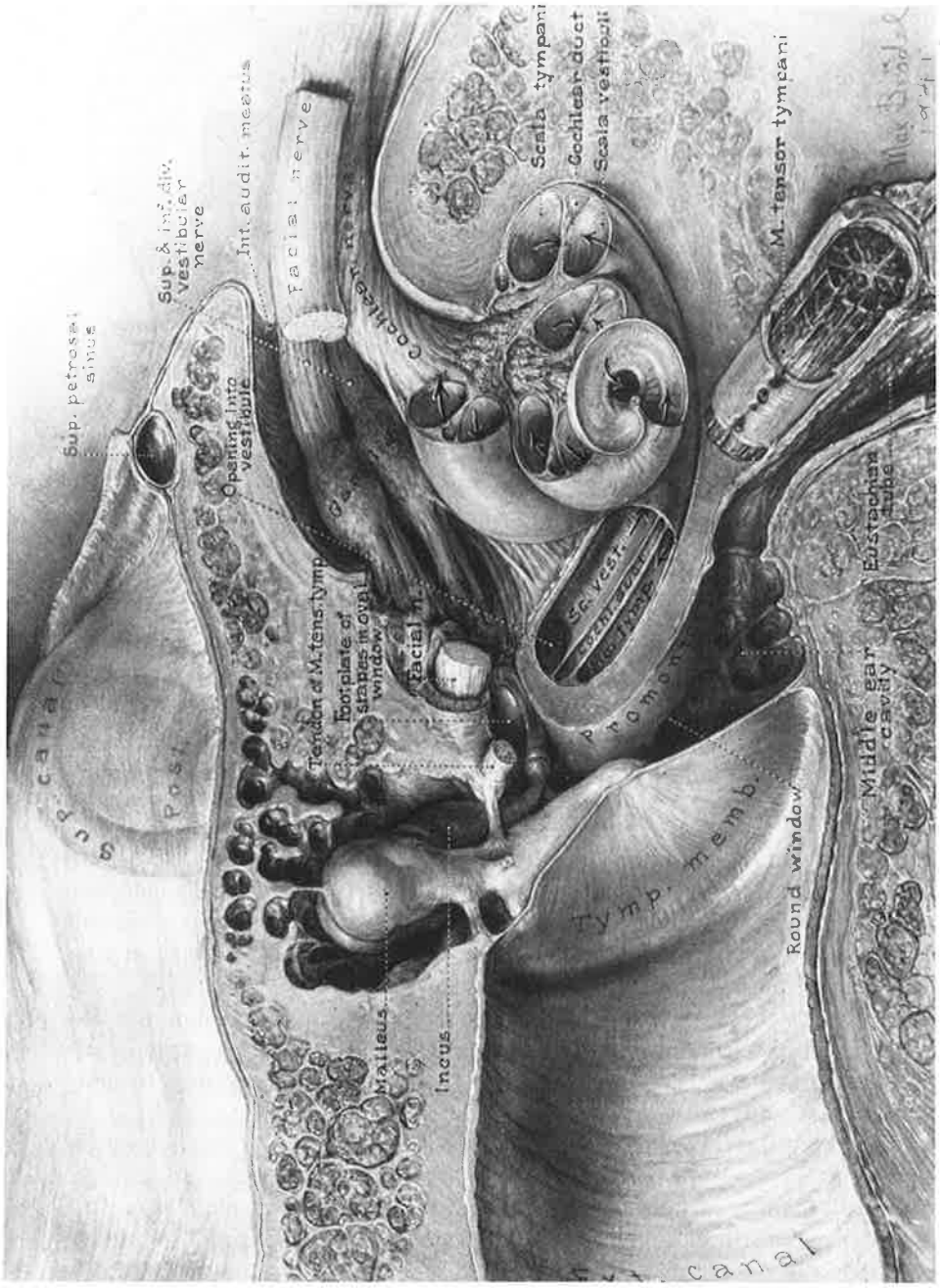


FIG. 1. In this detailed drawing of the human cochlea, most of the major components are identifiable. Sound is directed by the pinna down the ear canal where it becomes a plane wave owing to the ear canal's small diameter. The sound is transmitted to the cochlea via the ossicles. The motion of the stapes displaces the fluid in the vestibular chamber of the cochlea. An equal amount of fluid is displaced at the round window since the net volume of fluid within the cochlea is constant. (© 1941, Max Brödel, "Three unpublished drawings of the anatomy of the human ear." W. B. Saunders, 1946.)

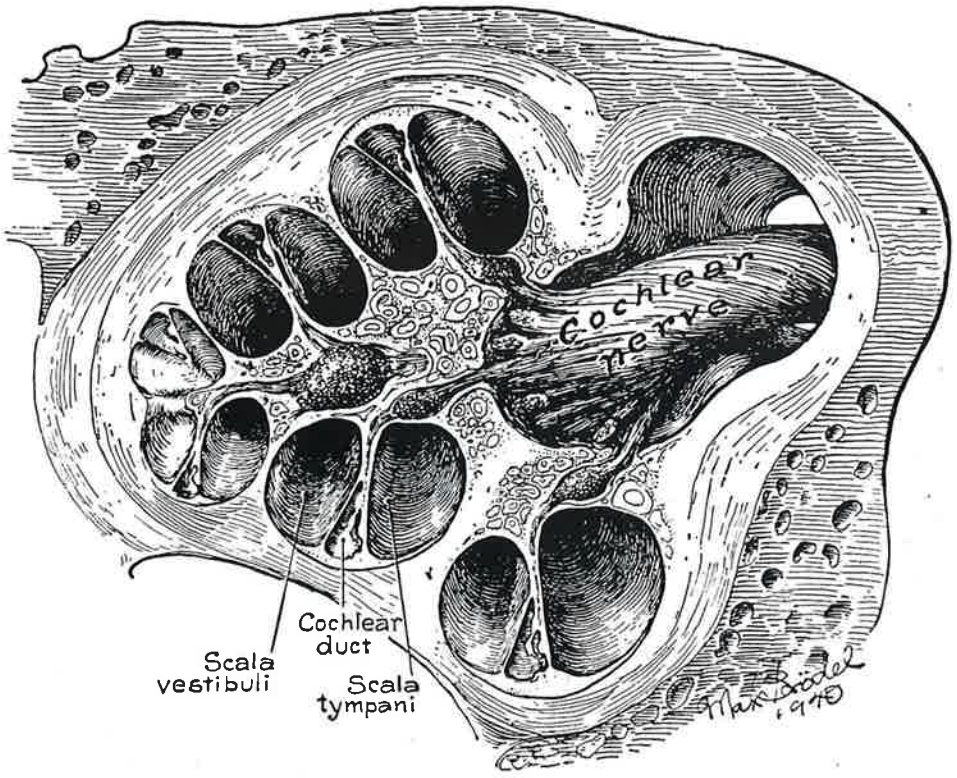


FIG. 2. As we move into the inner ear, we see the various fluid-filled chambers. The cochlear nerve forms the central core of the cochlea and extends into the VIIIth nerve, which also comprises the facial and vestibular nerve bundles. The cochlear duct, defined as the space between Reissner's membrane and the basilar membrane, is at an 80-mV potential This potential is important in the transduction process. (© 1940, Max Brödel from *1940 Year Book of the Eye, Ear, Nose and Throat.)*

a dead human cochlea, at sound levels well above our pain threshold, i.e., 120 dB SPL and above. Sound levels of this magnitude were required to obtain displacement levels that were observable under his microscope. These pioneering experiments were so difficult and important that von Békésy received the Nobel prize in 1961 for his experimental observations.

Through the years these experiments have been greatly improved, but von Békésy's fundamental observation of the traveling wave still stands. His original experimental results, however, are not characteristic of the responses seen in more recent experiments in several ways.

Today we find that the traveling wave has a more sharply defined location on the basilar membrane for a pure tone input than observed by von Békésy. In fact, according to more recent measurements, the response of the basilar membrane to a pure tone can change in amplitude by about five orders of magnitude per millimeter of distance along the basilar membrane. To describe this response, it is helpful to call on one of the early models of macromechanics, the transmission line model, which was investigated by Zwislocki (44,45) and later elaborated by Peterson and Bogert (28). This model is also frequently called the one-dimensional model.

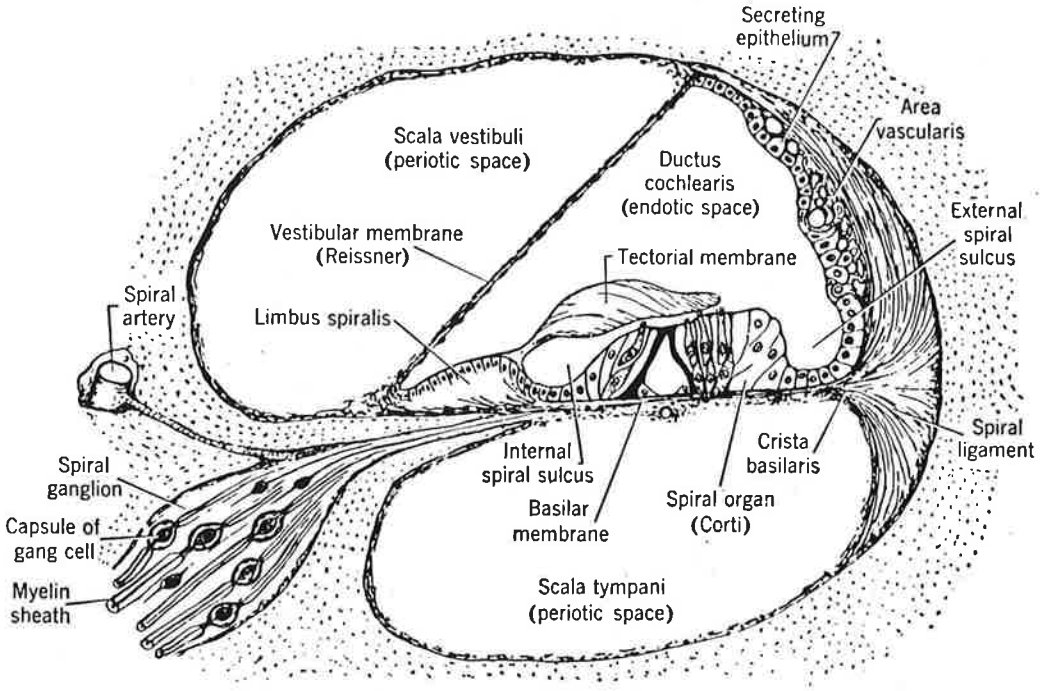


FIG. 3. We show here a more detailed cross section of the cochlear canal with tectorial membrane, cochlear nerve, basilar membrane, and other structures of significance. (© 1943, Rasmussen.)

THE TRANSMISSION LINE MODEL OF THE COCHLEA

Between the years of 1931 and 1950, Ranke (30) formulated the first hydrodynamic models of the cochlea. Ranke's main contribution was his studies of the fluid effects in the cochlea. He came to many important and fundamental findings, but his work remained either greatly misunderstood or ignored and is only now beginning to be appreciated (39,41).

The transmission line model of Zwislocki (45) was first introduced in 1945 as a simplified version of the more complete formulation of Ranke (30). Zwislocki's theory was more easily evaluated, but, as Ranke has pointed out (30), it was not as accurate as the more complete theory. A modern version of the Zwislocki model is shown in Fig. 4. The stapes input pressure p_{in} is at the left, with the input velocity v_{in}, as shown by the arrow, corresponding to the stapes velocity. This model represents the mass of the fluids of the cochlea as electrical inductors. Frequently electrical circuit networks are useful in describing mechanical systems. This is possible because of an electrical to mechanical analog that relates the two systems of equations, and the electrical circuit elements comprise an accepted standard for describing these equations owing to their frequent use. From the circuit of Fig. 4, it is possible to write the equations that describe the system, and many engineers and scientists find it quicker to read these circuit diagrams than to interpret the equations.

Different points along the basilar membrane are represented by the cascaded sec-

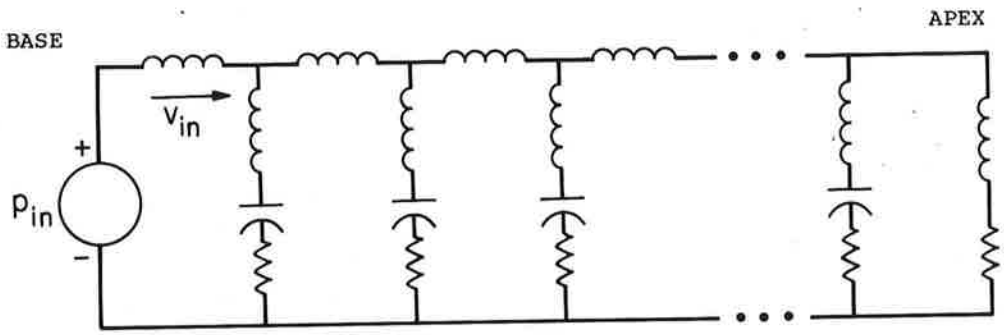


FIG. 4. The most commonly exploited basilar membrane-cochlear model is the transmission line model. In this model the inductors represent the mass of the cochlear fluid (series elements) and the basilar membrane mass (shunt inductors). The inductor values are frequently assumed to be independent of their position along the length of the cochlea. The stiffness of the basilar membrane is represented by the shunt capacitors. The stiffness is position dependent and is usually assumed to vary exponentially with position. The basilar membrane is stiffest (smallest capacitance) near the stapes. Thus the resonant frequencies of the shunt elements, taken in isolation, are largest at the stapes (base) and smallest near the helicotrema (apex). This model, called the transmission line model or one-dimensional model, has been an important research tool since it was introduced by Zwislocki in 1948 (44). The model does not have as sharp a high frequency cutoff as two- and three-dimensional models. However, it does capture many of the essential features of the system in a qualitative way, such as the traveling wave observed by von Békésy.

tions of the transmission line model. Thus the position along the model line corresponds to the longitudinal position along the cochlea. The series (horizontal) inductors represent the fluid inertia along the length of the cochlea, and the elements connected to ground (the common point along the bottom of the figure) represent the mechanical (acoustical) impedance of an element of the corresponding section of the basilar membrane. Each inductor going to ground represents the mass per unit length of the basilar membrane section, whereas the capacitor represents the compliance (stiffness) of the section of basilar membrane. The compliance is believed to vary systematically with a stiffness that decreases exponentially along the length of the cochlea. Thus each piece of basilar membrane is tuned to a different frequency, since the stiffness changes with position. For convenience, we assume here that the mass of the basilar membrane remains constant along its length, which roughly speaking, seems to be the case.

During the following discussion it will be necessary to introduce the concept of impedance, which may be foreign in its most general form, but is actually a simple concept. Impedance is defined under conditions of pure tone stimulation; thus impedance is a function of frequency. For example, the impedance of the tympanic membrane (TM) is defined as the pure tone pressure in the ear canal divided by the resulting TM volume velocity (the velocity $\times$ the area of motion). The pressure and velocity referred to here are conventionally described by complex numbers to account for the phase relationship between the two. Other common impedance definitions are the voltage/current ratio in an electrical circuit, and the force divided by the velocity in mechanical systems.

To understand the inner workings of our circuit of Fig. 4, let us assume that we excite the line at the stapes with a sinusoidal current of frequency f. Because of conservation of charge (charge cannot be created or destroyed in this circuit), the

total current through the basilar membrane must equal the current at the stapes. The physical law that we are modeling is not conservation of charge, since the cochlea is not an electrical circuit, but conservation of fluid mass or, equivalently, conservation of the fluid volume within the scalae since the fluid is incompressible.

When the stapes is displaced, thereby producing a fluid volume displacement in the upper scala (Figs. 2 and 3), the net volume displacement of the basilar membrane must displace an identical volume. Simultaneously, the round window membrane connected to the scala tympani must bulge out by an equal amount. In practice the motion of the basilar membrane is quite complicated. However, the total volume displacement of the basilar membrane, at any instant of time, must be equal to the volume displacement of the stapes or of the round window membrane.

Consider next where the fluid current v_{in} will flow or where it can flow. For a given input frequency, the basilar membrane impedance has a minimum at one point along the length of the cochlea. The impedance of interest here is that of each group of three elements in the series in Fig. 4, i.e., the inductor-capacitor-resistor combination going to ground at each point along the length of the cochlea. These three elements in this configuration have special significance because at one frequency the impedance of the inductor and capacitor cancel each other, and the only impedance element remaining is the impedance (resistance) of the resistor. Thus, at one point along the length of the cochlea, for a given frequency, the impedance is small, namely that point where the basilar membrane compliance reactance cancels its mass reactance. This point is called the resonant point. At that point the basilar membrane appears to have a hole in it (e.g., the flow resistance is all that remains of the impedance). To the left of the resonant point, the basilar membrane is increasingly stiff (having a large capacitive impedance), and to the right of the resonant point, the impedance is a large mass reactance (inductive impedance). In fact, in this region the impedance is largely irrelevant since little current will flow past the hole. Thus the fluid current has maximum flow basal to where the impedance has its minimum.

Of course the above description is dependent on the input frequency f, since the location of the hole, or impedance minimum, is frequency dependent. If we were to put a pulse of current in at the stapes, the highest frequencies that make up the pulse would be filtered out near the stapes, whereas the lower frequencies would propagate down the line. As the pulse travels down the basilar membrane, the higher frequencies are progressively removed, until almost nothing is left when the pulse reaches the right end of the model (the helicotrema end or the apex of the cochlea).

From this description it is possible to understand why the various frequency components of the signal are mapped out on the basilar membrane.

Let us next try a different mental experiment with this model. Suppose that the input at the stapes were a slowly swept tone or chirp. What would the response at a fixed point on the basilar membrane look like? In Fig. 5, we show the model frequency response magnitude of the basilar membrane. This is the ratio of the basilar membrane displacement at one point along its length (the output) to the stapes displacement (the input), as a function of the frequency f at the input. The response is a bandpass response, with a shallow low-frequency slope and a very sharp high-frequency slope. We see in Fig. 5, that the maximum relative amplitude of vibration for the particular point chosen was at 1 kHz.

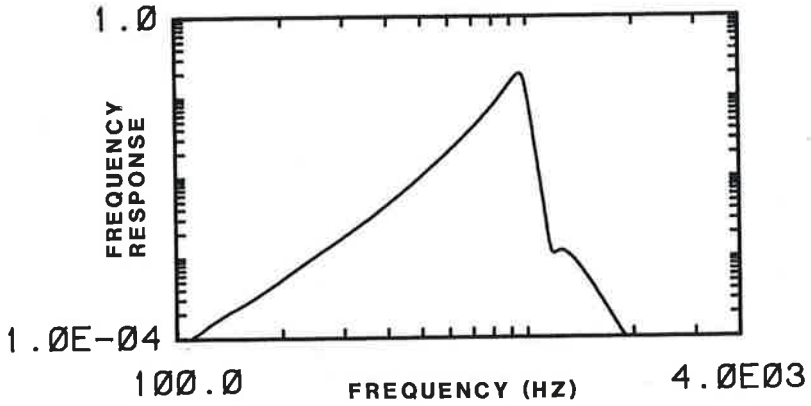


FIG. 5. When we view the model at a given place on the basilar membrane as a function of frequency, the response is found to be a bandpass filter. The slope on the high-frequency side of a real cochlea is place dependent and varies from 50 dB/octave for a low-frequency place, near the helicotrema, to more than 500 dB/octave for a high-frequency place in the base. This latter slope would give a 42-dB change in output for a semitone change in frequency (a change corresponding to going from C to C# on a musical scale). By comparison, the slope for the model on the low-frequency side of the model filters is quite shallow and between 12 to 18 dB/octave.

INADEQUACIES OF THE ONE-DIMENSIONAL MODEL

The transmission line model was a most important development since it was in agreement with the experimental evidence of the day, and it is based on a simple set of physical principles, i.e., conservation of fluid mass and a spatially variable basilar membrane stiffness. In fact, this model was the theory of choice until improved experimental observations were available in the late 1960s and early 1970s.

In 1976 Zweig and colleagues pointed out that accurate, but approximate, solutions for the transmission line equations could be found by the use of a method in physics called the "WKB" method (41,43). As further results became available, it eventually became clear that the one-dimensional transmission line theory was not totally satisfactory, since that theory did not agree with the more detailed and complete descriptions derived from a more rigorous analysis. This point was first made by Ranke (30), and again much later by Lesser and Berkley (23). It is now possible to compute the response of a two-dimensional (5) and even the response of a three-dimensional geometry (9). As the complexity of the geometry of the models approached the physical geometry, the solutions tended to display steeper high frequency slopes and therefore increased frequency selectivity.

A great deal of neural data from the VIIIth nerve is available that defines quite precisely the input–output properties of the cochlea at threshold levels. However, since the signals undergo significant transformations between the basilar membrane and neural measurement point, one cannot directly compare neural response curves with the basilar membrane model, at least not without careful consideration. We ultimately seek a model that accurately describes the 3,500 human inner hair cell outputs or the 30,000 neural signals.

What is important is that the frequency response, as computed by the transmission line model of the basilar membrane motion, is quite different from the response as estimated from the nerve fiber measurements. The difference can be on the order

of 20 to 40 dB (20 dB is a factor of 10, and 40 dB is a factor of 100) and appears to be even greater under some conditions. Thus, when the two-dimensional models showed sharpened responses relative to the transmission line model, the hope was that these more detailed models would converge to the response measured in the nerve fiber. Although a significant increase in sharpness was found, the desired convergence has not occurred.

NONLINEAR EFFECTS

A second area where the existing one-dimensional theory is inadequate follows from the nonlinear phenomena that have been experimentally observed, such as:

1. the frequency-dependent response-level compression as first observed by Rhode (31,32) in the basilar membrane response;
2. the frequency-dependent response-level compression as observed by Russell and Sellick (34) in the inner hair cell receptor potential;
3. the frequency-dependent response-level compression as observed by Kiang and Moxon (20), Allen and Fahey (4), and others, as measured neurally in response to a second subthreshold tone [this is a form of two-tone suppression (35)];
4. distortion components generated within the cochlea that have been measured by Goldstein and Kiang (12), Fahey and Allen (11), and others.

Since the transmission line theory is a linear theory, many researchers have studied ways of making the cochlear models nonlinear in order to study the numerous nonlinear effects (13,21). These models are still in the developmenal stage; therefore it is necessary to describe some of the data that they are trying to model rather than the models themselves.

The Basilar Membrane Nonlinearity

One of the most interesting of these nonlinear effects was first observed by Rhode (31,32) when he measured the input-output characteristics of the basilar membrane as shown in Fig. 6. He found the basilar membrane displacement to be related to the stapes displacement in a highly nonlinear fashion. For every 4 dB of level increase on the input, the output only changed 1 dB. This compressive nonlinearity was dependent on frequency and only occurred near the most sensitivity frequency for the point on the basilar membrane that he was measuring (e.g., the tip of the tuning curve). For other frequencies the system was linear, that is, 1 dB of input change gave 1 dB of output change for frequencies away from the best frequency. This nonlinear effect was highly dependent on the health of the animal and would disappear or not be present at all if the animal was not in its physiologically prime state. One interpretation of this nonlinear effect is that of a level- and frequency-dependent gain (amplification) that increases as the input level is reduced. From Fig. 6A it would appear that Rhode found approximately 35 dB excess gain at 7 kHz for 55 dB SPL relative to the gain at 105 dB SPL.

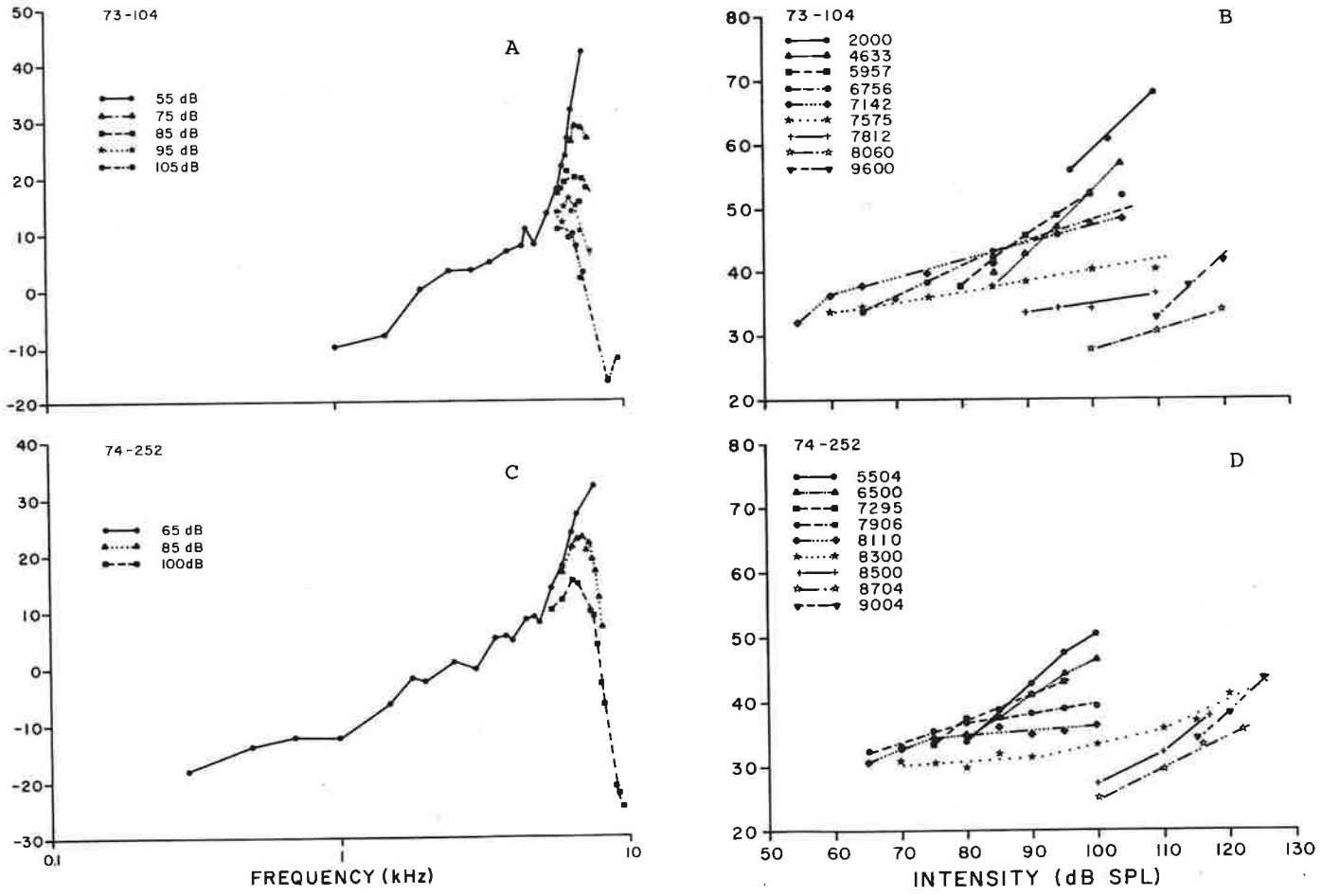


FIG. 6. The measurements shown in this figure are taken from Rhode (32) and show the basilar membrane displacement as a function of frequency normalized by the input level, for different input levels (**A,C**). In (**B,D**) we see the same data in a different format plotted as a function of input sound level with frequency as the parameter. Rhode was the first to find the nonlinear effect shown here (31,32), which may be interpreted as a frequency- and level-dependent gain, with increasing gain with lower levels. This extra gain is maximum near the best frequency of the basilar membrane measurement point.

The Receptor Potential Nonlinearity

In 1977, Russell and Sellick (34) found a similar result in the receptor potential of the inner hair cell of their guinea pig preparation—a frequency-dependent, compressive, nonlinear effect (Fig. 7). These two independently measured findings, at different points in the system, give credibility to the hypothesis that the basilar membrane response is inherently nonlinear and that at low-sound-pressure levels, the basilar membrane displacement is being amplified in a frequency-selective manner, producing the narrow-band tips on the tuning curves of high frequency neurons at low levels (8). If this is the case, then there should be a correlate of this phenomenon in the neural signal. In Fig. 8 we see the effect of adding a low-frequency bias tone, below the neurons threshold, on the frequency response of a neuron (4,11,20). Such a family of neural tuning curves are qualitatively similar to the responses found by Rhode (32) and Russell and Sellick (34).

A Nonlinear Paradox

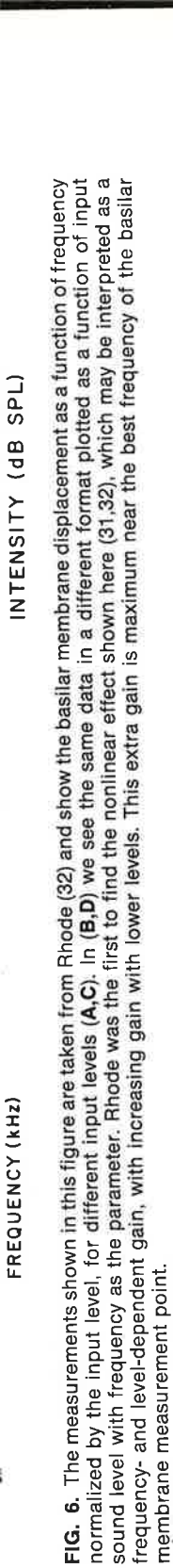
It is interesting to note here the paradox between the volume conservation law and the nonlinearity found by Rhode (32). The first law says that the volume displacement of the basilar membrane must be equal to the stapes volume displacement at each instant of time. Rhode observed that the basilar membrane displacement is not proportional to the input displacement but appears to have excess gain near the best frequency. This implies that the traveling wave must redistribute along the basilar membrane length, as a function of the input level, in a highly constrained manner. This in turn would require that the neural phase must change with level, which in general is not found below 4 kHz (2). One way out of this paradox is to add an extra degree of freedom between basilar membrane motion and hair cell excitation. A second approach is to note that the experimental evidence for the nonlinear excess gain is all above 4 kHz, and therefore perhaps the excess gain is not present below 4 kHz, where the neural phase data have been measured.

TWO-DIMENSIONAL COCHLEAR MACROMECHANICS

In this section we return to the linear models and try to give a bit of the flavor of the extended hydrodynamic theories of cochlear mechanics, so that the reader may better appreciate how and why they represent an improvement on the transmission line theory.

The first step toward a more manageable theory was taken by Ranke (30) in 1950 in what he called a "short-wave" theory. Short-wave theory is most accurate near the cutoff frequency, whereas long-wave theory (the one-dimensional model is a long-wave theory) is best basal to the cutoff frequency (39,41). Ranke's attempts were historically significant (39,41) but never actually developed into a useful theory for several reasons. For example, it is not known how to optimally interface the long-wave model to the short-wave model, since some sort of matching procedure is required.

Then in 1972, Lesser and Berkley (23) proposed a rectangular box model of the cochlea in which the scalae were straight and the cochlea was assumed to be sym-



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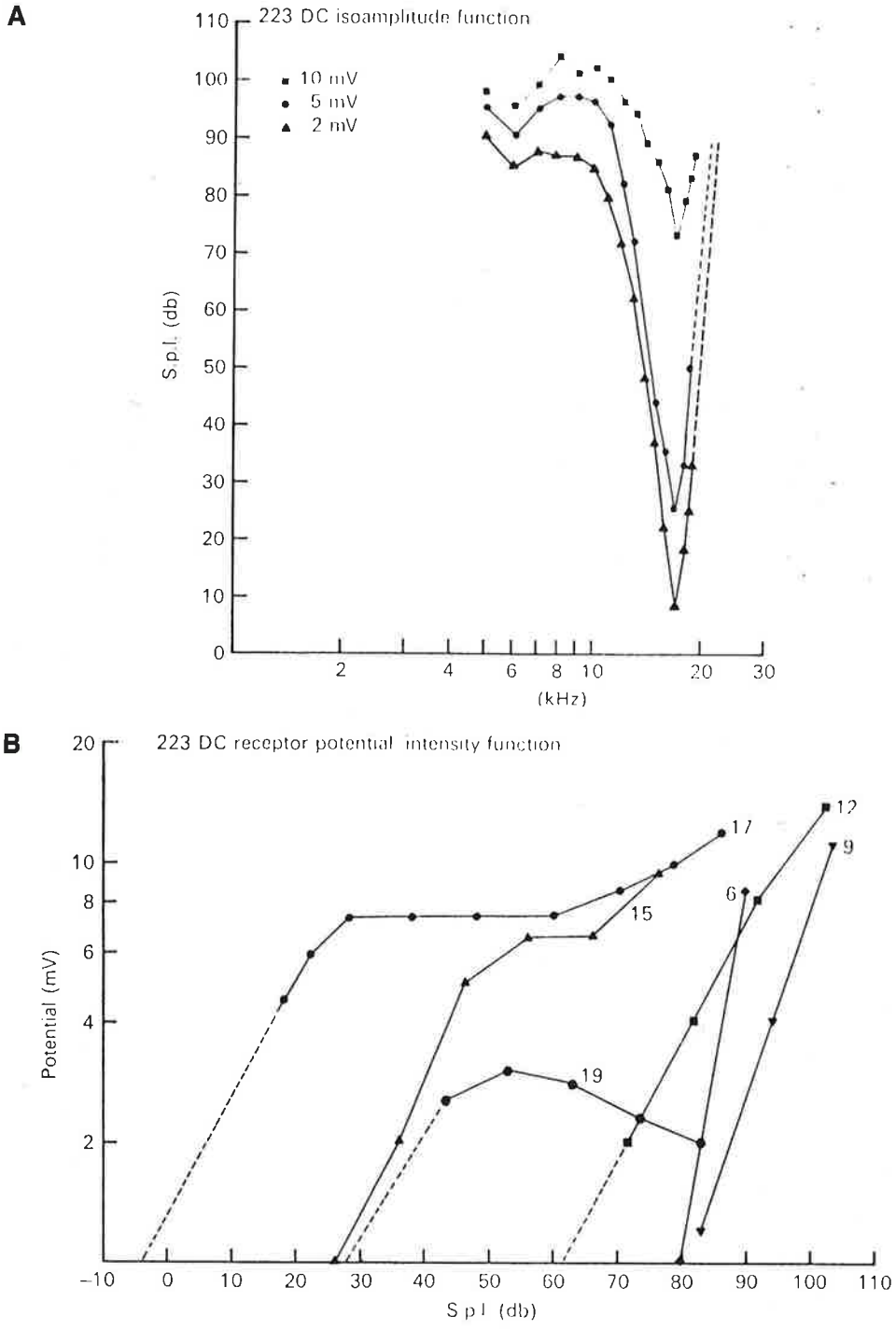


FIG. 7. Russell and Sellick (34) found a nonlinear and frequency-dependent effect in the receptor potential similar to that found by Rhode for the basilar membrane (32). In (**A**) we see the sound-pressure level, in dB, required to obtain 2, 5, and 10 mV of receptor potential in an inner hair cell. The format of this figure differs from that of Rhode in that the response is not normalized by the input sound level. In (**B**) we see curves similar to Rhode's that show nonlinear compression of the response. Not shown are the rectifying effects of the cilia, which produce a large DC component in the inner hair cell response.

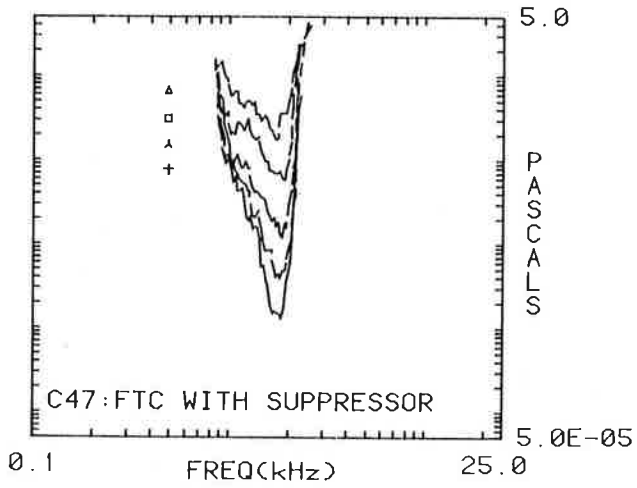


FIG. 8. When the response of a neuron is measured in the presence of a second (suppressor) tone we see that it may have a strong attenuation effect, even though the suppressing tone produces no response when present alone. The symbols define the frequency and level of the suppressor tone that was presented while the tuning curve was measured. When the suppressor-tone level was increased, the threshold of the tuning curve increased in a frequency-dependent way. The lowest threshold tuning curve was measured with the suppressor tone turned off. One interpretation of this effect is that at low levels of input sound pressure, the extra basilar membrane gain (as described, for example, in Fig. 6) is high. When the suppressor tone is present, it suppresses the extra gain, just as a high-level signal does in Fig. 6. According to this model, the suppressor signal does not give rise to an output when presented alone because of the high pass filtering that results from the tectorial membrane resonance model as described in Fig. 18C.

metric about the basilar membrane. This geometry is shown in Fig. 9. A main point of their paper was to demonstrate the importance of extending the models to two dimensions because of the effect of this extension on the solutions, a point that had been made years earlier by Ranke (30). Their line of reasoning inspired research that kept people busy computing for at least 10 years. As mentioned, via numerical methods, we have now moved beyond the two-dimensional formulation into the realm of three-dimensional models. More time is needed to evaluate fully the significance of these more detailed calculations and models, but it presently appears that they alone do not close the gap, as was originally hoped, between model and experiment. Thus the most important problem that still remains unsolved in cochlear theory is explaining the sharpness of tuning of the neurally measured response. Although the two-dimensional models brought the neural data and model calculations into agreement on the high-frequency side of the tuning curve, they did not improve the match on the low-frequency side. The most recent experimental measurements either indicate or are consistent with a 20-dB difference between basilar membrane responses and neural responses on the low-frequency side of the tuning curve. Such a transformation will be discussed next.

Cochlear Micromechanics

Micromechanics refers to the mechanics of the organ of Corti. The most commonly accepted description of the motion of the organ of Corti was proposed by ter Kuile

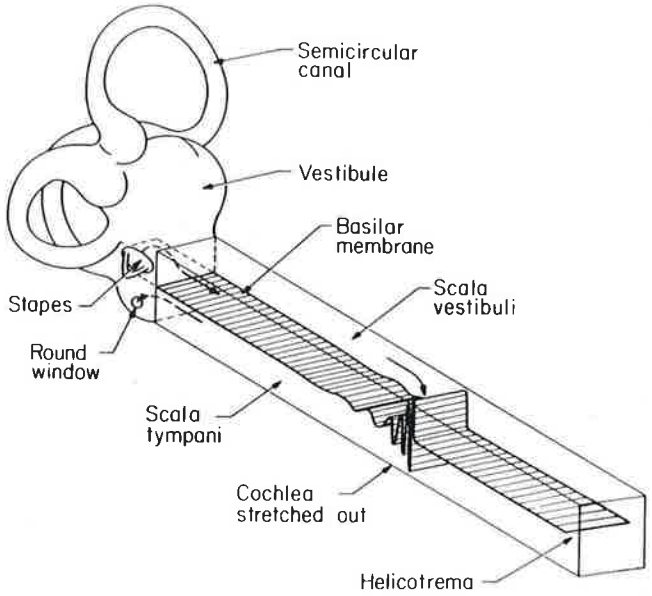


FIG. 9. This figure shows the traveling wave at one point in time on the basilar membrane. Because of the dispersive nature of the basilar membrane, a wake appears behind the main pulse. This pulse also becomes broader as it travels down the basilar membrane owing to the attenuation of the higher frequency components. (From ref. 43.)

in 1900 (22). His concept is shown in Fig. 10 where we see how he proposed that the displacement of the basilar membrane could drive the hair cells in a radial mode of excitation. In Fig. 11 we see a similar description of this mode of excitation from Allen (1). A simple analysis of the model reveals that the vertical motion (the y direction) of the basilar membrane is linearly related to the radial shearing motion (the z direction) seen by the cilia of the inner hair cells, which are known to be the transducers that sense the motions of the basilar membrane. Thus the model of ter Kuile is equivalent to a lever that linearly converts the vertical basilar membrane motion into radial shearing motions appropriate for the excitation of the inner hair cells. (The word motion is used to avoid the important question of whether velocity or displacement is the actual inner hair cell excitatory stimulus.)

The ter Kuile model seemed adequate as a first step, but several important problems remained. First, there have been no direct observations to confirm the ter Kuile model nor are there likely to be any in the near future, because of the inherent difficulty in making observations of such small motions in such difficult places. Second, we cannot yet be sure, given the present experimental data, if the neural and basilar membrane responses are in agreement with each other, as described in the previous section. It was hoped that a simple modification of the ter Kuile model might bring together the various theories and the experimental data. We will argue this possibility here.

Basilar Membrane versus Neural and Hair-Cell Tuning

At this point it is again necessary to remove ourselves from the models and look at some experimental data in order to understand the nature and magnitude of the

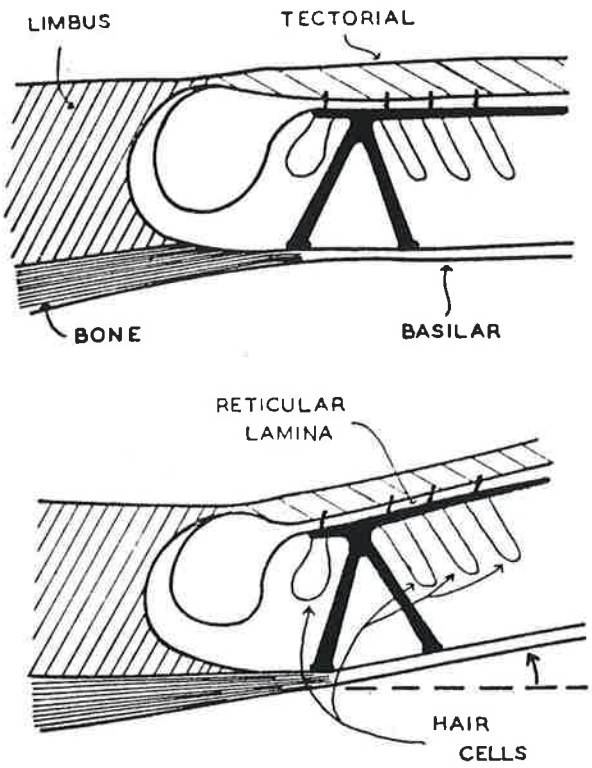


FIG. 10. In 1900, ter Kuile (22) first described his model of how the vertical displacement of the basilar membrane is transformed into a radial shearing required to drive the inner hair cell cilia. At that time it was generally assumed that the tall cilia of each inner hair cell was connected to the tectorial membrane. It is now generally believed that inner hair cells are not driven directly by the tectorial membrane, but are dragged by the surrounding fluid that is in phase with the displacement. This would happen because the viscous boundary layer (a thin fluid layer where viscous forces dominate) is greater than the 6-μm distance between the tectorial membrane and the top surface of the hair cells (this surface is called the reticular lamina). As a result, the relative shear of these two surfaces acts as a mechanical resistor, or dashpot, as it is referred to in mechanical terms. The mechanical equivalent of the entire system is a lever, or electrically, it is a transformer. (From ref. 1.)

Davis 1958, Laryngoscope 68 359-382; Pickles p.28

discrepancy between the mechanical tuning of the basilar membrane and neural signal. In Fig. 12 we see tuning responses from Sellick et al. (37) of the basilar membrane as compared with a neural measure, where the responses differ by approximately 10 to 20 dB on the low-frequency side of the response.

The voltage in the inner hair cell was first measured by Russell and Sellick (34). This voltage, called the receptor potential, is tuned like the neuron. In a later paper, Sellick et al. (38) show much more detail, as seen in Fig. 13. These results consistently show a difference on the low-frequency side of the characteristic frequency. In the summary of their 1983 paper (38) they stated: "In conclusion, a demonstration of inner hair cell tuning at the level of the basilar membrane continues to elude us."

Robles et al. (33) also have compared basilar membrane tuning with a neural measure. Their summary result is shown in Fig. 14. Again on the low-frequency side of the tuning curve, they find a difference, but in their case, the difference is in the form of a large variance, which they indicate by error bars (see the displacement response at 2.8 kHz). It is interesting to note that in the frequency region near 1.75 kHz the neural signal is less sharply tuned than either of the mechanical measures, an observation unique to all such experiments.[1]

Is the difference between basilar membrane, neural, or hair cell measures signif-

[1] The measurements of basilar membrane motion by Khanna and Leonard (18) are not direct comparisons with neural or receptor measures. As a result of the normalization procedure used by them and the ear canal standing wave they report (19), their data are not a direct test of this question. Furthermore, they have not observed the nonlinear compression as seen by Rhode (31,32), Russell and Sellick (34), Sellick et al. (37,38), and Robles et al. (33).

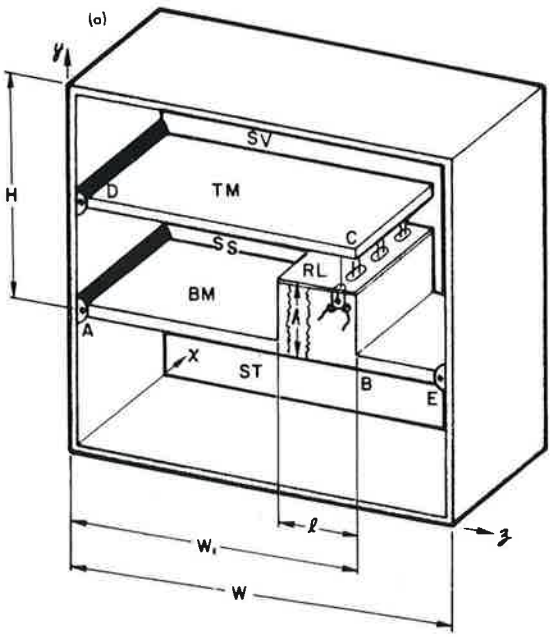
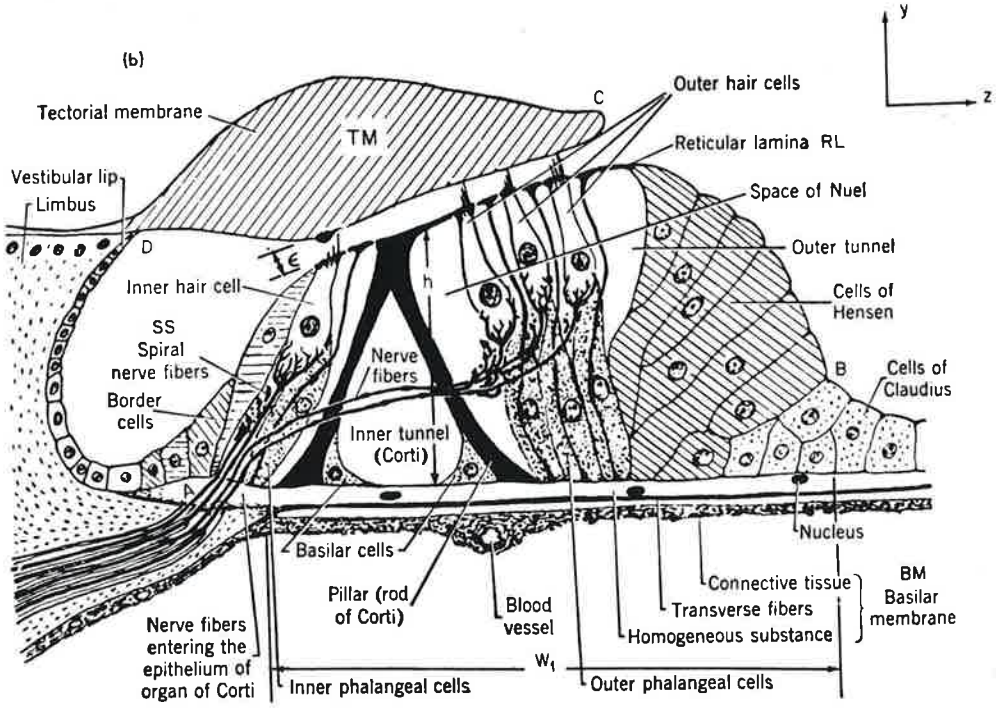


FIG. 11. We show here a labeled three-dimensional representation of the previous figure (**a**) and compare it with a detailed labeled drawing of the organ of Corti (**b**). The inner hair cells seen in (b) are the transducers that signal the central nervous system (CNS). The purpose of the outer hair cells is still unknown other than the obvious structural one. The cilia length of the outer hair cells define ϵ, the subtectorial space. The neurons connected to the outer hair cells are for the most part efferent neurons. It has been shown that the CNS can modify, to some extent, the mechanical properties (e.g., the stiffness) of the outer hair cell cilia. The inner hair cells on the other hand appear to be passive displacement detectors that input to the afferent primary neurons. (From ref. 1.)



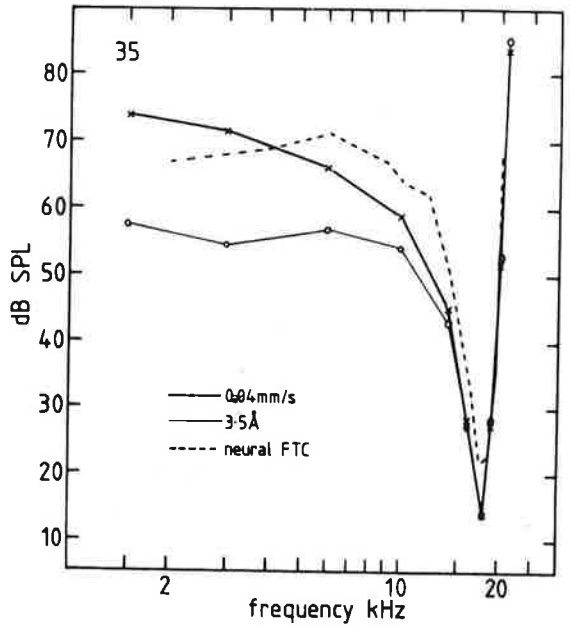


FIG. 12. Basilar membrane isovelocity (x) and isodisplacement curves (○) compared with a neural tuning curve (*broken line*) derived from the guinea pig spiral ganglion with a comparable characteristic frequency to that of the basilar membrane measurement point. The spiral ganglion data were courtesy of D. Robertson. (From ref. 37.)

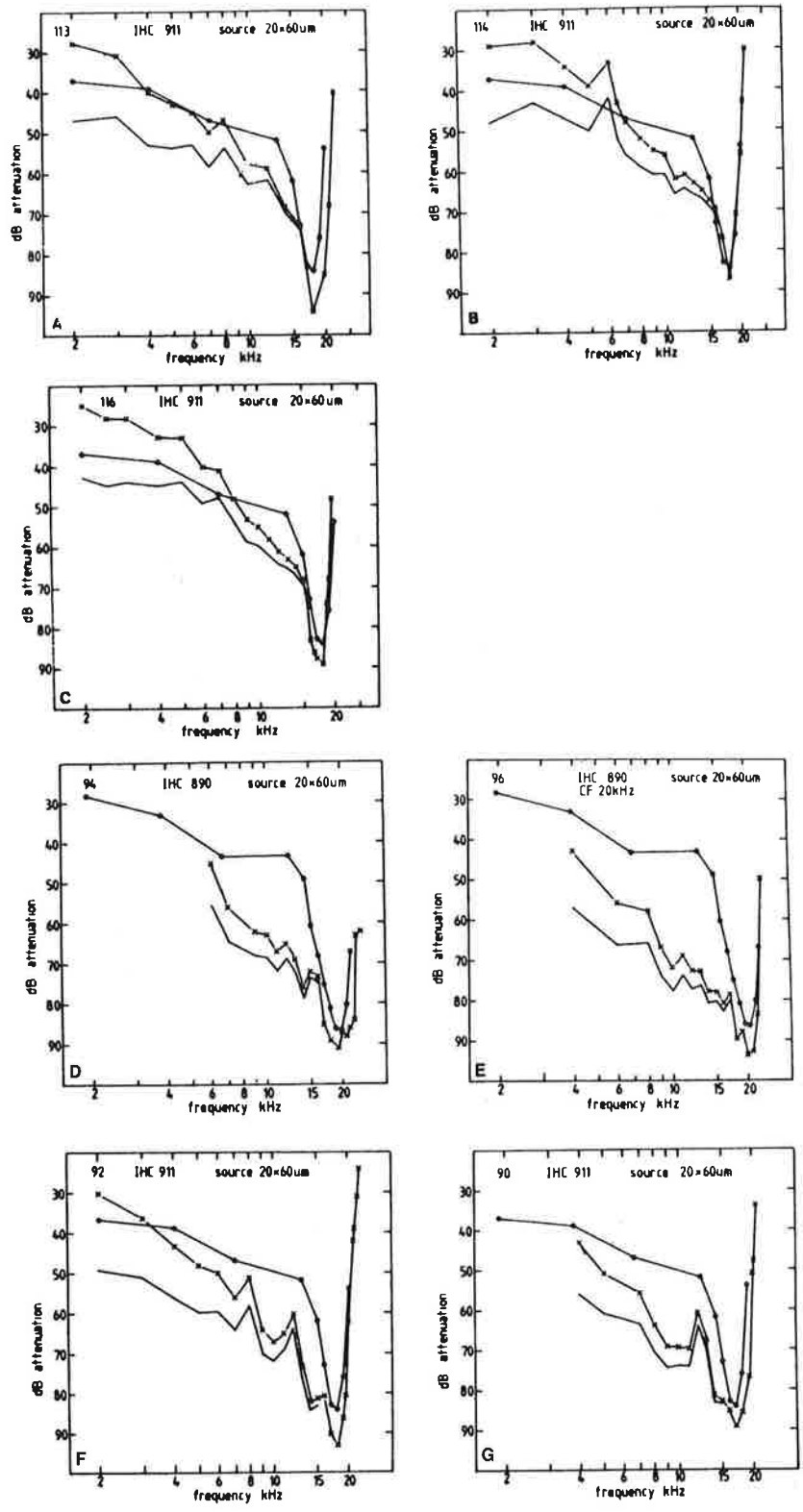
icant, or is it an artifact of the experimental technique? Unfortunately, we cannot yet be sure of the answer to this important question. At present the Neely and Kim (26) model accounts for neural tuning data, or receptor potential data, for neurons tuned above 5 kHz. This model assumes that basilar membrane tuning is equal to neural tuning. The Allen model (1,3) describes neural data for frequencies below 5 kHz, but in that model, neural and mechanical tuning differs by approximately 20 dB one-half octave or so below the best frequency. Thus two micromechanical models that make quite different assumptions have been shown to fit tuning data in different frequency regions. Until the experimental questions are resolved, it seems that this theoretical question must remain open.

MICROMECHANICAL MODELS

We next discuss two classes of theories that attempt to model the experimentally observed frequency selectivity. The first is based on the idea that the tectorial membrane vibrates at its own resonant frequency, near the resonant frequency of the basilar membrane. In 1980, different versions of this resonant tectorial membrane approach were independently proposed by Zwislocki and Kletsky (48) and Allen (1). The second is the theory of Neely and Kim (26), which calls on the idea of an active, or negative resistance, basilar membrane. Most recently, Neely and Kim (27) have published a more comprehensive theory in which they merge the resonant tectorial membrane model of Allen (1) with their active basilar membrane theory.

Zwislocki's Tectorial Membrane Models

Zwislocki (46) has proposed a number of tectorial membrane models for sharpening the basilar membrane response. In 1979 he and Kletsky (47) proposed a model for


113 IHC 911 source 20×60um
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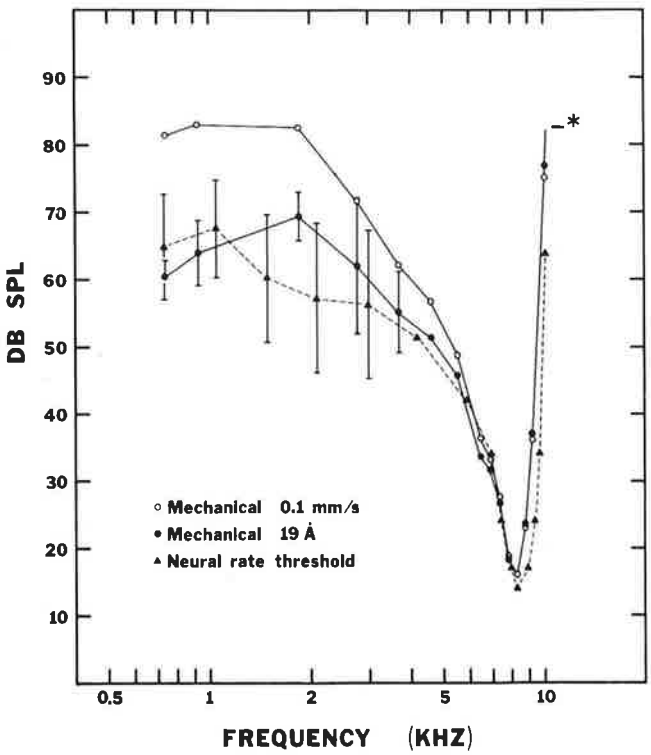

FIG. 14. Comparison of mechanical and neural response measurements. Both the displacement (●) and the velocity (○) are shown for comparison. The neural measurements (*broken line*) and the basilar membrane displacement are shown with error bars that represent one standard deviation of the measurement. What is unusual about this comparison is that the neural response is less sharply tuned than either of the mechanical responses. (From ref. 33.)

sharpening based on longitudinal smoothing by the tectorial membrane, which resulted from the longitudinal mechanical properties of the tectorial membrane, followed by a difference that resulted from the usual ter Kuile shearing motion at the inner hair cells. With some simple analysis, one may show that this model is similar in its effect to the spatial difference model proposed by Hall in 1977 (13). This model is not, however, a resonant tctorial membrane model.

In 1980, Zwislocki and Kletsky (48) proposed two new approaches to this problem, which they called models I and II. Model I is a resonant reed that is mass loaded. The resonant system is meant to represent the tectorial membrane mass and the stereocilia stiffness.

FIG. 13. This figure summarizes one of the major problems in hearing research today, namely the observed difference between the measured basilar membrane frequency response and hair cell measured frequency response. The problem is that most measurements of basilar membrane frequency response are not as sharply tuned as the hair cell responses. **A,B,C:** Measurements of the motion of the small Mossbauer source placed on the edge of the basilar membrane compared with the isoamplitude curve at 0.9 mV inner hair cell d.c. receptor potential (○). Basilar membrane isovelocity curve at 0.04 mm/sec (X). Basilar membrane isodisplacement curve at 3.5 Å (continuous curve). **D,E,F,G:** As for A, B, C but with the small source placed in the middle of the basilar membrane. (From ref. 38.)

This model of excitation distinctly differs from the model of ter Kuile, since there is no analog of the spiral limbus-tectorial membrane coupling. Their model differs from that of Allen (1) in exactly this way, since in Allen's model, the radial stiffness of the tectorial membrane was specifically taken into account in a functionally significant way. In Allen's model, the coupling element plays an important role in the excitation of the cilia. As a result, the shape of the frequency response owing to the radial resonance in Allen's model is quite different from that measured by Zwislocki and Kletsky (compare Fig. 18C with Fig. 2 of ref. 48). In summary, these two systems are *not* isomorphic.

Zwislocki and Kletsky's model II (48) seems to be a partial joining of the spatial smoothing model (47) and model I described above. This model consists of a parallel bank of resonating reeds tuned to slightly different frequencies. The reeds are connected, along the longitudinal axis, with a nonlinear elastic medium that mechanically couples them. Again, as in model I, this system has no spiral limbus analog. The model system is shown in a photograph in the original paper, along with some experimental results showing the suppression effects they saw of one tone on a second. Both models I and II are described as nonlinear models, but only the first is a sharpening model.

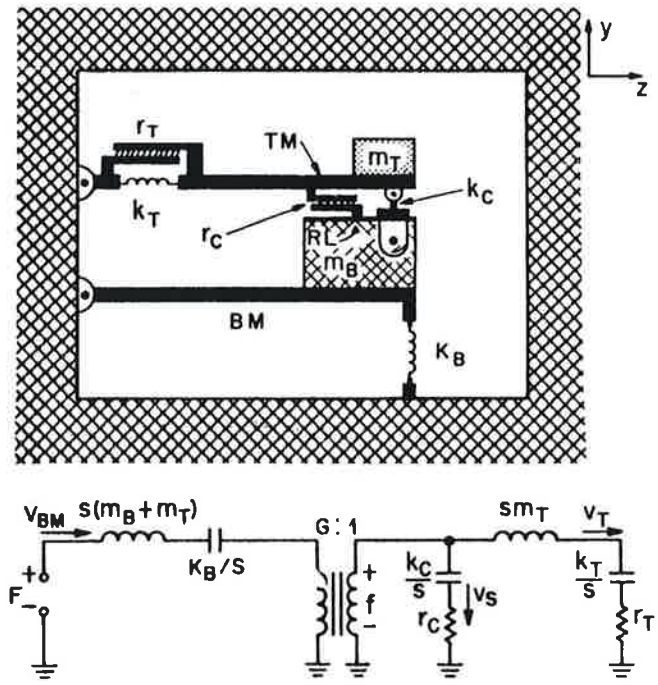

FIG. 15. A model assumption that allows one to match neural tuning data is to introduce a spring, or elastic element, in the tectorial membrane (element k_T of the figure). The addition of this element gives rise to a response cancellation owing to resonance in the response function describing the relation between the basilar membrane and the shear seen by the hair cells. This may be shown by analyzing the electrical equivalent circuit given in the lower part of the figure. (From ref. 1.)

Allen's Tectorial Membrane Model

In Fig. 15 we see a model extension of the ter Kuile model where the tectorial membrane is given a new degree of freedom to vibrate in the radial direction (1), depicted here as the z direction. On the low-frequency side of the tuning curve for this model a partial cancellation of the shear motion occurs at the site of the inner hair cells, relative to the up-down motion of the basilar membrane. This cancellation is a result of the added degree of freedom (the elastic tectorial membrane element labeled k_T). This cancellation could account for the difference frequently observed between basilar membrane motion and neural response below the characteristic frequency. In Fig. 16 we show cat neural tuning data for several neural units, and in Fig. 17, the model result using the linear two-dimensional macromechanical model coupled to the resonant tectorial membrane micromechanical model (3). In the model calculation we have held the model output constant and plotted the resulting input

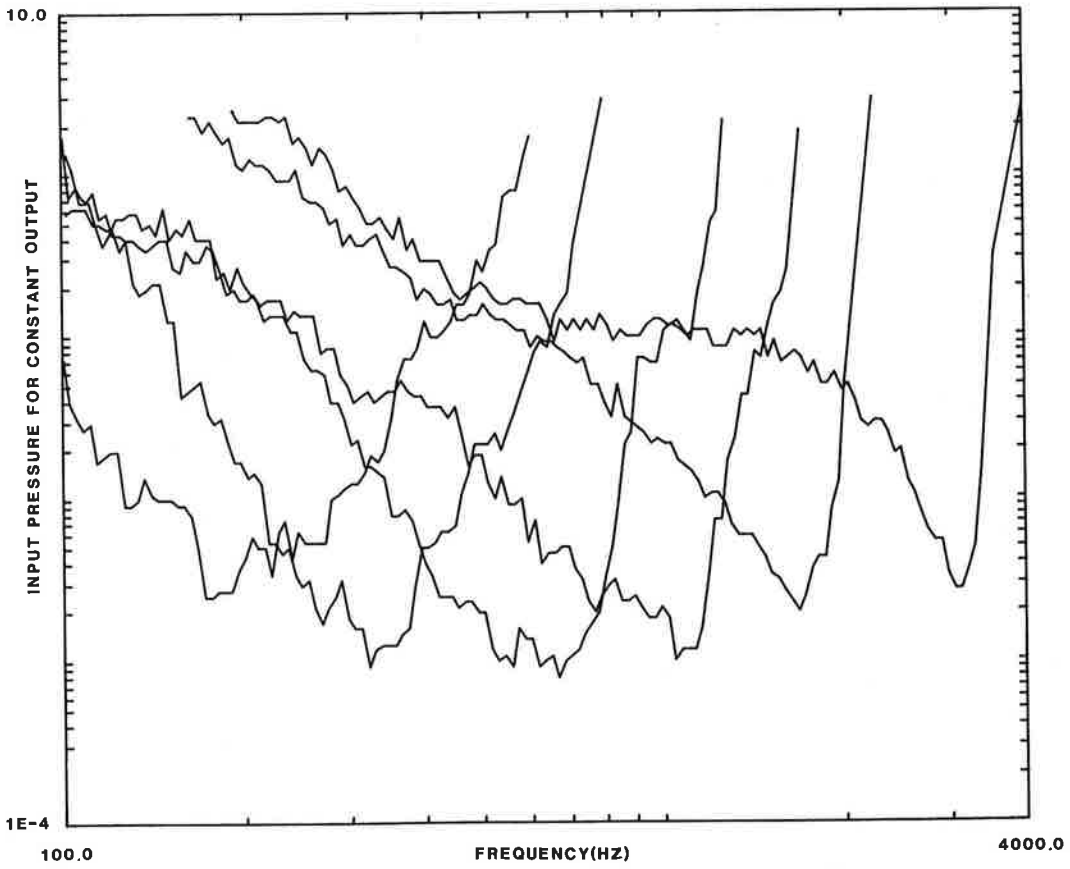

FIG. 16. We show here six low-threshold tuning curves from the cat that are equally spaced on the log-frequency axis. Only units having characteristic frequencies between 100 Hz and 4 kHz are displayed because this is the important frequency range for speech communication. No similar data are available for humans. However, all known mammals give similar results. Note the amplitude range of the plot that covers a 10^5 range or 100 dB.

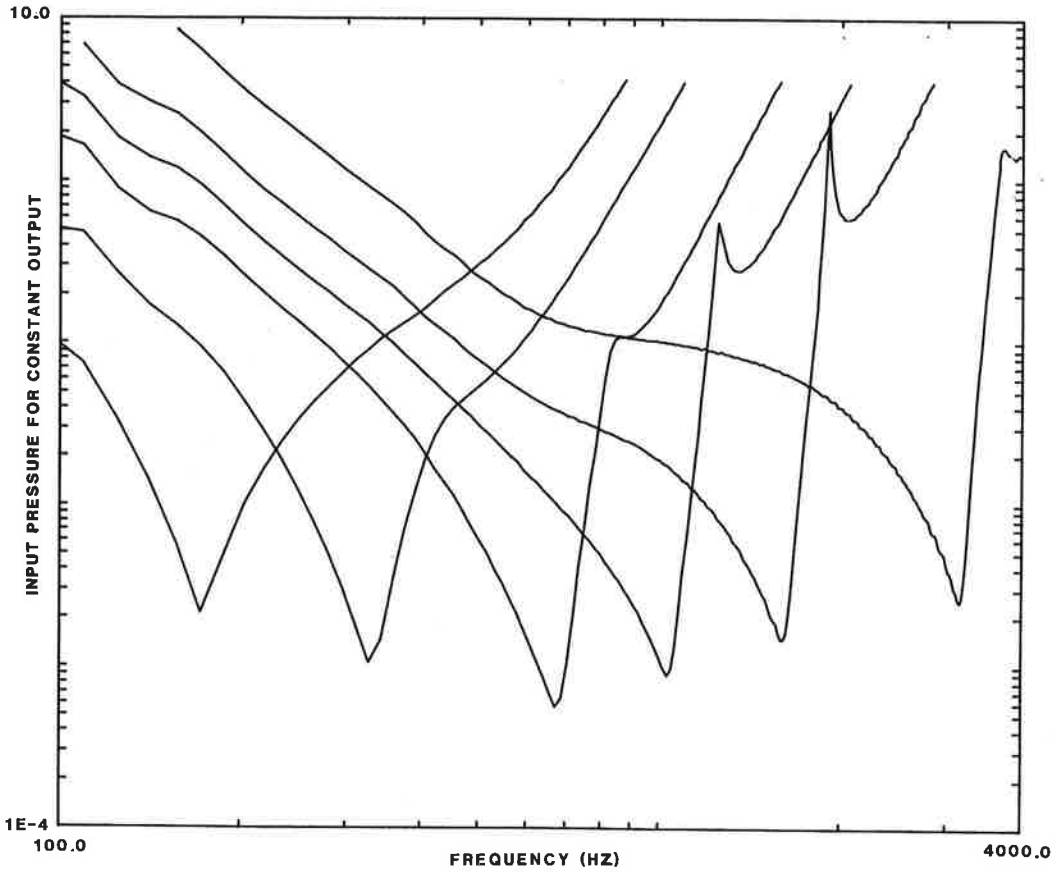

FIG. 17. The cochlear and middle ear models are used to simulate the ear canal pressure for a constant output, which here was assumed to be the shear velocity of the tectorial membrane-reticular lamina. The model calculation was done in the frequency domain with a linear two-dimensional cochlear model. The basilar membrane micromechanical model is that defined in Fig. 15.

pressure in the ear canal. Intermediate model results (not shown) for the cochlear input impedance and the cochlear microphonic also agree with experimentally observed results.

In Fig. 18 we show four measures from the model as a function of position along the basilar membrane, for six different input frequencies: In (A) we see the model neural output, given constant input pressure in the model ear canal; in (B) we show the model neural phase; in (C) we show the model transfer function magnitude relating the basilar membrane to hair cell displacement, which results from the resonant tectorial membrane model; and in (D) we see the basilar membrane impedance magnitude for the resonant tectorial membrane model, which is required when calculating the basilar membrane velocity using the macromechanical model. For these results we assumed that the model neural output was proportional to the TM-RL shear velocity as described in the legend for Fig. 17.

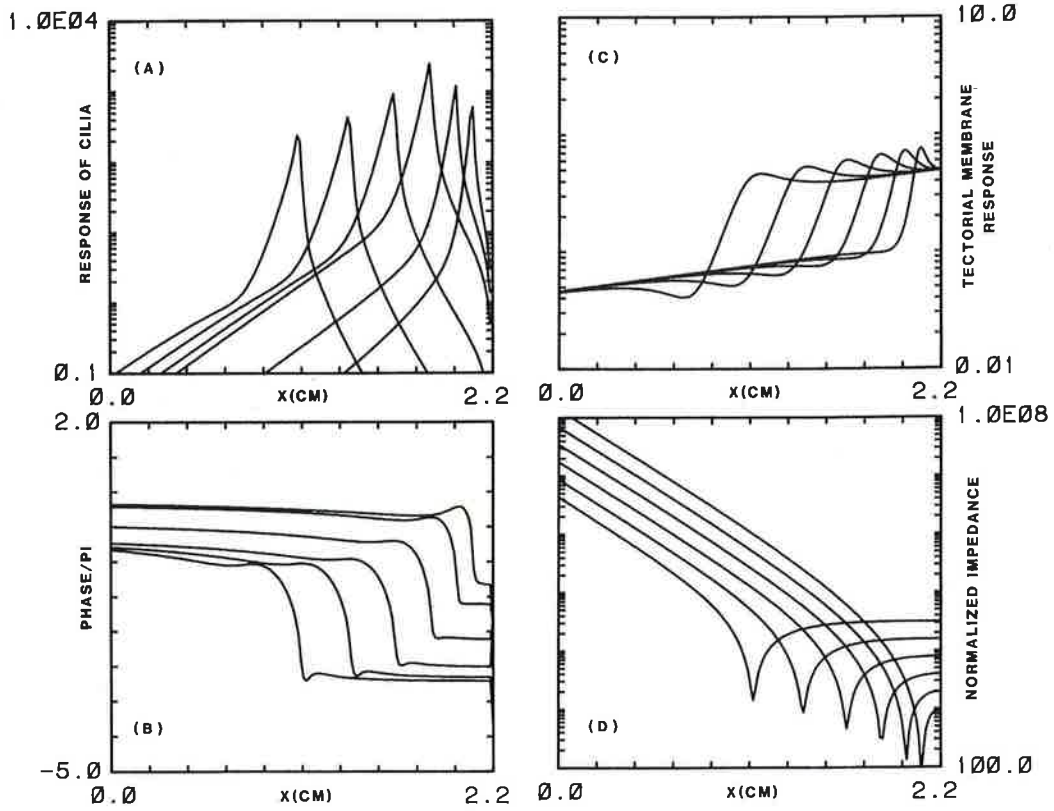

FIG. 18. This figure illustrates how the response varies as a function of position along the basilar membrane, for six different input frequencies. Panel **A** gives the shear velocity; **B** shows the shear phase, **C** shows the basilar membrane to shear transfer function magnitude (this is defined as the ratio of the cilia response to the basilar membrane response), and **D**, the model basilar membrane impedance magnitude. Note particularly the effect of the resonant tectorial membrane on the tuning curve, as shown in (C). The effect of the tectorial membrane in this model is to change the quasi-low-pass basilar membrane transfer function (Fig. 5) into a bandpass filter as seen in Fig. 17.

From Figs. 16 and 17 it is clear that the model does a reasonable, but not perfect, job of describing the neural data. Note that the resonant tectorial membrane transfer function (Fig. 18C) has a 20-dB "sharpening" effect on the response for frequencies below the cutoff frequency, which is close to the difference observed by Sellick et al. (38) as seen in Fig. 13. Our model effort does not at present attempt to account specifically for the Sellick et al. data.

Neely and Kim's Negative Resistance Model

A second and alternative approach to account for sharp neural tuning has been proposed by Neely and Kim (26) and has been worked out in some detail by Neely in his Ph.D. thesis (25). This model calls on the concept of negative damping, or resistance, in the basilar membrane. This model distinguishes itself from the resonant

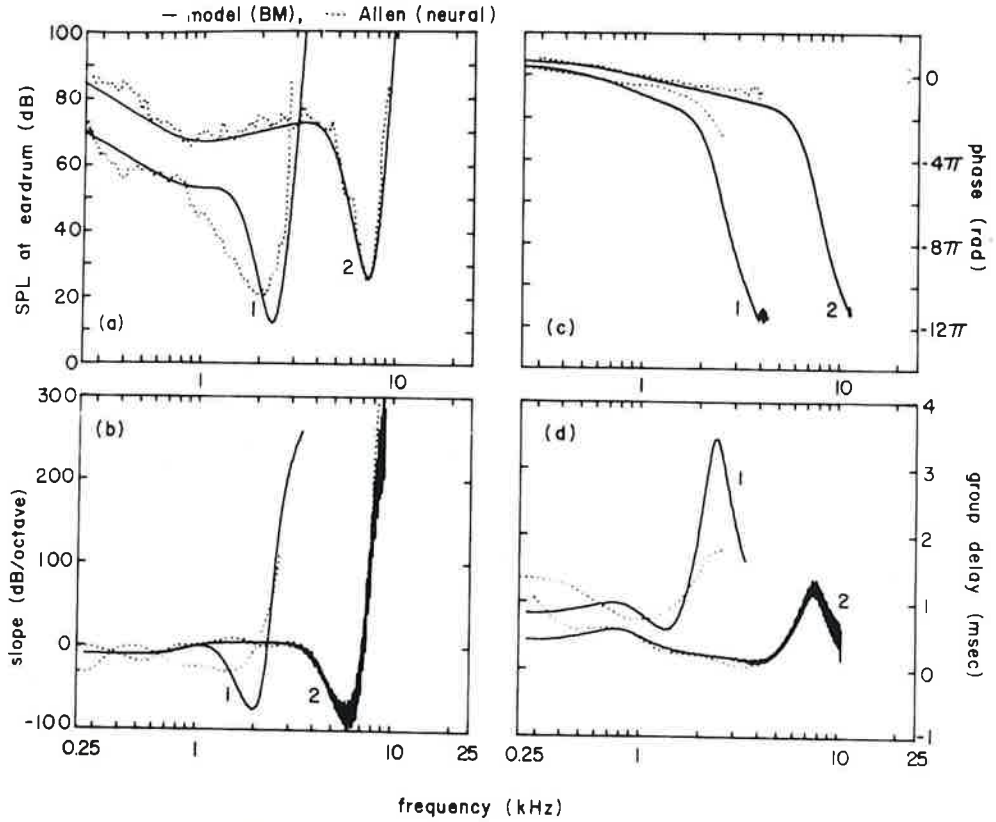

FIG. 19. Comparison of neural data (*dotted lines*) and model results (*solid lines*) from Neely and Kim's active basilar membrane model. (**a**): Threshold tuning curves; (**b**): slope of the tuning curves; (**c**): phase of the response; and (**d**): group delay of the response. The neural phase data in (c) and (d) are plotted after removing 1.2-msec delay attributable to acoustic, synaptic, and neural spike propagation delays. (From ref. 26.)

tectorial membrane models owing to one of its basic assumptions, i.e., the model assumes that the neural and the basilar membrane responses are identical. Therefore the original ter Kuile model was used unmodified in Neely's theory (the tectorial membrane was assumed to be rigid in the radial direction (26) (Fig. 2). One serious problem with this model is the lack of a definable relationship between the model parameters and the cochlear anatomy.

The results of Neely and Kim (26) shown in Fig. 19 are a very impressive match to high frequency neural tuning curves, both magnitude and phase, where phase data are available. In general, the higher the characteristic frequency of the neural data being matched, the better the model fit.

In a recent paper, Neely and Kim (27) join the resonant tectorial membrane model of Allen (1) with an active source that represents active outer hair cells. This model gives the best fit to tuning curve data to date for any of the models, if the entire 100 Hz to 30 kHz hearing range is considered. This paper also improves on the earlier paper (26) by having a definable correspondence between many of the model parameters and the cochlea anatomy, with the important exception of the active source pressure.

The use of a negative resistance is supported by the observations, first made by Kemp (15), of emissions from the cochlea.

Evoked Echoes and Spontaneous Emissions

In 1958 Elliott (10) observed that the threshold of hearing was not a smooth function of frequency, but that it fluctuated in a quasiperiodic manner with a period of a few hundred hertz. Such microstructure could be characteristic of low-level standing waves attributed to slight mismatches at different positions along the basilar membrane (17).

Later it was observed by Kemp in 1978 (15,16) that low-level dispersive reflections may be found in response to a pulse of sound in the ear canal. The delay involved approximately corresponds to a round trip travel time along the basilar membrane. The reflections are nonlinear in their behavior since they grow at less than a linear rate with increasing input pulse level. Because of the nonlinear character of the echoes, it will not be easy to model them until the nonlinear properties of the basilar membrane are better understood.

A third somewhat bizarre observation was then made with the finding that narrow-band tones emanate from the human cochlea (16,42). In animals, similar tones have been correlated with damage to the cochlea. It would be natural to ask if the microstructure in the hearing threshold previously observed correlates to these narrow-band tones, the speculation being that the tones are just biological noise passively amplified by the presumed standing waves mentioned above. Such narrow-band noise would have a Gaussian amplitude distribution, and the amplitude distribution of the tones seems to be closer to that of a pure tone, which is contrary to the standing-wave model.(6).

When these spontaneous emissions were first observed, many researchers were quick to conjecture that the cochlea was an active system that occasionally became unstable (8,16). Hence models that incorporate negative damping, such as Neely's (26), are interesting. The use of negative damping in the model serves the function of sharpening the tuning of the cochlear filters. It also has the capability, in theory, of making the basilar membrane oscillate, thus giving rise to the emissions that were observed by Kemp (15).

The source of the proposed negative damping is still unknown, but we believe we know where to look for it. In 1985 Brownell et al. (7) found that isolated outer hair cells change their length when placed in an electric field. This has led to the speculation that outer hair cells act as linear motors directly driving the basilar membrane. The displacement of the linear motors would probably be a function of the outer hair cell receptor potential, which in turn is modulated by both the position of the basilar membrane (forming a tight feedback loop) and the efferent neurons that are connected to the outer hair cells (forming a very slow feedback loop). The details of this possibility are the topic of present-day research. The work of Liberman and his colleagues gives important constraints on how this system might work. In Fig. 20 we see a figure from Liberman and Dodd (24) that indicates the complex relationships between the state of the inner and outer hair cells and the neurally measured frequency response. Perhaps an improved understanding of this interaction will lead to the breakthrough that we need in describing the cochlear frequency selectivity and the nonlinear characteristics of the basilar membrane.

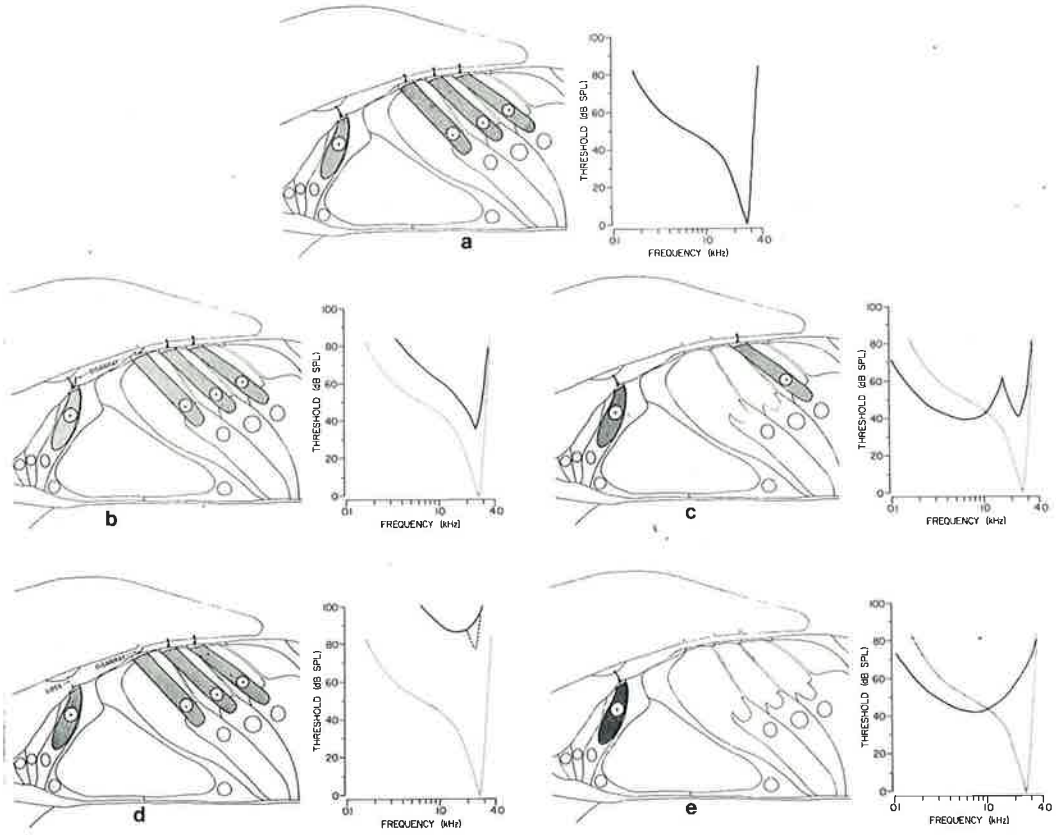

FIG. 20. Schematic representation of a normal organ of Corti (**a**) and four different damage states. Each damaged state is shown with the particular tuning curve abnormality that was found to arise from radial fiber innervation from such a region. From this figure we see that the tuning of the inner hair cell is systematically dependent on the nature of the damage. (**b**): Damage to the inner hair cells raises the threshold of the unit but does not significantly change the shape of the tuning. (**c**): Damage to the first and second rows of outer hair cells enhances the frequency-dependent notch seen just below 2.0 kHz. We interpret this notch as having the same physics as the model effect described in Fig. 18C, which results from the resonant tectorial membrane (Fig. 15). The tip is also missing, which might suggest a loss of basilar membrane gain because of the partial loss of outer hair cells. Note how this loss is accompanied by a lower threshold in the tail region (below 1.0 kHz). This could be interpreted as a loss of the cancellation in that frequency region owing to the shifting of the "zero" of the resonant tectorial membrane. (**d**): Inner hair cell loss uniformly increases the threshold. (**e**): Total outer hair cell loss results in a hypersensitive tail threshold. This is an extension of the result from (c). (From ref. 24.)

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