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A physicist's guide to the ear

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Integrative Exercise

Physics Department

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The ear is able to detect sound energies spanning 12 orders of magnitude at frequencies ranging from 20-20,000 Hz. This paper discusses the physical mechanisms underlying the stunning capabilities of the auditory system. We follow a sound wave from the external ear to transduction into an electro-chemical signal for processing by the brain. We discuss the middle ear in terms of impedance matching and derive an expression for sound transmission from one medium to another. We focus on the inner ear and pay particular attention to the function of the basilar membrane and the properties of the traveling wave both in terms of differential equations that describe the system as well as the impedance of the membrane itself. Finally, we examine the properties of the outer and inner hair cells with particular focus placed on the motile properties of the outer hair cells. We explain the mechano-electrical transduction of the inner hair cells.

Motion appears in many aspects—but there are two obvious kinds, one which appears in astronomy and another which is the echo of that. As the eyes are made for astronomy so are the ears made for the motion which produces harmony.

Plato

From the first observations of the cochlear partition it was clear that they represented a system about which physical science provided little knowledge and that many years would be required to understand clearly.

Georg von Békésy

You can observe a lot by watching.

Yogi Berra

Introduction

Organisms that can detect sound posses a huge advantage over those that cannot. The detection of sound, however, is very complicated, requiring an exquisitely designed detector. Evolution commands amazing power and when life *literally* depends on the quality of an organism's sound detector, tremendous amounts of time and energy can be devoted to the problem of making one. Thus, Nature has developed exquisitely sensitive biological detectors, whose incredible precision is matched by daunting complexity. Indeed, the auditory system boasts a tantalizing collection of problems for the physicist: the ear must convert a sound wave into an electro-chemical signal. It must first transmit that sound from an air medium to a fluid medium and then filter it and amplify it to prepare it for mechano-electric transduction. Finally, it must transduce the mechanical energy into the electro-chemical language of the nervous system. This whole process must occur more or less instantaneously and in fact, the ear is one of the fastest responding sensory mechanisms. Furthermore, the sound must be faithfully recorded, but also compressed to fit the information constraints applied by the nervous system: there are only so many neurons and they can only fire so fast. Finally, detection must occur over frequencies from 20Hz to 20kHz and over intensities spanning 12 orders of magnitude!

In this paper we will follow a sound wave from the environment's air medium to its electro-mechanical transduction into a nerve signal, focusing specifically on the transmission of sound across the air-fluid interface and the subsequent filtering and amplification of that sound that occurs in the inner ear. Finally, we will briefly discuss mechano-electric transduction.

Physiology and function of the outer and middle ear

Getting sound into the body

In this paper we will think of sound as periodic fluctuations in the pressure of an elastic medium, or more simply, the periodic movement of the molecules that make up an elastic medium. We therefore must first determine how the ear transfers periodic movement of air molecules in the environment to periodic movements of the fluid inside the body, the job of the *outer* and *middle* ears (Fig. 1).

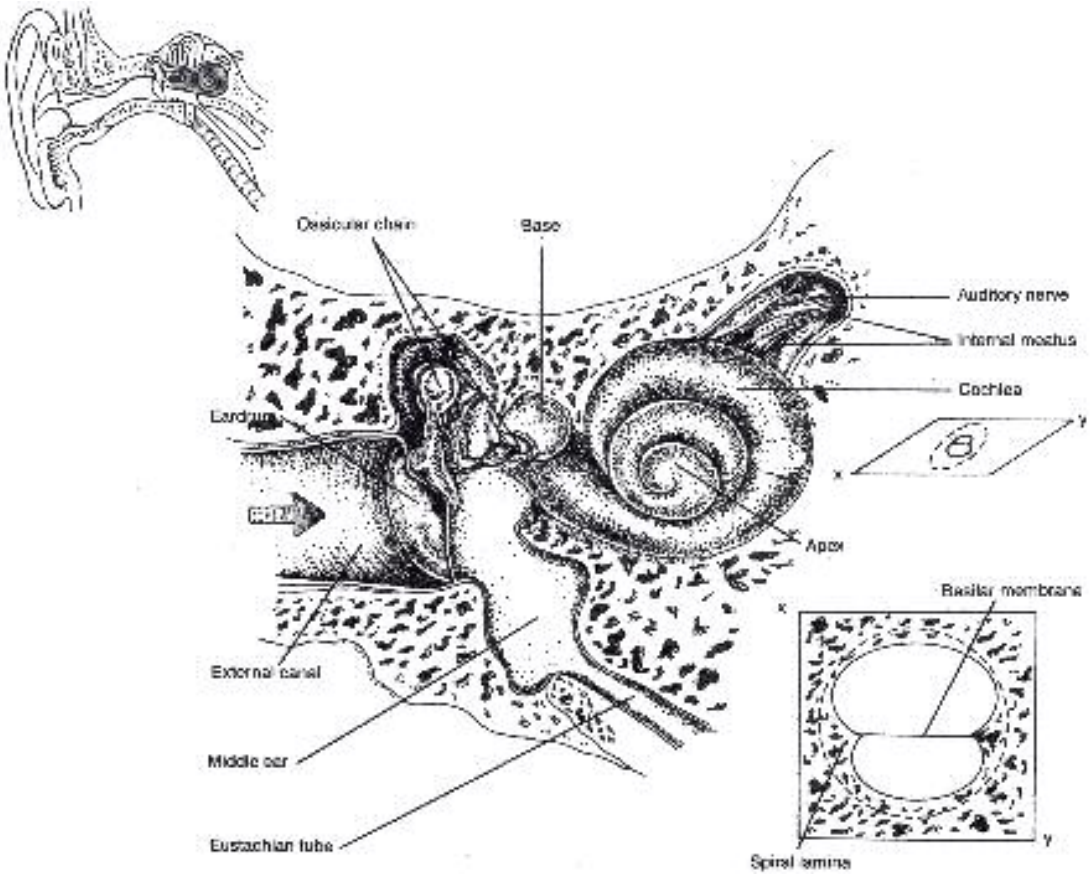

Figure 1: A diagram of the hearing system consisting of the outer, middle, and inner ears. We will focus on the eardrum: the bridge between the outer and middle ears; the middle ear bones (here labeled *Ossicular chain*): impedance matchers that transmit sound from the outer to inner ears; and the cochlea: a fluid filled tube (see inset) responsible for transduction of the sound signal into an electro-chemical signal.[1]

The outer and middle ears are mechanical vibrators and amplifiers; the former gathers sound and the latter transmits and transforms it. When a sound wave enters the

ear, it propagates to the eardrum, imparting kinetic energy to it in the form of mechanical vibrations. Connected to the eardrum is a chain of three bones that bridge the space from the outer ear to the inner ear. It serves as the solution to the problem of getting sound waves that were once in air into a fluid-filled body.

These bones serve to transmit sound from the air medium of the environment to the fluid medium of the inner ear. As we will show in the following section this task is not trivial and represents the ear's first major challenge.

Why is it so hard to transmit sound from an air to a fluid medium?

We want to find out how the properties of two adjacent media affect the power transferred from one medium to the next. Let's do this by thinking about the pressure waves in the two media. Assume there is some incident pressure wave p_i, a reflected pressure wavep_r, and a transmitted pressure wave p_t defined as follows:

$$\begin{aligned} p_i &= P_i e^{-ik_1 x} \\ p_r &= P_r e^{ik_1 x} \\ p_t &= P_t e^{-ikx} \end{aligned} \qquad (1)$$

Now assume that we know P_i and want to find P_r and P_t. Let's also assume that the following boundary conditions exist across the boundary between the two media:

1. The power incident on the boundary is equal to the power reflected off of the boundary plus the power transmitted through the boundary.
2. The velocity across the boundary is continuous.

Since p_i, p_r, and p_t completely describe the pressure wave and therefore the pressure in the system, because of our first boundary condition we can assume that

$$p_i(x=0) + p_r(x=0) = p_t(x=0), \qquad (2)$$

where x=0 is the position of the boundary. Since at $x = 0$ $e^{ikx} = 0$, we conclude that it must be the case that at the boundary

$$P_i + P_r = P_t, \tag{3}$$

giving us one equation for two unknowns and leaving us to find a second equation relating our pressure-wave amplitudes.

To do this we must introduce the concept of acoustical impedance, a term that will be critical in our discussion of the ear in general. Qualitatively, we can think of impedance as resistance to movement and in turn acoustic impedance as resistance to the movement required for sound propagation. Quantitatively we will think of it as the ratio of acoustic pressure to volume velocity,

$$Z = \frac{P}{Av}, \tag{4}$$

where Z is the acoustic impedance, A is the area propagating the sound, and v is the velocity of that area.[2] We can thus express the velocities of the waves in the following manner:

$$v_i = \frac{P_i e^{-ik_1 x}}{AZ_1}, \quad v_r = \frac{-P_r e^{ik_1 x}}{AZ_1}, \quad v_t = \frac{P_t e^{ik_2 x}}{AZ_2}. \tag{5}$$

The acoustical impedance of the incident and reflected waves is the same since they are in the same medium, only the transmitted wave experiences different acoustical impedance. Applying our second boundary condition we find that

$$v_i(x = 0) + v_r(x = 0) = v_t(x = 0), \tag{6}$$

and therefore

$$\frac{P_i}{AZ_1} - \frac{P_r}{AZ_1} = \frac{P_t}{AZ_2}. \tag{7}$$

The area term cancels and we are left with

$$\frac{P_i}{Z_1} - \frac{P_r}{Z_1} = \frac{P_t}{Z_2}. \qquad (8)$$

We can use (3) and (8) to relate P_i to P_t ,

$$P_t = P_i \frac{2}{1 + Z_1/Z_2}, \qquad (9)$$

We are ultimately interested in the ratio of the powers of the incident pressure wave to the intensity of the transmitted pressure wave. For sound waves the power is given

$$I = \frac{P^2}{Z}. \qquad (10)$$

where I is the power of the sound wave. (10) can be understood by thinking about it in terms of units. Calling upon our definition of Z we can rewrite this

$$I = PvA \qquad (11)$$

The units of pressure are N/m^2, velocity is m/s, and area m^2. Multiplying these together yields N m/s, or Watts, the units of power.

Using (10) we can define a transmission coefficient for the ratio of transmitted power as

$$T = \frac{I_{transmitted}}{I_{incident}} = \frac{P_t^2/Z_2}{P_i^2/Z_1}. \qquad (12)$$

Combining (12) with (9) we find that

$$T = \frac{4 Z_2/Z_1}{\left(1 + Z_1/Z_2\right)^2} \qquad (13)$$

We now define R, the ratio of the acoustic impedances of the two media, as $R = \frac{Z_1}{Z_2}$ and rewrite (12) as

$$T = \frac{4R}{\left(1 + R\right)^2}. \qquad (14)$$

A plot of this function is shown below (Fig. 2). It is clear that the maximum sound intensity is transferred when the acoustical impedances of the two media are equal.

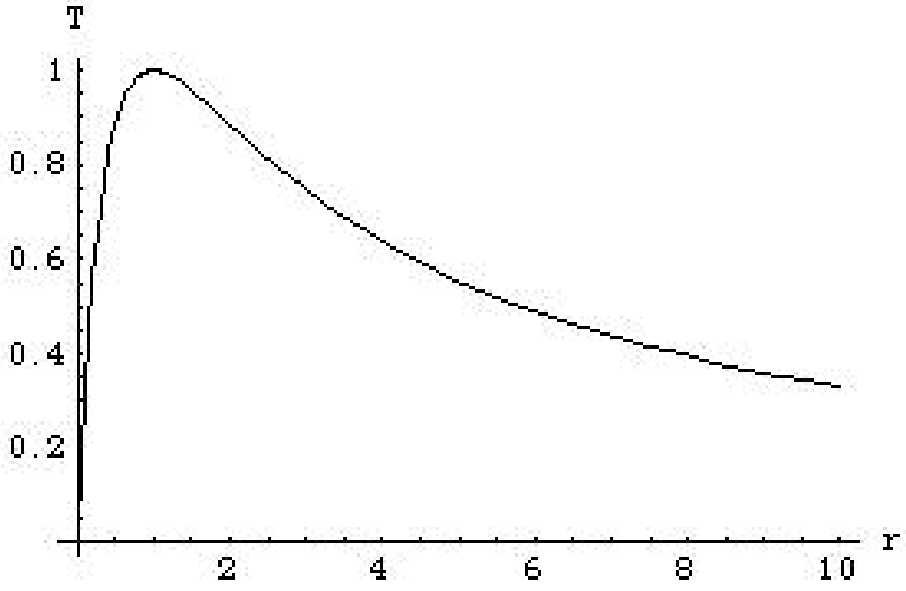

Figure 2: A plot of our transmission function. Note that where $R = 1$ the maximum power is transferred. To either side of this peak the power transfer drops off steeply.

The ratio of the acoustic impedances of water and air is 3880:1.[3] Therefore, for the ear R=0.00027 and T=0.001 meaning that 99.9% of the sound would be lost if the ear was a simple air/water interface.

The ear's solution

As we see in Figure 3 the ear is not a simple air/water interface. Indeed, the bone chain of the air-filled middle ear serves to "match" the impedances of the air of the outer ear on one side with the fluid of the inner ear on the other.

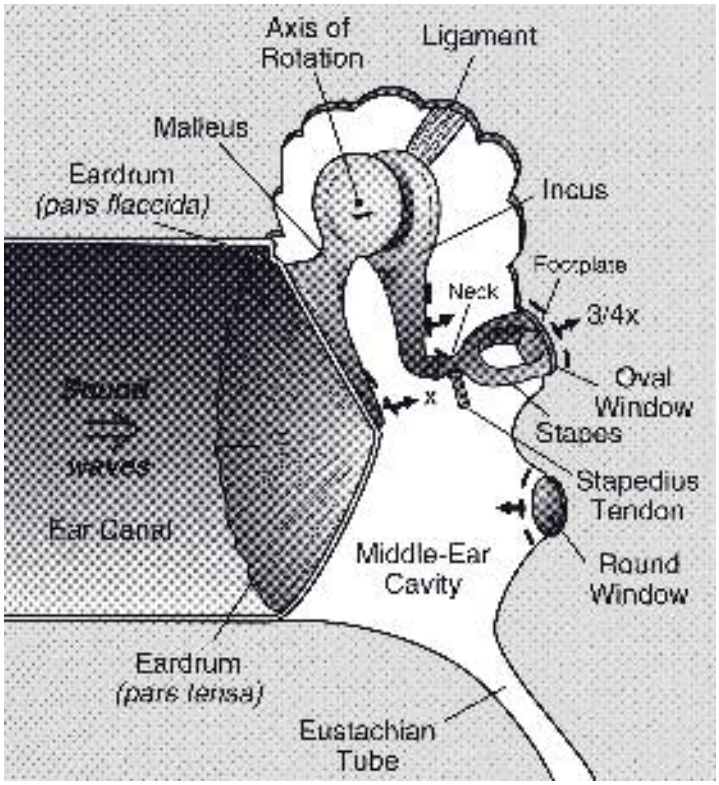

Figure 3: The middle ear is an impedance "matcher", amplifying the incoming sound waves so that they may enter into the fluid-filled inner ear without significant loss of acoustic power.[4]

The eardrum, the membrane separating the outer and middle ears, is about twenty times larger than the oval window, the membrane separating the middle and inner ears.[5] This difference in area is the pressure-amplifying mechanism of the middle ear that allows it to reduce the affects of the impedance mismatch. The bones of the chain act as levers to further amplify the pressure across the middle ear. Thus, the ear overcomes the differences in impedances of air and water and transmits sound into the fluid-filled inner ear.

The inner ear

The cochlea

The stapes, the final bone in the chain of the middle ear is fused to the oval window, a membrane opening onto the fluid-filled cochlea of the inner ear. The

vibrations of the stapes cause the oval window to vibrate, resulting in fluid displacement in the cochlea. A cross section of the cochlea (Fig. 4) shows that it is divided into three, fluid-filled chambers, which for our purposes we will think of as two chambers, reducing the upper two to a single undivided chamber. The basilar membrane separates these two chambers. We will study its oscillations in detail. Atop the basilar membrane sits the organ of Corti, responsible for sound transduction and detection. The organ of Corti runs the entire length of the cochlea, always resting on the top of the basilar membrane.

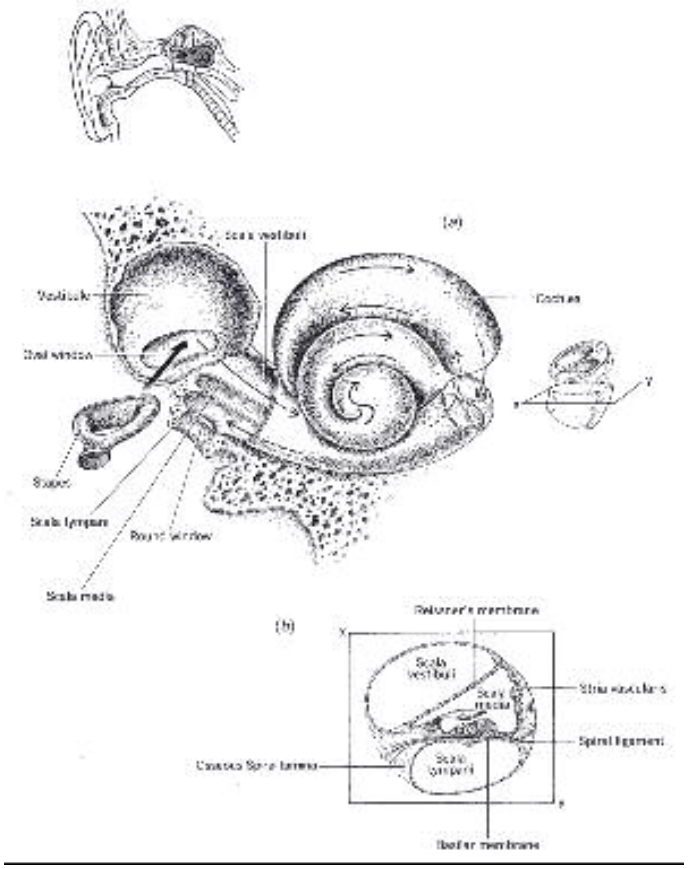

Figure 4: The final bone in the bone chain of the middle ear, the stapes, is fused to the oval window, which opens into the cochlea. Stapes movement results in fluid flow within the cochlea (see arrows in figure) that ultimately results in sound detection. A cross section of the cochlea is shown in the lower right corner. An important structure to note is the basilar membrane, the strip that divides the lower chamber from the upper section of the cochlea. Atop the basilar membrane sits the organ of Corti.[6]

The basilar membrane

If we stretch out the cochlea we can think of the upper chamber and the lower chamber as long sections separated by the thin basilar membrane (Fig. 5). The stapes is attached to the upper section at the oval window. The lower section ends at the round window, below the stapes. We need to understand how this strip, the basilar membrane, moves if we want to begin to understand the inner ear's methods of sound transduction.

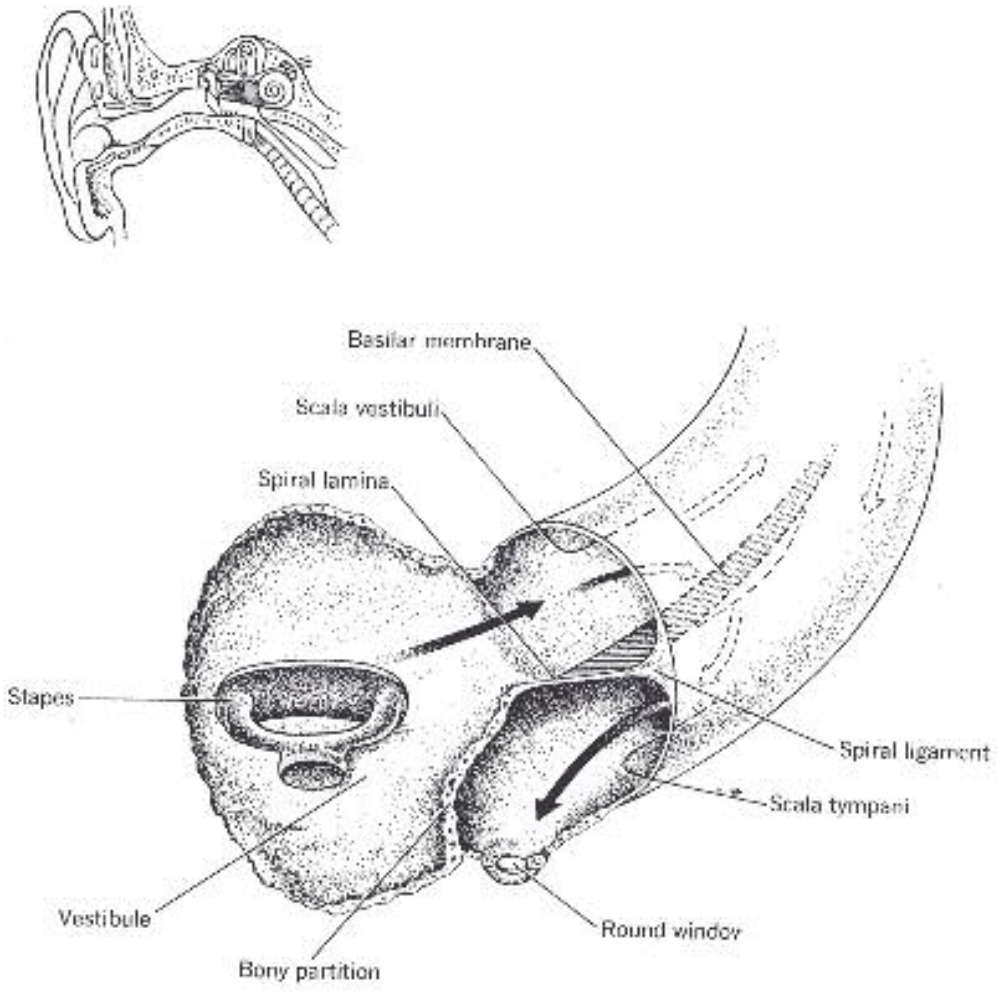

Figure 5: Looking into a cross section of the cochlea. The arrows here indicate how the fluid flow caused by the movement of the stapes affects the basilar membrane, the strip running up through the center of the cochlea. We will think of the basilar membrane as a simple strip separating two fluid filled chambers and we will discuss how fluid movement determines the movement of the membrane.[7]

The basilar membrane's movement directly affects the movement of the organ of Corti, the ear's mechano-electric transducer. Thus, understanding basilar membrane movement is critical to understanding the function of the hearing system. We will thus devote the next main section of the paper to an analysis of basilar membrane function.

Basilar membrane function

This section constitutes the main thrust of the paper. I am going to discuss the development of our theories of basilar membrane function, leaving out most of the details and less significant theories and focusing primarily on the key ideas that provide a basis for our current understanding of the ear. By the end we will have a model of how the basilar membrane responds to fluid movement in the cochlea.

Helmholtz and the resonance place theory

During the winter of 1857 Herrmann von Helmholtz gave a lecture at Bonn in which he presented a new theory of hearing based on resonance and Ohm's acoustic law, the relatively new ideas about acoustic waves published by Ohm and based on the work of Fourier.[8] Helmholtz thought of the basilar membrane as a strip of tuned resonators, transversely stretched across the cochlea, sort of like a piano. Each of these resonators had a natural frequency at which it vibrated, and thus when the ear was stimulated with a signal of, 440 Hz say, the 440 Hz resonator would vibrate, telling the brain that it had just heard an A. Since Fourier and Ohm had shown that complex waves are nothing more than combinations of pure tones, this collection of resonators would perform a Fourier transform of the waveform, breaking it down into its component parts. Thus Helmholtz thought of a system that could detect essentially any acoustical wave, where the place of

stimulation of the basilar membrane determined the frequency component of the wave, and the degree of displacement determined the wave's amplitude.

There were, however, significant problems with Helmholtz's theory and there is a clear argument against resonance being the primary means of sound detection. In order to have a resonator as frequency-selective as Helmholtz's rods are, the resonator must be very lightly damped. The more a resonator is damped, the lower its degree of frequency selectivity. The rods Helmholtz proposed would have to have had practically no damping if they were to posses the kind of frequency selectivity in existence in the ear. However, the less a resonator is damped, the more it resonates. Thus, if we were to have lightly damped resonators in our basilar membrane, they would ring far longer than is observed. We could not have the kind ability to make such fine temporal discriminations if we had ringing resonators.[9] Thus, either there is a very lightly damped resonator with high frequency discrimination but low temporal resolution, or there is a highly damped resonator with low frequency resolution but high temporal precision. There cannot be a resonator that is both lightly and heavily damped. Therefore, resonance alone is not sufficient for a theory of hearing.

Georg von Békésy and the traveling wave

While Helmholtz presented an elegant, but ultimately incorrect theory of the hearing system, he did so without ever observing what he was describing. Observing the cochlea, however, is difficult, involving opening a hole in the skull large enough to see into without damaging the cochlea so much that function is impaired. Furthermore, the movements of the basilar membrane that come from regular sound stimuli are on the order of a few nanometers, much less than the wavelength of visible light. Undaunted by

these challenges, Georg von Békésy devised a means of opening the cochlea and observing it in action.*

Von Békésy ingeniously devised a method of grinding through the skull to expose the inner part of the cochlea and the basilar membrane without detrimentally damaging the mechanism.† After grinding a hole in the skull (Fig. 8) von Békésy would sprinkle silver filings onto the basilar membrane (since it is transparent) and covered the hole with a glass plate, glued onto the bone. He would then remove the two bones adjacent to the

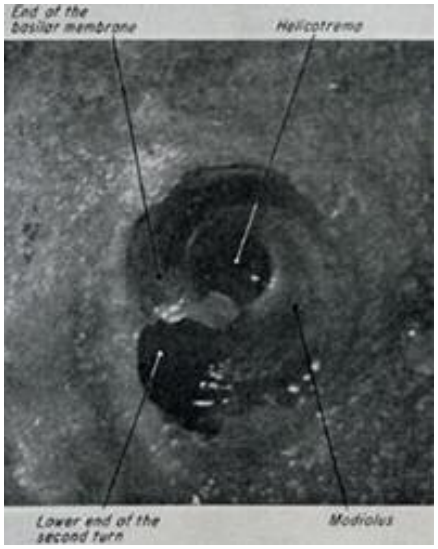

Figure 6: A picture taken by von Békésy of an exposed cochlea after grinding a hole in the skull.[10]

stapes and cement a fine wire to the stapes. This wire led to one arm of an electronically activated tuning fork and served as the driving mechanism.[11] Von Békésy would then observe the action of the basilar membrane using a technique he referred to as

* "Because for a century no numerical values concerning the mechanical properties of the cochlear partition were available, there were no restrictions on the imagination, and probably every possible solution to the problem was proposed. It seemed to the writer that the only way to solve the problem was to open up the cochlea and observe the action during the presentation of a tone." (von Békésy 1960 p. 461)

† "A temporal bone from a fresh cadaver was freed of all unnecessary tissues, and the apex of the cochlea was ground down with a fine grinding device until a small opening appeared, but without any damage to the basilar membrane…this grinding is difficult and is successful only occasionally even when done with greatest care." (von Békésy 1960 p. 425)

stroboscopic illumination. He used a stroboscope to illuminate the cochlea at a frequency twice that of the stimulus frequency and recorded the position of the basilar membrane during illumination. In order to achieve levels of displacement that could be detected with visible light, von Békésy was forced to stimulate the stapes at very high energies: "often the experiment was brought to a close by a ripping of the stapes out of the oval window." [12] Thus, von Békésy was able to observe the action of the basilar membrane during the presentation of a tone, something that at the time had yet to be achieved.

Observing the basilar membrane: the emergence of the traveling wave

The question is, of course, what was it that von Békésy saw when he observed the basilar membrane in action? He correctly identified the motion of the membrane as a type of traveling wave. That is, he saw rapidly moving, low-amplitude, long-wavelength displacements close to the stapes that got slower, higher in amplitude, and shorter in wavelength as they progressed down the membrane. At some point, though, von Békésy found that the energy of the wave completely disappears. At what is now called a critical point, the movement of the basilar membrane abruptly stops. Furthermore, directly before this critical point on the basilar membrane is where the point of maximum displacement is found (Fig. 7).

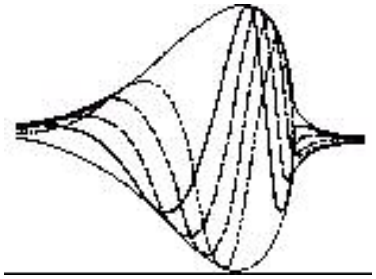

Figure 7: The motion of the basilar membrane as observed by von Békésy for a given stimulus frequency. The horizontal direction is distance along the basilar membrane and the lines represent the displacement of the membrane. From right to left each line represents a point later in time. It can be seen here how the wavelength gets progressively shorter and the amplitude greater until some critical point where the wave drops off. The wave envelope is traced around the maximum amplitude of the wave packets.[13]

Von Békésy also observed that the location of the critical point and the point of maximum displacement were dependent on the stimulus frequency. The higher the stimulus frequency, the closer to the base of the basilar membrane the critical point was and the lower the stimulus frequency, the further away from the base. In other words the basilar membrane was found to be tonotopically organized: a given stimulus frequency corresponds to a given location.[14]

How did von Békésy show that Helmholtz's assumptions were false?

Remember that Helmholtz postulated that basilar membrane movement occurs as a result of resonance with the sound stimulus. For a driven harmonic oscillator we know that resonance occurs when there is a phase shift of $\pi/2$ between the driving force and the displacement of the oscillator. Furthermore, the phase difference between the driving force and the displacement goes from 0 to π.[15] Thus, in order to determine if the basilar membrane were stimulated solely by resonance, von Békésy examined the phase relationship between the membrane displacement and the driving force at the stapes. Figure 8 shows the expected basilar membrane response if it were a purely resonant system.

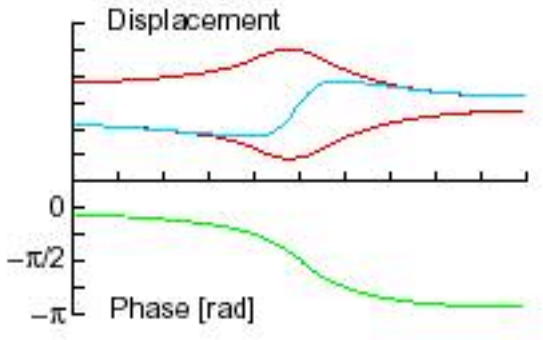

Figure 8: The upper part of this figure shows the displacement of the basilar membrane if its movement were caused by resonance with the sound stimulus. The red lines make up the wave envelope and the blue shows a possible displacement curve of the basilar membrane at a particular point in time for a given frequency. The x-axis represents distance from the stapes. The lower portion displays the phase relation between the driving frequency and the basilar membrane response. The phase shift goes from 0 to π for a resonant system. Note that at the point of resonance (where the red lines are maximally displaced) the phase shift corresponds to $-\pi/2$ as is expected at resonance.[16]

This is, however, not what von Békésy observed. Instead, he saw a basilar membrane response that more resembled what is shown in Figure 9.

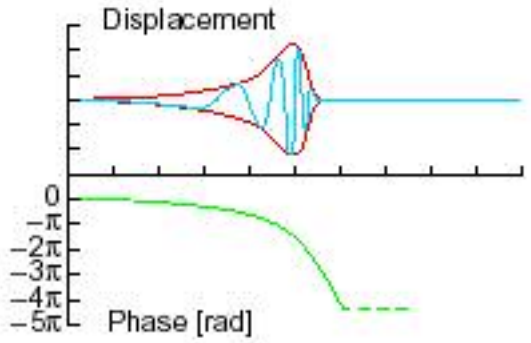

Figure 9: This shows the basilar membrane displacement as a traveling wave, as was observed by von Békésy. The x-axis represents distance from the stapes. Note that the phase relation is not that of a resonant system. Instead, the further away from the base of the membrane, the greater the phase shift of the membrane displacement. This corresponds to the phase properties of a traveling wave. Note that after the critical point the wave completely disappears—there is no phase relation.[17]

Specifically, von Békésy found that the phase relationship between the basilar membrane displacement and the driving stapes descends well below π and furthermore, that the point of maximum displacement of the membrane does not occur at a phase shift of π/2, but instead for a much greater phase shift. In fact, he found that the further away from

the stapes he measured the phase shift, the greater the observed phase shift was. Thus, von Békésy was able to conclude that the basilar membrane is not a simple resonant system. He instead found that traveling waves propagating down its length cause its oscillations.

How can we model the traveling waves in the basilar membrane?

The mass/spring oscillators

Any solution to the following differential equation is a traveling wave:

$$\frac{\partial^2 \Psi(x,t)}{\partial t^2} = \upsilon^2 \frac{\partial^2 \Psi(x,t)}{\partial x^2}, \qquad (15)$$

where Ψ is the wave function and υ is the velocity of the traveling wave.[18]

As an example, one set of solutions to this are traveling waves of the form

$$\Psi_{travel}(\vec{r},t) = A\cos\left(\vec{k}\cdot\vec{r} - \omega t\right), \qquad (16)$$

where $\vec{k}$ points in the direction of propagation of the traveling wave, which for our purposes is along the basilar membrane, and $\vec{r}$ is a position vector.

A more qualitative description of a traveling wave is the wave that shoots down a rope tied to a wall that has just been given a quick jerk. The traveling waves in the basilar membrane arise for a similar reason: the membrane is "jerked" by fluid displacement caused by the movement of the stapes. We will now develop a model that describes how it is the traveling waves in the basilar membrane arise.

We can think of the basilar membrane as consisting of a series of masses and springs.[19] These masses (Fig. 10) respond to movement of the fluid within the cochlea and serve as the medium that propagates the traveling wave. While the individual masses are not directly coupled to one another, they are coupled via the fluid in the cochlea.

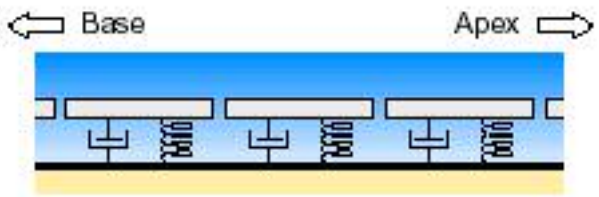

Figure 10: The mass/spring oscillators that make up the basilar membrane in our model.[20]

We can model these masses using simple differential equations, thinking of the masses and springs as driven harmonic oscillators.[21] The driving force is the fluid movement caused by the motion of the stapes pushing the mass down onto the spring. We can represent our mass/spring oscillator with the familiar

$$m\frac{d^2x}{dt^2} + h\frac{dx}{dt} + kx = f(t) \qquad (17)$$

where *f(t)* is the driving force applied by the stapes, *m* the mass, *h* the viscosity of the fluid and *k* the stiffness of the spring. Our basilar membrane, however, consists of a set of *N* driven harmonic oscillators, which we can represent as follows,

$$m_i\frac{d^2x_i}{dt^2} + h_i\frac{dx_i}{dt} + k_ix_i = f_i(t) \qquad (18)$$

where $i = 1,...,N$.

In Helmholtz's model we would be almost done here, simply having to define *f(t)* as the force generated by the stapes. Namely,

$$f_i(t) = -G_ia_i(t), \qquad (19)$$

where G_i are positive constants. If we stopped here, though, we would have *N uncoupled* mass/spring oscillators and no traveling wave: a crude approximation. We must go beyond Helmholtz's model to consider in addition a hydrodynamic term, accounting for

the coupling between the oscillators, as well as a term accounting for the drag of the fluid, a shear-viscosity term.

The hydrodynamic term is written $-\sum_{j=1}^{N} G_i^j \frac{d^2 x_j}{dt^2}$ and represents the force caused by the acceleration of oscillator *j* and transmitted to oscillator *i* by the fluid. The shear-viscosity term is given by $s_i^+\left(\frac{dx_{i+1}}{dt} - \frac{dx_i}{dt}\right) + s_i^-\left(\frac{dx_{i-1}}{dt} - \frac{dx_i}{dt}\right)$ and arises from the viscous forces caused by the different velocities of the different mass/spring oscillators. Here s_i^+ and s_i^- are the viscosity coefficients on each side of a mass *i*.

We are now ready to combine these various terms into a single equation describing the motion of our set of driven oscillators. We put all our additional terms over with the force term:

$$m_i \frac{d^2 x_i}{dt^2} + h_i \frac{dx_i}{dt} + k_i x = -G_i a_i(t) - \sum_{j=1}^{N} G_i^j \frac{d^2 x_j}{dt^2} + s_i^+\left(\frac{dx_{i+1}}{dt} - \frac{dx_i}{dt}\right) + s_i^-\left(\frac{dx_{i-1}}{dt} - \frac{dx_i}{dt}\right) \qquad (20)$$

shuffling things around a bit and setting $s_i^+ = s_i^- = s_i$ for simplicity we find that

$$\sum_{j=1}^{N}\left(G_i^j + m_i\delta_{ij}\right)\frac{d^2 x_j}{dt^2} + h_i \frac{dx_i}{dt} + s_i\left(2\frac{dx_i}{dt} - \frac{dx_{i+1}}{dt} - \frac{dx_{i-1}}{dt}\right) + k_i x = -G_i a_i(t) \qquad (21)$$

where the Kronecker delta is defined $\delta_{ij} = 1$ for $i = j$ and $\delta_{ij} = 0$ for $i \neq j$.

This is an equation of motion for the basilar membrane, showing how in the simplest sense the basilar membrane can be thought of as a collection of driven harmonic oscillators coupled hydrodynamically via fluid flow. It is interesting to see that the fluid coupling is like a mass term, in the sense that it is a coefficient of a second derivative, and that the viscous drag from the fluid is like a drag term, it goes with a first derivative.

Differential equations such as these are solved computationally to calculate how the basilar membrane behaves in various conditions. The model derived above, however, is much simpler than the models used in basilar membrane modeling today. We won't be solving this equation here and instead will consider the impedance of the membrane to model its function. This allows us to draw on what we have already learned about impedance from our discussion of the middle ear.

The impedance based model

We are still going to think about the basilar membrane as a series of masses and springs, but instead of using second order differential equations for harmonic oscillators we will think about the impedance of a given mass and spring.[22]

A fundamental property of the basilar membrane is the change in the stiffness of the springs from base to apex. The closer to the base of the basilar membrane a given spring is located, the stiffer that spring will be. The spring stiffness can be represented by an exponentially decreasing function of the form $k_i = k_o e^{-\alpha x}$ where x is the distance along the membrane, k_o some initial stiffness, and α some positive constant.

Figure 11 The varying stiffness of the basilar membrane. The stiffness decreases exponentially from base to apex.[23]

It is this change in stiffness that results in waves propagating down the membrane. We will now examine the effects of this changing stiffness on the impedance of each mass spring oscillator in the basilar membrane.

It can be shown that the magnitude of the acoustic impedance of a given oscillator is

$$|Z| = \frac{1}{A^2}\sqrt{\left(m\omega_0 - \frac{k}{\omega_0}\right)^2 + D^2} \quad , \qquad (22)$$

where Z is the acoustic impedance, A the surface area of the mass, m mass of the oscillator, ω_0 the frequency of the oscillator and correspondingly the driving force at the stapes, k the stiffness, and D a damping term.[24]

Furthermore, we know that for a driven harmonic oscillator resonance occurs when $\omega_{res} = \sqrt{\frac{k}{m}}$. Therefore at resonance the $\left(m\omega_{res} - \frac{k}{\omega_{res}}\right)^2$ term of (22) goes to zero and the acoustic impedance at resonance, Z_{res}, is a minimum:

$$Z_{res} = \frac{D}{A^2}. \qquad (23)$$

But what happens to the mass whose spring has a higher stiffness than the one corresponding to ω_{res}? Or to the one with a lower stiffness, for that matter? We will see that herein lies the key to the motion of the basilar membrane.

Let's step back for a moment and think of our system qualitatively. Let's consider driving the stapes at a particular frequency, ω_{res} say, that is the resonant frequency for some mass/spring pair of spring constant k_{res}. When the stapes pushes into the cochlea it displaces the cochlear fluid. This displaced fluid must have somewhere to

go and so pushes down on the basilar membrane trying to transfer the energy of the displacement across it into the lower chamber where it could be released at the round window (Fig. 12).

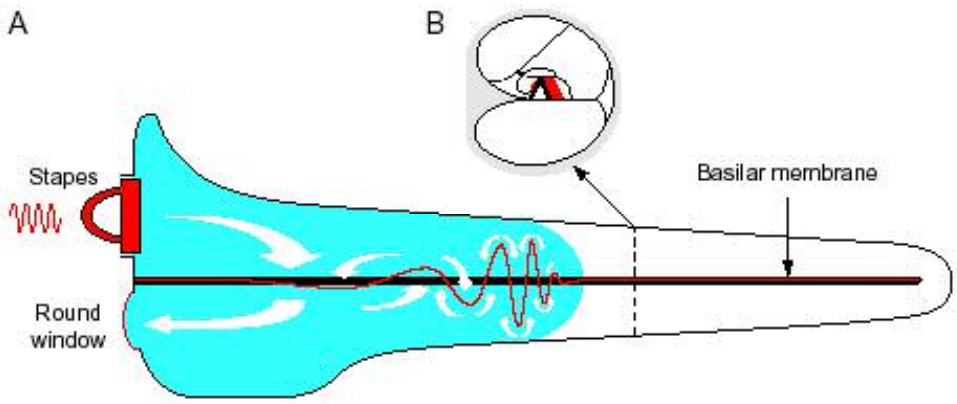

Figure 12 A: The basilar membrane and the stapes. White arrows indicate fluid movement in the cochlea. The black line down the center is the basilar membrane, the red box the stapes, the red wave the displacement of the basilar membrane. Note that coming off of the stapes the fluid pushes down on the basilar membrane and also rebounds away from the positive displacements. B: A transverse cross section of the cochlea showing the basilar membrane's location.[25]

The moving fluid displaces one of the masses in our mass/spring model, a mass that is fairly close to the stapes. This mass/spring oscillator is not the one whose resonant frequency is our driving frequency. Let's say that it is much closer to the base of the basilar membrane than our resonant mass/spring oscillator and so the stiffness of its spring, k_{stiff} say, is much higher than k_{res}. This in turn means that the acoustic impedance of this particular mass/spring oscillator is going to be very high for the driving frequency in question. Specifically, the stiffness k_{stiff} is going to dominate the impedance it is so large. Thus

$$\left|Z_{stiff}\right| \approx \frac{1}{A^2}\sqrt{\left(\frac{k_{stiff}}{\omega_0}\right)^2 + D^2}\,, \qquad (24)$$

and we know that $k_{stiff} >> k_{res}$ and so, since $\omega_0 = \sqrt{\frac{k_{res}}{m}}$ we have that $|Z_{stiff}| >> |Z_{res}|$. Thus the acoustical impedance of our first mass/spring oscillator is too high for the energy of the displaced fluid to pass through it. The spring is too stiff. It should be noted that there is some driving frequency that would be the resonant frequency for this mass/spring pair, namely that frequency equal to $\sqrt{\frac{k_{stiff}}{m}}$. For our given driving frequency, though, at this point of the basilar membrane the fluid will simply bounce quickly off the mass/spring oscillators whose spring constants are too stiff. This accounts for the low amplitude, high velocity oscillations seen at the part of the basilar membrane considerably closer to the base than the mass/spring pair that corresponds to the resonant frequency for a given stimulus.

Figure 13 shows approximately what happens when the fluid hits one of the mass/spring oscillators in our model.

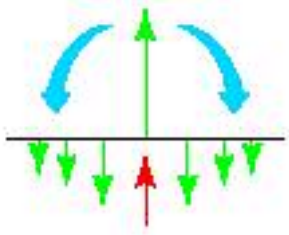

Figure 13: The black line corresponds to a single mass/spring oscillator in the basilar membrane. The arrows indicate fluid flow in the cochlea. When a mass/spring oscillator is pushed down by fluid it initially moves down but then the spring rebounds, pushing up on the fluid on top of the mass. This fluid is subsequently displaced and pushed off to the side, displacing the adjacent masses. Since the apical mass is less stiff than the basal mass it undergoes a greater displacement, explaining why the traveling wave propagates down the basilar membrane.

From this figure it is obvious why if the mass/spring oscillator is too stiff, the fluid displacement rapidly progresses down the membrane. Little time is lost in the small

displacements of the almost immovable masses and their impedance is so high that essentially no energy can be transferred through the basilar membrane to the other side.

What happens, then, as the springs get progressively less and less stiff? The impedance correspondingly decreases until the point of resonance where it is a minimum. As the springs get less and less stiff the masses are displaced more and more. This greater displacement decreases the velocity of the waves propagation down the basilar membrane, shortening its wavelength since $v = f\lambda$. This corresponds to von Békésy's observations. The impedance, though, is always too high for the energy to be effectively transferred to the other side of the basilar membrane. This results in the springing back of the masses at higher and higher amplitudes, as they are more greatly displaced. Their rebounding signifies the return of the energy back onto the upper part of the basilar membrane—it is not strong enough to make it across and was more or less rejected by that particular mass/spring oscillator. Finally, at the point of resonance, the acoustic impedance is a minimum and the energy is completely transferred. This results in very little rebound of the mass/spring oscillator back into the upper part of the membrane, explaining why the point of resonance does not correspond to the point of maximum displacement.

After this point of energy transfer the impedance again increases. This time the mass term dominates since the stiffness is so low. The impedance is then given as

$$|Z_{mass}| \approx \frac{1}{A^2}\sqrt{(m\omega_0)^2 + D^2}, \qquad (25)$$

which is clearly greater than Z_{res}.

Furthermore, essentially all of the energy was transferred from the upper part to the lower part of the basilar membrane. Therefore, there should be essentially no

displacement of subsequent mass/spring oscillators, first because of the higher acoustical impedance and second because of the lack of energy in the upper part of the basilar membrane. This corresponds to observations of basilar membrane movement where we see an abrupt drop off in displacement after the critical point corresponding to the resonant frequency. Figure 14 pictorially outlines the process just described.

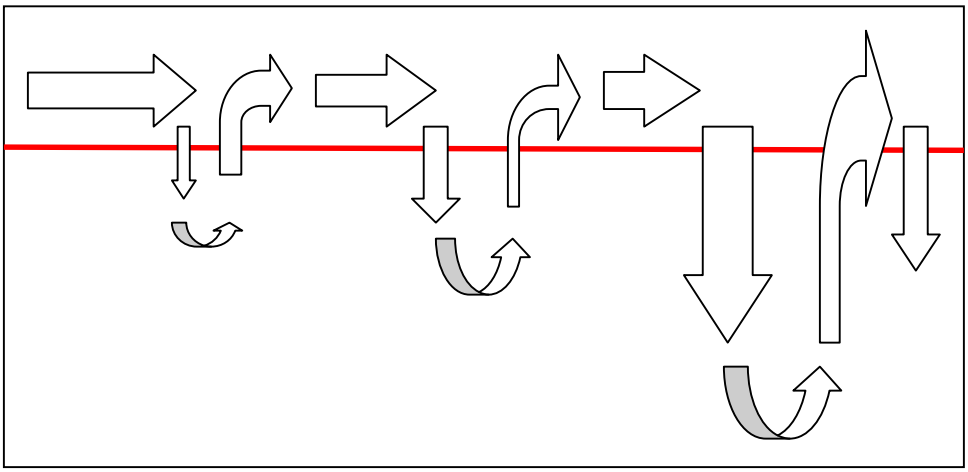

Figure 14: An energy flow diagram for the cochlea. As sound energy enters the cochlea (horizontal arrows) some of it is absorbed by the mass/spring oscillators of the basilar membrane (the red strip). The mass/spring oscillators rebound, however, causing more fluid movement, and in turn more energy in the upper portion of the membrane. Right before the mass/spring oscillator tuned to resonate with the stimulus frequency displacement is at a maximum. At the point of resonance, the energy is completely absorbed by the mass/spring oscillator and all of it is transmitted to the lower chamber with none being sent back to the chamber above.

Our model, while simple, explains the properties of the traveling wave observed by von Békésy. While it makes clear simplifications and ignores much of the subtlety of basilar membrane function, it is useful in understanding the basics of its behavior.

We will see that there is more to the cochlea than was observed by von Békésy but let's not fail to realize the contribution he made to our understanding of the hearing system. Von Békésy was awarded the Nobel Prize for his work in 1961, an award he

richly deserved. By looking into the cochlea and observing its function, von Békésy successfully solved a challenging problem. But he was unable to solve it entirely. We will now consider what von Békésy was unable to: the live cochlea.

What we have done thus far constitutes the bulk of our paper. The details of the physics of the following section are beyond the scope of this paper and a mere description of the functioning of the system in physical terms is provided.

What von Békésy didn't see: the cochlear amplifier

Johnstone and Boyle and the Mössbauer effect

In October 1967, six years after von Békésy was awarded the Nobel Prize, two researchers from the University of Western Australia, B. M. Johnstone and A. J. F. Boyle, reported using the Mössbauer effect to observe the movement of the basilar membrane in live cochleae (see Appendix B). Their article of little over a page conclusively demonstrated that there was much more to the basilar membrane than what had been found by von Békésy. Using the Mössbauer effect, they were able to calculate basilar membrane displacement within 10% accuracy in a live ear, allowing them to conclude that the basilar membrane motion they saw, "resembled that obtained by Békésy but was more sharply peaked" (Fig. 16).[26]

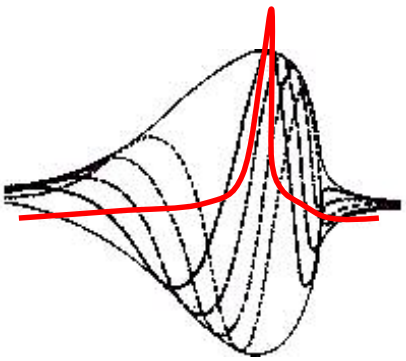

Figure 15: Here is von Békésy's sketch of the movement of the basilar membrane in black. In red, interposed in front, is more or less the kind of displacements that were found by Johnstone and Boyle. They saw a much narrower spike that was significantly more larger at the point of the basilar membrane that corresponded to the stimulus frequency.

Thus Johnstone and Boyle found that the basilar membrane did not passively respond to the oscillation of the stapes, but instead actively filtered the signal. Its displacement was greatly increased very near the point corresponding to the stimulus frequency and greatly dampened at other points. Their findings went on to be confirmed by further experiments that employed other methods of observing the live cochlea such as interferometers (see Appendix B).

Indeed, there is much further evidence pointing to the existence of a cochlear amplifier. It, for example, would be expected to make the basilar membrane response to stapes movement non-linear. That is, there would not be a one-to-one correspondence between input stimulus level and basilar membrane response. It has in fact been shown that in the living ear the movement of the basilar membrane is exceedingly non-linear as well as being very sharply tuned.[27] Furthermore, it can be shown using a thermodynamic argument that the tuning in the live basilar membrane is so sharp that it can't all come from the vibratory energy taken from the original sound input.[28] The cochlea must add some energy to the system, tuning itself.

In order for the cochlea to tune itself it must selectively control basilar membrane motion. This means that at the location on the basilar membrane corresponding to a particular stimulus frequency there is amplification, while at adjacent locations there is damping. Therefore, the energy introduced by the cochlea must be dependent on the nature of the stimulus, implying that the cochlear amplifier is a feedback mechanism. Furthermore, the amplifier must be local, in the sense that it can act at those sites along the basilar membrane that need amplification and damping. It must also be frequency dependent. In other words, the cochlear amplifier is distributed along the basilar

membrane and capable of introducing energy into the system in a frequency dependent way. In 1985 cells in the cochlea were discovered that met the qualifications for a cochlear amplifier exactly.

Outer hair cells are the cochlear amplifier

As discussed in the earlier physiology section the organ of Corti rests on top of the basilar membrane. Within this organ there are three rows of so called *outer hair cells* and a single row of *inner hair cells*, so named because of the stereocilia protruding from the cells' tops that resemble tiny hairs. The organ of Corti (Fig. 16A) and the hair cells within it run along the entire length of the basilar membrane.

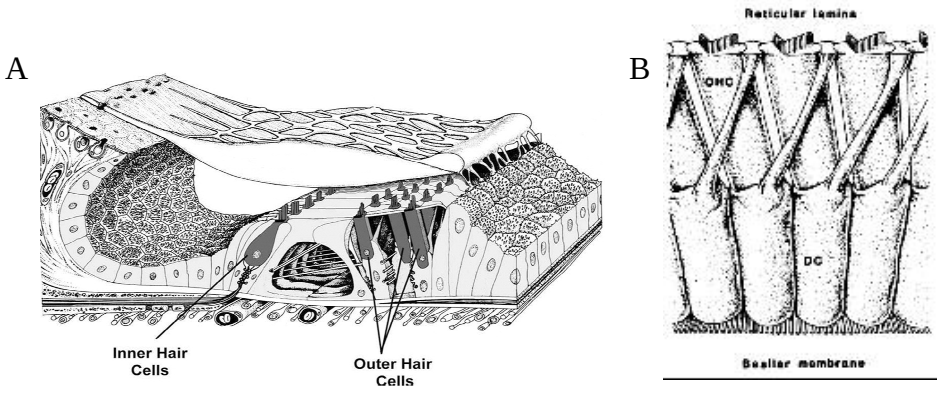

Figure 16: A: the organ of Corti, which rests upon the basilar membrane. The three rows of outer hair cells and single row of inner hair cells can be seen.[29] B: the outer hair cells in detail. At the bottom of this sketch is the basilar membrane; the top of the sketch shows the stereocilia of the outer hair cells.[30]

In 1985 William Brownell reported observing that outer hair cells (Fig. 16B) changed their shape based on the voltage across their cell membrane. We can think of a cell as an RC circuit where the resistor consists of the ion channels that allow charge in or out of the cell and the capacitor consists of the cell membrane, which is impermeable to charge flow. Along these lines we can think of the voltage across the capacitor (the cell membrane), which is governed by charge flow into the cell (Appendix A). Brownell

observed that when outer hair cells are removed from the cochlea and then stimulated electrically, their size and shape change.[31]

Two years later Jonathon Ashmore improved upon Brownell's findings, using a photoelectric diode to record the changes in cell length (Fig. 17). He was able to precisely measure how the cell's shape changed based on how much light shown past it onto the photoelectric diode.

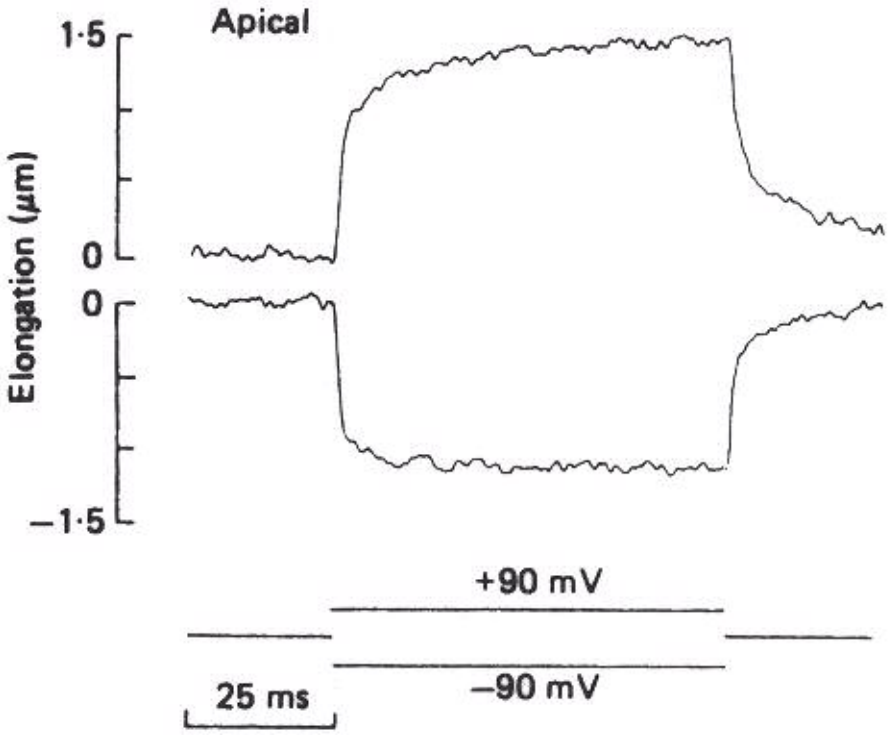

Figure 17: Ashmore's data. The upper two traces show the elongation of the outer hair cell (y-axis in μm) over time (x-axis where the bracket at the lower left corresponds to 25ms). The lines at the bottom show the voltage steps across the outer hair cell membranes.[32]

How could it be that these cells change their shape based on the potential across their membranes?

How outer hair cells work—motor proteins

Perhaps one of the most interesting findings concerning the outer hair cells is that the change in shape is not actually caused by charge moving *across* the cell membrane, but instead is caused by the movement of charge *within* the membrane itself. This is remarkable because excitable cells almost always function based on charge movement across the membrane (Appendix A).

In 1994 it was found that outer hair cell motility does not stem from charge movement across the membrane. Instead, it was found that when the voltage across the outer-hair-cell membrane was changed, the current across the membrane remained consistently inward. Normally, as the voltage across a cell membrane changes, there is a point when the electrical forces cancel out the forces of chemical dispersion and the current flowing in or out of the cell carried by a particular ion will go to zero (Appendix A). At voltages lower than this *reversal potential* the current would be inward, say, and at higher voltages it would be outward or vice-versa. In general, as the voltage across a cell membrane changes, and ions are moving across that membrane, at some voltage their movement will stop and at subsequent voltages will reverse. This was not observed. Instead, a current was found that corresponded to a two state system within the cell membrane (Fig. 18).[33]

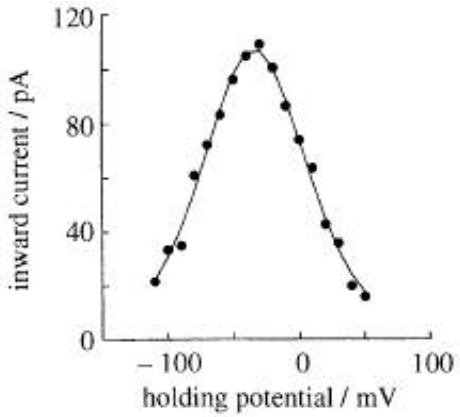

Figure 18: The inward current recorded across the membrane of the outer hair cell fits a Boltzmann distribution for a two state system. Since the current does not reverse, it simply maximizes at a particular voltage, there is no charge flow across the membrane there a shift from one state to another within the membrane. This shift of state is related to the outer hair cells dynamic shape changing properties.[34]

What could this two state system be? And how could it be related to the shape changing properties of the outer hair cells?

A system with these properties is not unprecedented in electrophysiology. Voltage-gated ion channels are some of the most studied phenomena of the field and exhibit exactly this two state behavior, where a current associated with the change of state can be measured within the cell membrane. Voltage-gated ion channels are proteins that sit in the membrane of excitable cells. They exhibit two possible configurations both of which are dependent on the voltage across the cell membrane. For some voltages the ion channels are "open" i.e. in a conformation state that allows a specific ion or type of ion to pass through them whereas at other voltages the channels are "closed" i.e. in their second conformation state where ions may not pass.

In fact, one of the ways that the function of voltage-gated-ion-channel proteins was determined was by measuring the "gating current", the intramembrane current that is perfectly analogous to the current shown in Figure 18. In the case of the voltage gated ion channels this gating current arises from the movement of charged amino acids

making up the protein within the cell membrane. This movement is what results in the protein moving from one conformation state to the other.

It is true that the outer hair cells have an unusually high density of membrane proteins, a higher density at around 6000/μm^2 of any known cell![35] It has been proposed that motor proteins make up a large percentage of the proteins found in the outer hair cell membrane. It has in fact been shown that motor proteins are the key to the motility of the outer hair cells. The main protein for outer hair cell motility was identified in 2000 and named *prestin*. It by itself exhibits the same fast motile properties as the outer hair cells.[36] (Zheng et. Al 2000).

The outer hair cells' motor protein relies on charged particles imbedded in the cell membrane. The current shown in Figure 18 comes from the movement of these charged particles that is caused by changes in the voltage across the cell membrane. Figure 19 shows how the charge movement results in the outer hair cells' motile properties.

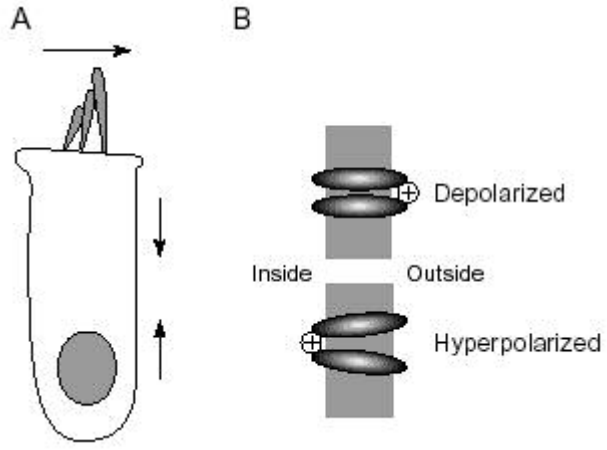

Figure 19: Motor proteins change the shape of outer hair cells (A). The voltage across the membrane determines the position of charges within the protein (B). If the voltage is more positive (depolarized), the charge would be on one side whereas for more negative voltages (hyperpolarized), the charge would be on the other. The position of this charge determines the structure and in turn the size of the protein. Since there are so many of these proteins in the membranes of the outer hair cells, its shape can be easily changed as they pass from one state to another.[37]

How outer hair cells act as the cochlear amplifier

It is here where we do not have the privilege of standing on very solid ground. Honestly, we don't know how the outer hair cells work in consort with the basilar membrane. It is recent enough that we even know more or less how they work on their own. There are a few things that we can say about how things must be or probably are. Furthermore, we can put a term into our differential equation that models the motion of the basilar membrane that will take account of the outer hair cells.

So what we want to know is how the motion of the outer hair cells actually affects the motion of the basilar membrane. We want to be able to explain the spike that was observed by Johnstone and Boyle.

The following figure shows roughly how elongation or contraction of the outer hair cells in the organ of Corti could affect basilar membrane oscillation.

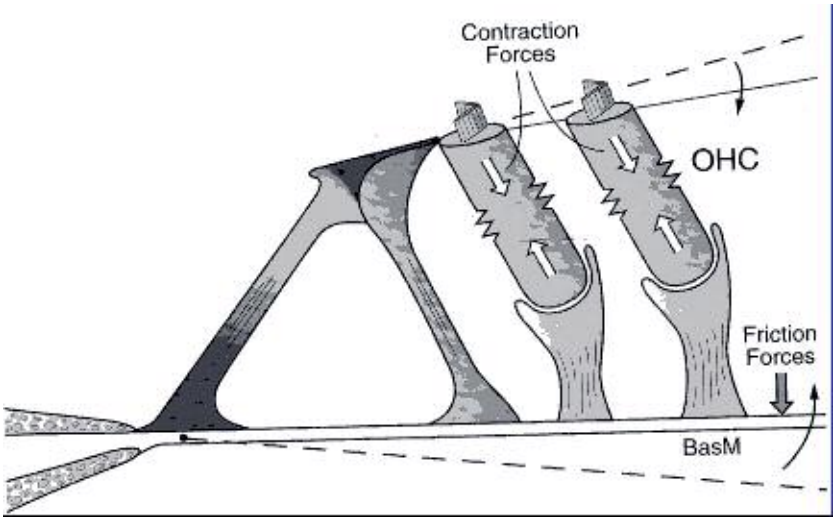

Figure 20

Figure 21: A cross section of the organ of Corti showing how we think outer hair cell forces act as the cochlear amplifier. When the hair cells contract, the basilar membrane is pulled upwards. When the hair cells elongate, the membrane is pushed down. Thus, movement of the membrane can be either dampened or amplified by the hair cells. Specifically, at positions on the membrane that correspond to the stimulus frequency the basilar membrane motion will be amplified, while at other positions it will be dampened.[38]

The outer hair cells will act as an amplifier, or anti-damper at positions of the basilar membrane very near to the position corresponding to the stimulus frequency. There they will contract, pulling the membrane up with them. At other locations, however, the hair cells will act as a damper, increasing their size and reducing the movement of the membrane. This is roughly how the tuning observed by Johnstone and Boyle can be explained.

We can imagine this mathematically be inserting an antidumping term into our differential equation modeling basilar membrane movement. To do this we simply need to insert yet one more force term into the equation since that is all the outer hair cells can impart onto the membrane. Let's call this force term $-U_i(\gamma_i)$ and have it represent the forces generated by the outer-hair-cell motors when the stereocilia of the hair cells undergoes a displacement of γ_i. Thus our differential equation becomes

$$\sum_{j=1}^{N}\left(G_i^j + m_i\delta_{ij}\right)\frac{d^2x_j}{dt^2} + h_i\frac{dx_i}{dt} + U_i(\gamma_i) + s_i\left(2\frac{dx_i}{dt} - \frac{dx_{i+1}}{dt} - \frac{dx_{i-1}}{dt}\right) + k_i x = -G_i a_i(t). \quad (26)$$

We group our new term with the other damping terms because that is the kind of flavor that this term has. It is unlike stiffness (the zero order terms), or mass (the second order terms).

Mechano-electric transduction: the inner hair cells

The movement of the stapes, the basilar membrane motion, and the tuning done by the outer hair cells all work toward a final goal: transduction of the mechanical energy of the initial sound signal into an electro-chemical signal. Within the organ of Corti is a single row of cells we call the inner hair cells, the mechanism behind the mechano-electric transduction in the ear. Similar to the outer hair cells, the inner hair cells consist

of a cell body out of the top of which protrude stereocilia. The movement of the stereocilia, caused by the movement of the basilar membrane and correspondingly the movement of the organ of Corti, is what causes the transduction of mechanical energy into an electro-chemical signal. Figure 21 shows a diagram of the inner hair cells.

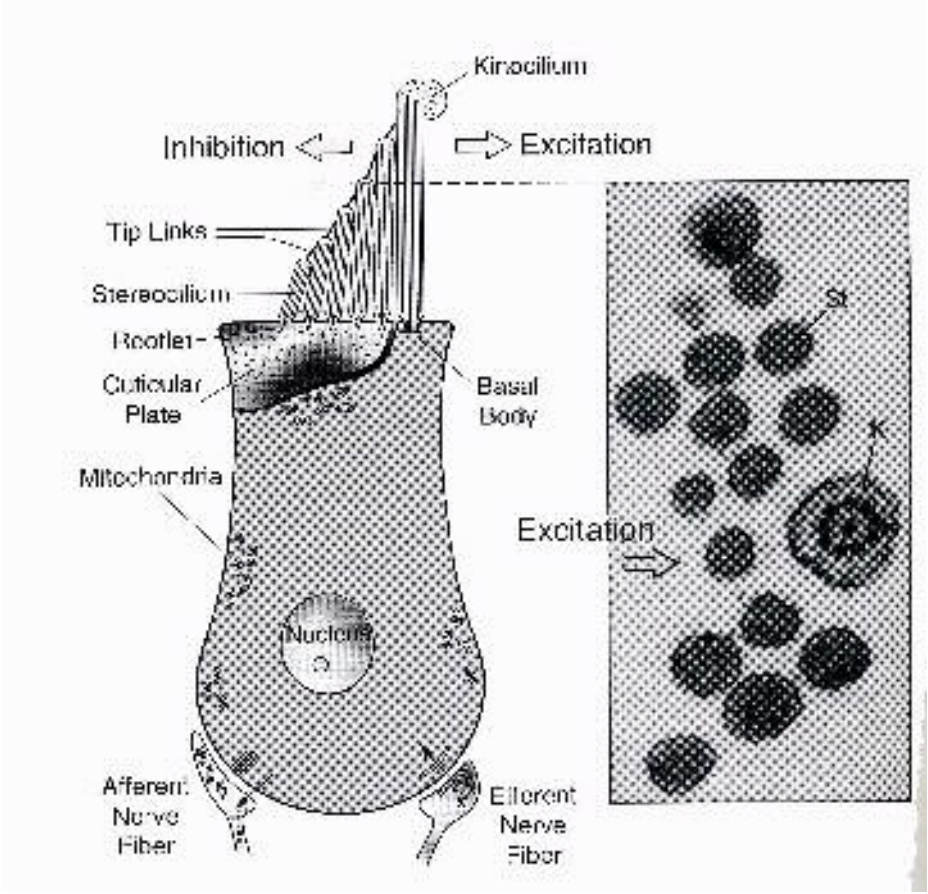

Figure 22: The left part of this figure shows a sketch of an inner hair cell. The stereocilia at the top of the cell are the vertical rod-like objects. The tip links can be seen as the springs connecting the stereocilia. Note if the stereocilia are moved to the right the hair cell is excited, the ion channels open, whereas if they are moved to the left then the hair cell is inhibited, ion channels are closed. The efferent nerve fiber at the bottom shows the neuron that will take the inner-hair-cell signal to the brain. The right part of the image shows an actual photo of hair-cell stereocilia from a single hair cell, taken looking down onto the stereocilia from above. Movement towards the large kinocilium (the big ball in the sketch labeled *K*) results in excitation.[39]

Movement of the organ of Corti causes the fluid surrounding the stereocilia of the inner hair cells to be displaced. This in turn forces the stereocilia to move. Since the

stereocilia are quite stiff, they simply pivot about their base. Running from the tip of each individual stereocilium is a *tip link* connecting the tip to the tips of the two adjacent stereocilia. This tip link can be thought of as a spring (Fig. 22).

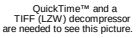

Figure 23: Two photos (left) and a sketch (right) of the tip links that connect hair cell stereocilia. The tip link can be thought of as a spring strung between the tips of the stereocilia. It is attached to two ion channels. When it is pulled tight, the ion channels are open.[40]

Movement of the stereocilia stretches or compresses these springs because of sheer between adjacent stereocilia. The springs are attached to ion channels on each side and when they are stretched these ion channels are opened; when they are compressed the ion channels are closed. Because of the chemical properties of the fluid surrounding the stereocilia (it has a high concentration of potassium ions) opening of the ion channels in the stereocilia tips allows positive charges to enter the hair cells. This lowers the membrane potential across the hair cell membrane, which in turn causes it to release neurotransmitter at the base. This neurotransmitter is detected by a neighboring neuron, which fires a signal alerting the brain that the particular inner hair cell has detected sound.

Conclusion

The process

We did it. We went from a sound wave to a brain wave and we talked about most (well…some) of the physics in between! Let's review just to remember where we started and where we finished.

It all begins with sound, a pressure wave consisting of periodic changes in air pressure density. This pressure wave causes our eardrums to vibrate, which in turn causes the bone chain in our middle ear to move. The bone chain, remember, is like an impedance matcher and serves to transform the sound pressure wave that had been in air into a sound-pressure wave in the fluid filling our inner ears. At the other end of the bone chain is the oval window, opening into the cochlea. The vibrations of the oval window result in fluid movement in the cochlea, causing a traveling wave to form in the basilar membrane. The outer hair cells filter and amplify this traveling wave forming it into a spike in the membrane that corresponds to a specific stimulus frequency. This spike in the membrane causes the organ of Corti at that point to move, which in turn causes the fluid surrounding the stereocilia of the inner hair cells to oscillate. The shearing of the stereocilia results in stretching and compressing of the hair cell tip links, which causes ion channels in the stereocilia to open and close. When the ion channels are open, positively charged potassium ions flow into the cell raising the voltage across its membrane. This finally, triggers the cell to release neurotransmitter from its base, which is in turn detected by a neighboring neuron that finally sends a signal to the brain saying that a particular hair cell had detected the presence of a sound.

Everything I didn't talk about but want to someday

This may strike you as ridiculous and perhaps even implausible, but the whole process is actually far more complicated than what I just described above. For example, how exactly do the outer hair cells affect the basilar membrane's motion? And for that matter, is just sticking that force term into the differential equation really going to account for their influence? And what about the inner hair cells? We barely talked about them. There must be more to it all.

Yes, I concede. There are about a hundred more comps papers waiting to be written about pretty much any single section of this one. But I have a word limit and so, to finish, I will simply refer you to the papers I and others will be writing in the years to come. There is a lot more to be said about the ear. We have a lot of watching to do, to follow Yogi's advice, and are still humbled by the truth of von Békésy's comment that physical science provides little knowledge about the true nature of hearing.

With the wisdom of those two and all the other people who have studied the ear in mind I hope to tackle the question of how it is that we can process speech sounds. This paper only talks about processing pure tones, and if you thought that was complicated, you should try speech at two hundred words a minute! So hopefully, someday soon, there will be a few more appendices to add.

Appendix A: The Nernst Equation

Neurons are characterized by their excitability. When the electric potential across an excitable cell's membrane exceeds a certain threshold, that cell will fire an action potential, the method of signaling in the nervous system. Therefore it is very important to have a means of talking about the membrane potential across cell membranes.

Action potentials occur when a cell's voltage depolarizes enough (goes from negative to closer to zero) that sodium-selective, voltage-gated ion channels in the cell membrane open. These ion channels allow positively charged sodium to rush into the cell, further depolarizing it until a certain positive-voltage limit is achieved. At this point the sodium channels are deactivated (essentially blocked) and potassium ion channels are opened, allowing potassium to rush out of the cell, hyperpolarizing it (making its potential more negative), and bringing it back to its steady state, readying it to fire another action potential.

But why does potassium rush out of the cell and sodium rush in? And what is the cell's "steady state" and how is it determined? Electrochemical gradients are the basis of the answer to both of these questions.

To answer these questions we will think of an idealized cell with a single ion channel that allows only potassium ions to pass. Furthermore, let's say there is far more potassium inside the cell than outside (a situation achieved in Nature thanks to the sodium-potassium pump). Since the intracellular potassium concentration, $[K^+]_{in}$, is so much larger than the extracellular potassium concentration, $[K^+]_{out}$,

$$[K^+]_{in} >> [K^+]_{out}, \qquad (A-1)$$

forces of dispersion push K^+ through the open ion channel and out of the cell. These forces of dispersion can be characterized by chemical gradients. Every time a K^+ ion leaves the cell it leaves an anion behind (our solutions are electroneutral to begin with). After enough K^+ ions have left the cell due to the chemical gradient, a sizable voltage across the cell membrane arises. The K^+ ions that leave are drawn to the outer part of the membrane where they "pair up" with anions on the other side of the membrane. In this sense the membrane acts like a capacitor with a voltage across it. This voltage is created by the positive K^+ ions on one side and the negative anions on the other. This voltage, though, is creates an electrical force that pushes the K^+ ions back into the cell. Remember, they are the only things that can get through the cell membrane. So we have chemical gradients pushing K^+ out and electrical gradients pushing K^+ in. Eventually things have to settle down so that this cell can once again be at a state of equilibrium. When will this happen?

Quite simply, it occurs when the electrical and chemical gradients are equal. That is, when the electrochemical gradient is zero. This occurs at what is called the ion's (in this case potassium's) reversal potential or E_r. This is defined by a potential because remember that we decided that the concentrations of K^+ inside and outside the cell were fixed. Therefore, the only thing that can change is the electric potential across the cell. But how can we find E_r?

We need to think about Boltzmann distributions and equilibrium. Let our cell be at E_r, which is, for our cell, also the resting potential since it is only permeable to K^+. We could completely describe the K^+ ions as being in two states, inside the cell, or outside of it. Furthermore, we could think about there being some probability of a given K^+ ion

being in an outside-the-cell state or an inside-the-cell state. Finally, using our knowledge of statistical mechanics, we can describe the ratio of those probabilities using a Boltzmann distribution:

$$\frac{p_{out}}{p_{in}} = \exp\left(-\frac{\Delta E}{kT}\right) = \exp\left(-\frac{u_{out} - u_{in}}{kT}\right), \qquad (A\text{-}2)$$

where p_{in} is the probability of K^+ being in the cell, p_{out} is the probability of K^+ being outside the cell, u_{in} and u_{out} are the intra and extracellualar energies respectively, T is the temperature in Kelvin, and k is Boltzmann's constant. We can replace the probability in the above equation with ion concentration c and the energy with molar energy U to get

$$\frac{c_{out}}{c_{in}} = \exp\left(-\frac{U_{out} - U_{in}}{RT}\right), \qquad (A\text{-}3)$$

where R is the gas constant ($R=kN$). Taking the log of both sides yields,

$$U_{in} - U_{out} = RT \ln\frac{c_{out}}{c_{in}}, \qquad (A\text{-}4)$$

where $U_{out} - U_{in}$ is the molar electrical energy difference of our cell's permeable ion. It is caused by the potential difference between the outside and the inside of the cell, $E_{out} - E_{in}$. Thus if we consider a mole of K^+ that has charge q it follows that

$$U_{in} - U_{out} = qF\left(E_{in} - E_{out}\right) = E_{tot}, \qquad (A\text{-}5)$$

where F is Faraday's constant. Substituting this in for $U_{out} - U_{in}$ it follows that

$$E_{tot} = \frac{RT}{qF} \ln\frac{c_{out}}{c_{in}} = E_r. \qquad (A\text{-}6)$$

For our cell with K^+ we would say

$$E_{K^+} = \frac{RT}{qF} \ln\frac{[K^+]_{out}}{[K^+]_{in}} \qquad (A\text{-}7)$$

This relation is called the Nernst Equation. [41]

Appendix B: Experimental methods of observing basilar membrane movement

Mössbaur Effect

Just as electrons paired with atomic nuclei have various energy levels, the nuclei themselves can occupy various energy states. And in the same way that an electron can move from a lower to a higher energy state through the absorption of a photon of the proper energy, so can a nucleus change energy states by the absorption of photons of particular energies. Thus, when gamma rays of particular energies strike atomic nuclei in ground energy states, the gamma rays are absorbed and the nuclei are excited. The *Mössbauer effect* refers to the technique of moving gamma rays relative to a stationary absorber, sending them through a range of energies due to the Doppler effect, and seeing which energies of gamma rays were absorbed by the absorber in order to determine the energy level structure of the atomic nucleus in question.

This effect can be applied to the hearing system in the reverse of how it is normally employed. Here, a gamma ray source is placed on the basilar membrane. An absorber with a known energy structure is interposed between the gamma source and a detector. Since the energy of the gamma ray is dependent on the velocity of the source, the rate of detection of the gamma rays is in turn dependent on the velocity of the basilar membrane, upon which the source is resting. The absorbed gamma rays cannot be detected. Thus, the velocity of the basilar membrane can be determined. This technique poses a particular challenge, however. The velocity function is decidedly nonlinear and undistorted measurements of basilar membrane velocity are possible only over a very short range.

Optical techniques: Interferometers and fiber optic probes

Most labs that currently observe cochlear vibrations employ optical techniques instead of the Mössbauer effect. This offers more sensitive measurements and more significantly is linear and so provides reliable measurements over a greater range of velocities than the Mössbauer effect technique.

One such optical technique is the heterodyne laser interferometer. Light from a single laser is split into two beams whose frequencies are then shifted so that they differ by some small Δf. The first beam is then reflected off of a vibrating target, in this case one that is fixed to the basilar membrane. The second beam simply serves as a reference. When the beams recombine a beat signal emerges. Depending on the vibrations of the target of the first beam there are phase modulations to the beat signal that are dependent on the changes in the optical path length of the light of the first beam due to the vibrations of the reflector. From these phase modulations the vibrations of the reflector and therefore of the basilar membrane can be calculated.

A second optical technique does not involve interferometry but fiber optic devices. Here, a fiber optic device that can both emit and detect light is inserted near the cochlea. Light from the device is shined on the basilar membrane, and the fiber optic device itself detects the reflections of this light off of the membrane. Movements of the basilar membrane are quantified in terms of changes in the amount of reflected light detected by the device.[42]

Annotated Bibliography

Texts on hearing

W. L. Gulick, *Hearing: physiological acoustics, neural coding, and psychoacoustics* (Oxford University Press, New York, 1989).

This was my first introduction to the hearing system. It is a good entry-level textbook written more to biologists than physicists. It covers a broad range of topics and helped me focus my interests. It also has an extensive bibliography that showed me my next steps.

C. D. Geisler, *From sound to synapse: physiology of the mammalian ear* (Oxford University Press, New York, 1998).

Geisler includes a lot more physics than Gulick. This was the book I really used to learn the details that actually went into my paper. It has derivations, equations, and a lot of insight into what is going on. The level of depth of my paper is similar to the level of depth in this book.

P. Dallos, A. N. Popper, R. R. Fay, Eds., *The cochlea* (Springer Verlag, New York, 1996).

This is a collection of nine extended reviews on all aspects of the cochlea. It is fascinating and deep. In my paper I only scratched the surface of what is discussed in this text. Reading it, though, was critical for me in developing an understanding of the topic, and gaining a sense of the vast complexity of the problems I was addressing.

Texts on physics

T. D. Rossing, *The science of sound*, second edition (Addison-Wesley, Reading, Mass., 1990).

While little of this book went into my paper, I turned to it whenever I was wondering something about the way sound worked. I specifically relied on it for learning about sound pressure, intensity, decibels, and a bit about resonance and waves.

I. G. Main, *Vibrations and waves in physics*, third edition (Cambridge University Press, Cambridge, 1993).

I used this text a lot to learn about traveling waves in general and particularly to learn about the way waves travel in fluid. It is a really nice physics text that discusses in detail almost every conceivable property of waves or vibrations. I specifically used it for a mathematical treatment of traveling waves as well as to derive an expression for the dispersion relation of the traveling wave in the basilar membrane. I initially thought that was going to be the way I explained its behavior.

Primary sources

R. Nobili, F. Mammano, and J. Ashmore. How well do we understand the cochlea? *Trends in Neurosciences*, **21**:159-167, 1998.

This paper might as well be a six-page summary of my own paper. Whenever I had a basic question about what I was talking about or was unclear about the best way to explain a certain point, I would refer back to this paper. I must have read it twenty times throughout the course of my project. I would recommend it to anyone simply for the sheer pleasure of reading such a succinct, well written, and interesting piece of writing. Its bibliography proved to by my jumping off point for most of my research.

G. von Békésy, *Experiments in hearing* (McGraw-Hill, New York, 1960).

This seven-hundred page behemoth outlines every aspect of von Békésy's work in staggering detail. He starts with ancient Persia and works his way to the traveling wave. It was published the year before he was awarded the Nobel Prize and clearly shows why he deserved it. I used it to learn about his experimental methods, to see what his approach was to studying the cochlea, and to learn about what he thought about basilar membrane function. It is an essential work for a project of this nature.

B. M. Johnstone, A. J. F. Boyle. Basilar membrane vibration examined with the Mössbauer technique. *Science*, **158**:389-390, 1967.

This tidy little paper of little over a page needs little discussion. It shows us conclusively that there is a lot more to a live cochlea than there is to a dead one. It is seminal in the study of the active cochlea.

W. E. Brownell, C. R. Bader, D. Bertrand, Y. de Ribaupierre. Evoked mechanical responses of isolated cochlear outer hair cells. *Science*, **227**:194-196, 1985.

What I like best about this paper is its simplicity. Brownell writes to *Science* saying basically that he extracted a live outer hair cell (good luck trying that at home) and was able to see that it moved in a voltage-dependent way. People had been nudging towards this conclusion for several years but he was the first to actually see it.

J. E. Gale and J.F. Ashmore. Charge displacement induced by rapid stretch in the basolateral membrane of the guinea-pig outer hair cell. *Procedings of the Royal Society of London* B., **255**:243-249, 1994.

I love this paper. In it Ashmore and Gale basically describe every single thing they could possibly do to an outer hair cell: they poke it, pull it, shock it, shake it, and all the while are recording the current flow across its membrane as well as monitoring its size. They calculate the capacitance of the hair cell and even determine how much force it exerts in Newtons! It got me really interested in the properties of the outer hair cells.

End Notes

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[3] Geisler, p. 27.
[4] Geisler, p. 40.
[5] Geisler, p. 41.
[6] W. L. Gulick, *Hearing: physiological acoustics, neural coding, and psychoacoustics* (Oxford University Press, New York, 1989), p. 94.
[7] Gulick, p. 115.
[8] Gulick, p. 60.
[9] Gulick, p. 62.
[10] G. von Békésy, *Experiments in hearing* (McGraw-Hill, New York, 1960), p. 425.
[11] von Békésy, pp. 425-427.
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[13] von Békésy, p. 462.
[14] W. L. Gulick, *Hearing: physiological acoustics, neural coding, and psychoacoustics* (Oxford University Press, New York, 1989), p. 65.
[15] I. G. Main, *Vibrations and waves in physics*, third edition (Cambridge University Press, Cambridge, 1993), pp. 59-61.
[16] R. Nobili, F. Mammano, and J. Ashmore. How well do we understand the cochlea? *Trends in Neurosciences*, **21**:159-167, 1998.
[17] Nobili 1998.
[18] Main, p. 146.
[19] see Nobili 1998 and Geisler, pp. 55-64.
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[23] Nobili 1998.
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[25] Nobili 1998.
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[29] This image was provided by Fernán Jaramillo, personal communication.
[30] W. E. Brownell, C. R. Bader, D. Bertrand, Y. de Ribaupierre. Evoked mechanical responses of isolated cochlear outer hair cells. *Science*, **227**:194-196, 1985.
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[35] Dallos p. 404.
[36] J. Zheng, W. Shen, D. Z. Z. He, K. B. Long, L. D. Madison, P. Dallos. Prestin is the motor protein of the cochlear outer hair cells. *Nature*, **405**:149-155, 2000.
[37] Nobili 1998.
[38] Geisler p. 81.
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[40] This image was provided by Fernán Jaramillo, personal communication.
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