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The cochlea as a graded bank of independent, simultaneously excited resonators: Calculated properties of an apparent 'travelling wave'

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ABSTRACT

There have been two main theories of how the cochlea works: resonance and travelling wave. The first says the cochlea comprises a bank of tiny resonating elements, like piano strings, which respond directly to sound pressure (the excitation is *in parallel* to the elements). The second considers that differential pressure across the basilar membrane causes a hydrodynamically coupled wave to propagate, like a ripple on a pond, from base to apex (i.e., the excitation is *in series*). Yet a bank of graded, independent resonating elements, if simultaneously excited, will give rise to an *apparent* travelling wave, as each element builds up and decays, governed by its Q. Here we model a bank of resonators ranging from 1 to 10 kHz and possessing Q values from 12 to 25, in line with reported values and in accord with a recent surface acoustic wave (SAW) model of the cochlea. When simultaneously excited, the bank shows an apparent travelling wave moving from base to apex with a speed of several metres per second, a value similar to experiment. We conclude that the 'travelling wave' can be interpreted as arising from resonant activity.

INTRODUCTION

The accepted theory of hearing is the travelling wave theory, due to Békésy (Békésy, 1960), in which a hydrodynamically coupled wave propagates along the elastic basilar membrane like a ripple on a pond. Certainly, it has much theoretical backing, derived from transmission line theory, and the observations seem to confirm that there is a wave of activity in the cochlea progressing from base to apex (at several metres per second).

But do such observations really provide confirmation of the theory? Perhaps it is only a happy coincidence, and the wave of activity actually derives from some other cause.

There are a number of reasons to question the validity of the travelling wave theory, and they are set out comprehensively elsewhere (Bell, 2004a, 2005). But to sharpen the discussion, it is worth setting out one particular point that the travelling wave theory asks us to accept. Experiments have shown that at the threshold of hearing (0 dB SPL), the displacement of the basilar membrane amounts to about 1 nanometre (de Boer and Nuttall, 2010), and that is *after* the cochlear amplifier has provided a boost of 10^3 or more, meaning the cochlea can detect a passive displacement of 10^{-12} m or less. That is a *subatomic* dimension, and fundamental physics makes us pause to think: can the auditory system really be that sensitive? Are we asking too much of the stereocilia on hair cells to detect such a deflection, which is presumed to be caused by a pressure difference across a basilar membrane? In other words, is this differential pressure really the effective stimulus to the cochlea, or are we missing the true stimulus and mistakenly looking at a side-effect?

This question is one that struck Békésy himself. In his Nobel Prize acceptance speech (Békésy, 1961/1999) he said:

> there are many types of waves [in the cochlea] and not just one. There are: (a) compression waves, (b) shear waves, (c) dilation waves, (d) Rayleigh waves, (e) ordinary bending waves. My question was, which of these waves are present in the inner ear, and which one contributes to the stimulation of the auditory end organs?

He goes on to say he "finally became convinced" that, at least at lower frequencies, "the ordinary bending of the basilar membrane furnished an adequate description of the vibrations that stimulate the nerve endings." At the same time, one should keep in mind that Békésy made his observations on cadavers using extreme sound pressure levels (120 dB or more). Could he have missed something and been misled?

There is reason to believe that the live cochlea operates in a different way, particularly at low sound pressure levels (<60–80 dB SPL), a region where the 'cochlear amplifier' (Davis, 1983) comes into play. I have come to the conclusion that Békésy's option (a) – compression waves – are far more likely to be the effective stimulus (Bell, 2007a). When one listens to an amplified recording of the ringing sounds emitted by a healthy human cochlea (so-called spontaneous otoacoustic emissions) they are reminiscent of a bank of chimes. To my ears, something seems to be ringing, and maybe

Helmholtz and his resonance theory were right after all (Bell, 2004b).

Based on comparative anatomy and related considerations, one can assemble a case that the outer hair cells (OHCs) in the mammalian cochlea are pressure sensors (Bell, 2008), and the test-tube like body of the cell detects the compression wave generated in the cochlear fluids by inward movement of the stapes. The wave sweeps through the cochlea at the speed of sound in water, meaning it could stimulate all the pressure-sensitive hair cells virtually instantaneously (within micro-seconds).

What happens if a bank of graded resonators are instantaneously excited?

This paper examines this question. If there is a bank of resonators in the cochlea that are set off by a compression wave, then might the result be something that resembles the conventionally understood travelling wave? If the answer is yes, then there is reason to suspect that resonance may underlie the effect, particularly if the assumption involved is a simple physical one (accepted sharpness of tuning, or quality factor Q, of the component resonators). By way of contrast, we should remember that the parameters underlying conventional travelling wave models are complex, and cochlear modellers, decades after Békésy, are still struggling to get the numbers right (de Boer and Nuttall, 2010).

The apparent speed of a wave moving along a bank of resonators can be simply calculated. All we need are the resonant frequency and Q value of each of the resonators and the distance between them. In the case of the cochlea, these are well known. The standard frequency–place map (Greenwood, 1961) gives the frequency and location of each resonator, and the Q values can be found from psychophysical and otoacoustic measurements (Shera et al., 2002).

Incidentally, these Q values also match the values calculated by a particular feedback model of how the three rows of outer hair cells work together to create a single tuned element, an aspect we will return to later.

We draw these accepted values together and simply calculate the apparent speed of a wave simultaneously excited in a bank of such cochlear resonators. As we will see, the calculated wave speed is in accord with accepted travelling wave velocities.

PARAMETER SET AND CALCULATIONS

The cochlea comprises a graded bank of oscillators set out upon the organ of Corti, which in turn lies upon the supporting structure of the basilar membrane. The oscillators range in frequency from 20 kHz at the base to 20 Hz at the apex, a range of 3 decades or 10 octaves. There are thousands of hair cells, both outer hair cells and inner hair cells, and they occupy a length of 30–35 mm in humans (Slepecky, 1996).

Our simple task is to take this bank of graded resonant elements in an idealised cochlea and then drive them all simultaneously with a stimulus. Depending on their frequency and Q, each will take a different time to reach maximum amplitude, an effect that will give rise to an apparent wave – an envelope of activity – travelling from the part where responses reach a maximum most quickly to where responses are slowest. We calculate the speed of this wave.

To simplify matters, we look only at the range of 1 to 10 kHz, since this is where information is readily available in the literature, although there is no reason that more detailed studies cannot extend the analysis to higher and lower frequencies.

To specify what happens in our selected system, a small number of well-known parameters are needed.

Quality factor, *Q*

Any oscillator will have a damping coefficient α (which will cause the amplitude of a passive oscillator to decrease), and more appropriately in the context of active oscillation, a cycle-by-cycle gain factor β (which will cause the amplitude to increase). Together α and β determine the sharpness of tuning, or quality factor Q, of the oscillation. The higher the Q, the greater the amplitude of the oscillation that will occur in response to an applied synchronous force (indeed, the oscillation will be Q times larger). The Q is defined to be the ratio of the frequency of the oscillator, ω_0, divided by the full-width, $\Delta\omega$, of the response curve at $1/\sqrt{2}$ of its height – the half power criterion (Fletcher, 1992, §2.5, 2.8). That is,

$$Q = \omega_0/\Delta\omega. \quad (1)$$

In other words, an oscillator that is sharply tuned will have a high Q, and one broadly tuned will have a low Q. Clearly, if one is dealing with a resonator sensor, and wish to make it respond to a weak force (signal) imposed on it, one is better off with a high-Q system (Jackson, 2004, Ch. 2).

More germane to our interests, Q is also a measure of how long an oscillator takes to respond to a transient. In general, an oscillator will take Q cycles to exponentially die away to a negligible amplitude ($1/e^3$) when the sustaining energy is switched off, or alternatively, it will take Q cycles to build up to maximum amplitude when a stimulus at the oscillator's natural frequency is applied. A feedback oscillator, for example, will take Q cycles to reach almost maximum output when it is switched on, and it is this condition we are imagining to occur when the cochlear amplifier is called upon to amplify a sound stimulus entering the cochlea.

Cochlear *Q*

In the case of the cochlea, the Q of all the individual resonators is well studied. Different approaches can be taken, but the two main methods are psychophysical (subjective) ones, in which the effective Q of the cochlear elements is inferred from masking experiments and otoacoustic (objective) techniques where the Q is derived from studies of the evoked otoacoustic responses of the cochlea to sound impulses. Although there are particular differences, work by Shera et al. (2002) has shown a broad coherence in the values obtained by the two methods. Shera and colleagues find empirically that the Q of the cochlea at any frequency f (in kHz) is given by

$$Q = 12.7 f^{0.3} \quad (2)$$

and this relation is plotted in Figure 1 over the range 1 to 10 kHz.

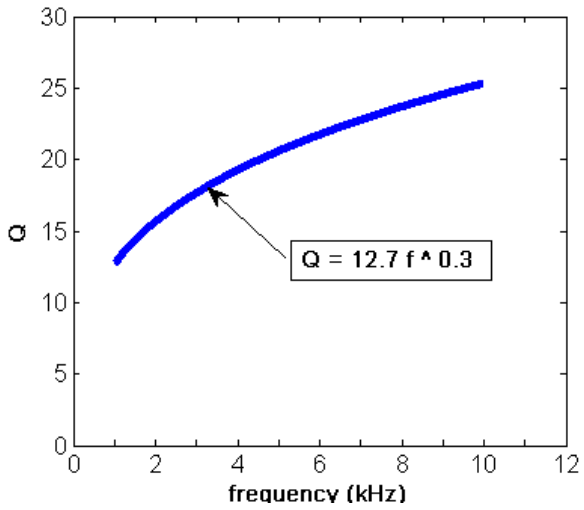


Figure 1. Variation of cochlear Q with frequency. The line marks the empirical function determined by Shera et al. (2002) from a combination of psychophysical and oto-acoustic measurements.

Calculation of wave velocity

This relation allows us to calculate the delay at each point resulting from simultaneous stimulation applied to all the cochlear oscillators. Each oscillator will reach a maximum response after Q cycles, and since one cycle is simply the inverse of the frequency, f, the time delay at each frequency will simply be

$$\text{delay} = Q \times (1/f) \times 1000 \text{ ms} \quad (2)$$

This relation is plotted in Figure 2.

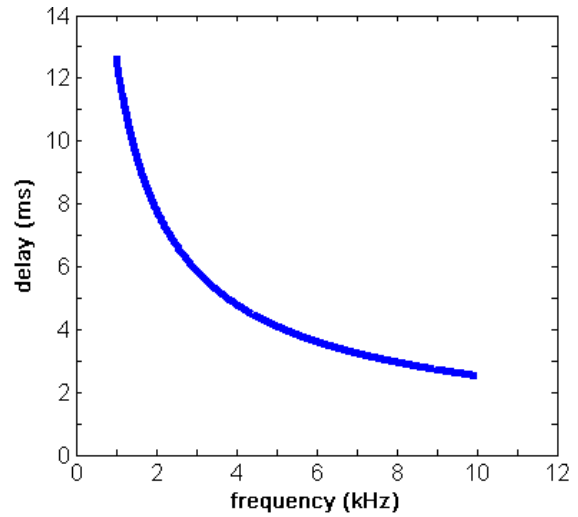


Figure 2. Delay of cochlear resonators (ms) against characteristic frequency.

Immediately we see that the shortest delay occurs at high frequencies (the 10 kHz point near cochlear base) and then progressively increases as we go towards the 1 kHz point, which is found near the cochlear apex. This can be directly interpreted as a wave of activity appearing to move from base to apex, which is precisely what the travelling wave theory tells us (although, to pre-empt the discussion, its underlying assumption is that the wave is due to coupling along the basilar membrane – that is, that the wave moves from one element to the next in a causal chain).

At this point we are in a position to ask what the speed of this apparent wave is. To calculate this we need to know the distances between the resonating elements, and this is simply a matter of referring to a map of characteristic frequency and distance from the apex. The well-known frequency–place map was first described in detail by Greenwood (1961), and has come to be standard in the field.

Frequency–place map

The frequency–place map tells us at what point in the human cochlea, measured in millimetres from one end (either base or apex), the maximum sensitivity to a particular frequency (the characteristic frequency) occurs. This map of frequency (in Hz) versus distance (in millimetres) is well studied (LePage, 2003), and a widely accepted mapping is that due to Greenwood (1961), which is expressed as:

$$\text{distance} = 16.7 \log_{10}(0.006046f + 1) \quad (3)$$

This logarithmic mapping of frequency to place is shown in Figure 3.

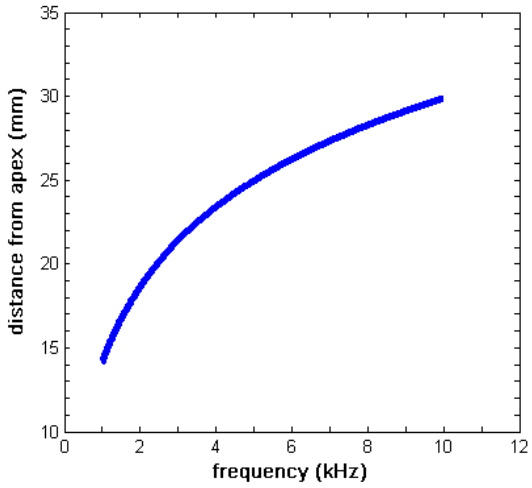


Figure 3. The Greenwood frequency–place map. The human cochlea is 35 mm long, and over this distance it is tuned from 20 Hz to 20 kHz. The range 1 to 10 kHz covers a distance of 14 mm to 30 mm from the apex.

It is now a simple matter of combining the information gathered so far to express the delay as a function of distance (Figure 4).

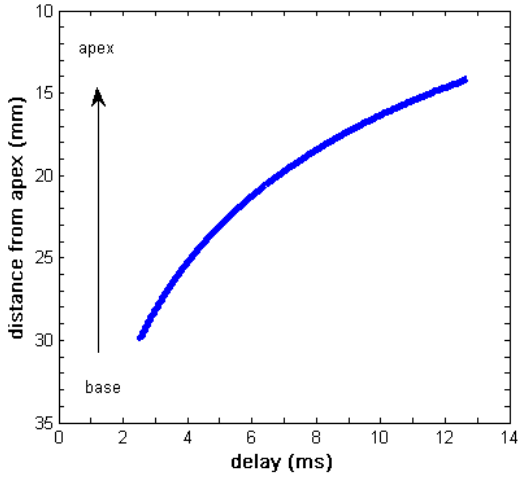


Figure 4. Cochlear delay as a function of distance. The y-axis is inverted so as to more easily appreciate that the wave is progressing (delay is increasing) from base to apex.

While Figure 4 conveys the important fact that the wave is progressing from base to apex, it also shows that the *slope* of the curve (distance per unit time, which is velocity) decreases from base to apex, meaning that the wave is slowing down. This is a characteristic feature of the classical travelling wave (de Boer and Nuttall, 2010). It is a straightforward matter to calculate the slope of the curve and derive a graph of velocity versus distance from the base (Figure 5).

Figure 5 shows the important result that the wave velocity begins at more than 4 m/s at the basal end and slows down exponentially to below 1 m/s at the apex. This curve bears a strong resemblance, both in shape and magnitude, to actual experimental travelling wave velocity curves. One of these, from Donaldson and Ruth (1993), was derived by measuring the latencies of auditory brain stem responses to different frequency bands. Some 24 subjects were measured, and the corresponding travelling wave delays and velocities were computed (Figure 6).

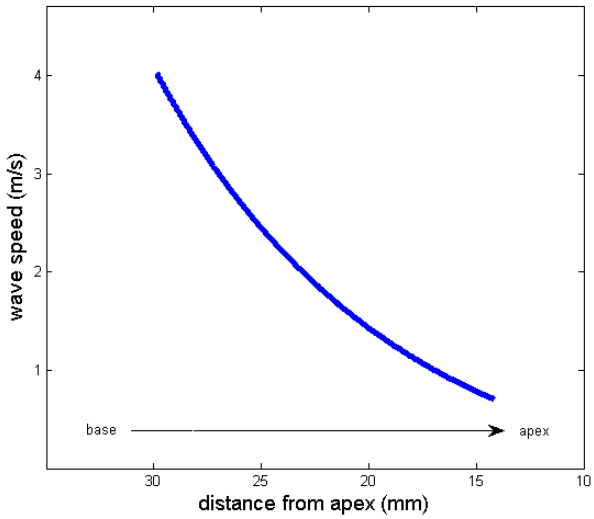
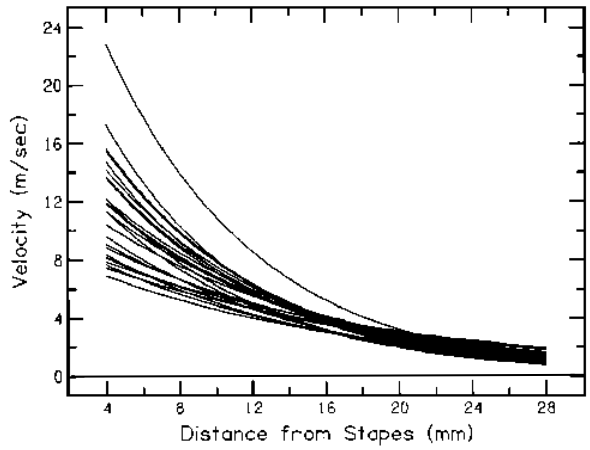


Figure 5. Apparent wave velocity along the cochlea in response to a simultaneous excitation of a bank of graded resonant elements. The wave starts at a speed greater than 4 m/s at the basal (high frequency) end and slows to less than 1 m/s at the apical (low frequency) end. These are typical travelling wave velocities.



Source: (Donaldson and Ruth, 1993), with permission of ASA

Figure 6. Experimentally measured travelling wave velocity in 24 human subjects. Delays in auditory brainstem responses were converted to velocity curves for each individual.

DISCUSSION

This work has shown a marked similarity between the apparent wave velocity calculated for a graded bank of oscillators (of specified Q) which are simultaneously excited, and the experimentally determined travelling wave velocity for humans.

Given the simplicity of the assumptions and their clear resonance foundation, this result is important. It has long been said that the observed cochlear delays, and associated wave velocities, were evidence in favour of the travelling wave theory and against the resonance theory. For example, it has been held that the delays available from resonance could only be less than half a cycle (deBoer and Nuttall, 2010, p.145). But this interpretation rests on assuming a completely passive system with low Q. However, we now know that the cochlea is active, and has Q values between 10 and 30. This expands the range of interpretation considerably.

Other investigators (Bergevin and Shera, 2010; Ruggero and Temchin, 2007; Meenderink and Narins, 2006) have noted similarities between the "travelling wave" delays in the hearing organs of humans and of other tetrapods, like frogs and lizards, that do not have anything like a travelling wave in their ears (frogs do not even have a basilar membrane).

Bergevin and Shera (2010) remark how the responses of lizard ears are "strikingly reminiscent" of those in mammals, despite major differences in inner ear morphology and function and that lizards "evidently lack traveling waves". They model the gecko ear as an array of coupled harmonic oscillators and find that the Q of the oscillators governs the build up time of the stimulus frequency otoacoustic emissions. The authors find that the mathematics of the lizard system is virtually the same as that of the mammalian one, but they leave that as a curiosity, maintaining that the two underlying mechanisms are fundamentally different. Yet, the diagram they offer of the lizard ear's anatomy suggests the system is set up to directly intercept a pressure wave.

In their studies of a frog, Meenderink and Narins (2006) found that stimulus frequency otoacoustic emission (OAE) delays had similar properties to human distortion product OAEs. Unwilling to adopt a resonance picture, people have suggested a travelling wave in the tectorial membrane, although Meenderink and Narins do suggest that simultaneous excitation of the sensory cells may occur.

Ruggero and Temchin (2007) made a comparison of human and non-mammalian animals, and found that response delays were similar in both cases. They are inclined to view the human case as one in which ripples convey energy along the basilar membrane, exciting the cells, and so they try to interpret the frog and lizard work similarly, again suggesting ripples in the tectorial membrane.

In the end, the issue often becomes a semantic one, because by travelling wave people mean different things. Some think of it as a hydromechanically coupled ripple on the basilar membrane, a causal entity that can deflect stereocilia, whereas others use it loosely to mean any moving wave front, however caused (although the resonance implications are rarely taken further). Here, I use the term 'ripples' to mean the hydromechanical wave generated in the way conventional travelling wave theory prescribes. For the wave generated by resonance, I use the term apparent travelling wave.

Ruggero and Temchin's analysis is not helped by them comparing post-mortem studies with live ones, and by equating signal-front delay to basilar membrane travel time (p. 154).

Only by specifying the effective Q of a system (and its filter delay or build-up time) and clearly distingishing the causal factors at work can we begin to disentangle what different authors mean. Unfortunately, the mathematics is often blind to the actual mechanisms underlying cochlea excitation.

Travelling wave and resonance

Békésy himself made an attempt (with Wever and Lawrence) to try to clarify what is meant by a travelling wave, saying that "nothing is implied about the underlying causes" (whether it be a ripple along the membrane or a stimulus conveyed through the cochlear fluids). This declaration of agnosticism was prompted by Wever and Lawrence's disbelief in a basilar membrane mechanism (they thought the signal reached the sensing cells through the fluids), but at the time (1954) there was no notion of an active cochlea and the idea that the organ could sustain a Q higher than 1 was met with disdain. The result was the article had no impact, and people continued to believe in a hydrodynamic travelling wave. A notable exception was Gold, who even in 1948 presciently described how an active cochlea with positive feedback could simultaneously overcome viscosity limitations and attain sharp tuning (Gold, 1948). But though he personally tried to convince Békésy that a travelling wave was too broad to allow sharp pitch discrimination, he was too far ahead of the times.

In another effort to clarify the semantics, which has caused so much trouble over the years, it is worth once again returning to Békésy's insightful distinction between his travelling wave theory and the resonance theory. He drew the analogy of a set of pendulums, of graded length, hanging on a rod, and this is illustrated in Figure 7.

In part A of Figure 7, the pendulums are connected with a rubber band, and the shortest pendulum is excited with a displacement. Because of the rubber coupling, the shortest pendulum excites each of its neighbours in turn, and a wave of excitation travels along the set. *The travelling wave carries energy*.

By way of contrast, in part B of Figure 7, the same pendulums have no coupling. In this case, to excite the pendulums, the rod is given a sharp twist, which displaces all of the pendulums simultaneously. Again, a wave begins to propagate along the set, but in this case *the wave carries no energy*.

Békésy actually built a set of pendulums and performed the two experiments, but again part A has dominated the field and part B has been virtually forgotten. The analogy to the cochlea is apt, for in one case the stimulus is a propagating ripple, and in the other it is a fast compression wave. I think the best distinction between the two situations is to say that in one case the stimulus is *in series* with the bank of tuned elements, and in the other it is *in parallel*.

The active cochlea

The finding of a cochlear echo (Kemp, 1979) created a revolution in our understanding of the inner ear, providing unmistakeable proof that the organ is an active device, not a passive one, and forcing us to reconsider all previous cochlear theory. All options were then back on the drawing board, including those of Helmholtz (1875). Kemp himself has tried to build the active cochlea on top of travelling wave theory, and has assumed that all the observed otoacoustic delays are travelling wave delays (Kemp, 2010). The standard theory of the active cochlea, that of coherent reflection filtering due to Zweig and Shera, assumes that the travelling wave delay between stapes and characteristic frequency on the basilar

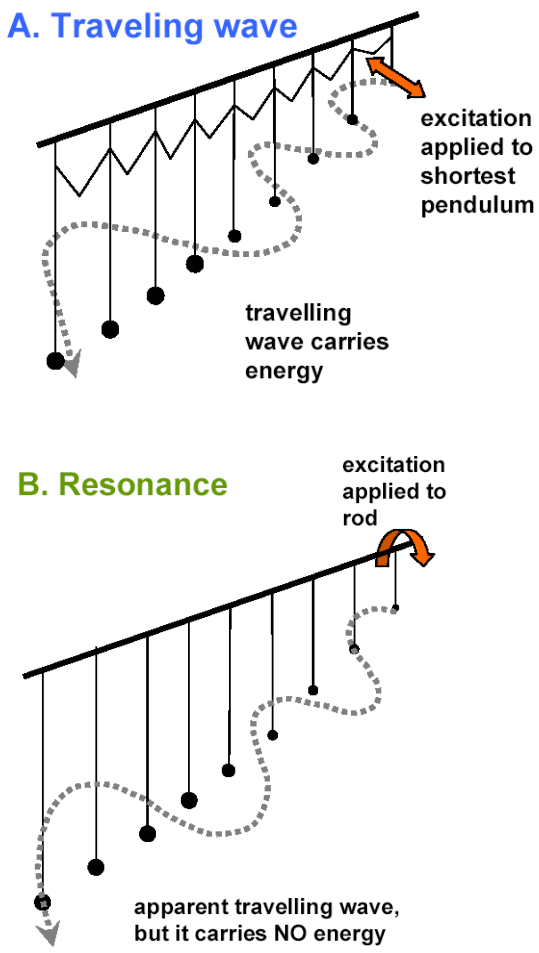


Figure 7. Békésy's pendulum analogy to illustrate the difference between a travelling wave and resonance. In a travelling wave (**A**), the excitation is applied to the shortest pendulums and the energy moves to neighbouring longer ones by rubber bands that supply coupling. In a resonant system (**B**), the pendulums hang from a common rod and are simultaneously excited by a short twist to the rod. In both cases a wave-like motion of the pendulums is seen.

membrane can be recycled multiple times, giving rise to long delays (Zweig and Shera, 1995; Shera, 2003).

It is notable that Shera and Guinan (2003, Fig. 4) and Shera et al. (2007) find that the Q of the cochlea can be expressed, in at least 4 species (including humans above 1 kHz and other species above 3 or 4 kHz), as

$$Q = N_{SFOAE} \quad (4)$$

meaning that the Q of the cochlea is equal to the number of cycles of signal required to build up a stimulus frequency otoacoustic emissions (SFOAE), just as expected for a driven resonator. Identifying the elements that are resonating is the foundation for understanding what is going on here. In the Bergevin and Shera paper discussed earlier, for example, the presumed elements were taken to be rotating bundles, but this remains uncertain (their model requires rotation to cause bundle deflection, but for small angles the displacement component, sin θ, will vanish, leaving only a vertical component that will not displace the bundles). The alternative, mentioned above, is that the arrangement of the lizard's ear seems well suited to intercepting sound *pressure*, and this mecha-

nism has been put forward for stimulating the human cochlea (Bell, 2007a).

The mammalian cochlea is immersed in watery fluid, a medium whose characteristic impedance is such that, for a given sound power, the pressure component is 1600 times greater (and the displacement 1600 times less) than it is in air. Physically, therefore, detecting the pressure component of the sound in a fluid is a much easier task than detecting the displacement component (Bell, 2005). Again, a measure of speculation is required, but if it is true then the resonating element can be identified as a single, place-specific 'string' on the basilar membrane. In both cases, it follows that the Q of these resonant structures will relate directly to their build up time, and the remarkable similarity between human and lizard ears can be understood.

A SAW model and its resonant elements

At this point it seems apt, and will aid understanding, to describe the cochlea model motivating this paper, a model in which high Q values can be attained at each individual point on the basilar membrane. The model also has the virtue of having its resonant elements able to be simultaneously stimulated by compression waves as they sweep through the cochlear fluids.

In this connection, it is noteworthy that the outer hair cells are in direct hydraulic connection with all of the cochlear fluids, and that a number of inexplicably fast cochlear phenomena have been reported in the literature (Bell, 2005).

The model views the three rows of outer hair cells as forming a surface acoustic wave (SAW) resonator (Figure 8), a topology in which each triplet of OHCs, carrying both sensors (stereocilia) and motors (cell bodies), is involved in a positive feedback loop (Bell, 2006, 2007b). Following Gold's ideas, the feedback provides the sharp tuning and overcomes viscosity. Moreover, the feedback is mediated by a so-called "squirting wave" in the gap between the tectorial membrane and the organ of Corti (Bell and Fletcher, 2004), and the dispersive properties of this wave (velocity inversely proportional to the cube of frequency) mean that the distance between the rows can, based on actual cochlear dimensions, tune the system from 20 Hz to 20 kHz.

A persistent difficulty with the travelling wave theory is that it is so difficult to tune over the entire auditory band, and most modellers don't even try, restricting themselves to a much lesser range. The real difficulty is that the mass and compliance of the basilar membrane are called upon to vary by a factor of 10^6 in order to vary the frequency over 3 decades. Mass is reasonably constant, so getting the basilar membrane stiffness to vary by hundreds of thousands means its Youngs modulus is called on to vary by more than that between foam rubber and tungsten. Attempts have been made to increase the mass term, but without much effect. A range of other drawbacks of the travelling wave model, including the difficulty of how an active process can manage to sustain a ripple from base to apex without succumbing to the limitations of noise (a problem highlighted by Gold), have been catalogued in Bell (2005).

With the SAW model, the tuning derives from a standing wave between the rows of OHCs, and the output of this tuned system is an oscillating parcel of fluid that stimulates the inner hair cells, which in turn send a signal to the brain. In effect, the OHCs are the preamplifiers (or regenerative receivers) in the system, and they constitute what has been called the "cochlear amplifier" (Davis, 1983).

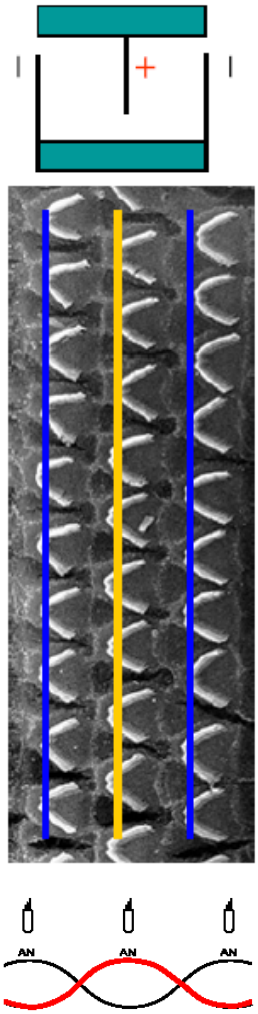


Figure 8. A surface acoustic wave (SAW) model of the cochlea in which the three rows of outer hair cells (with V-shaped stereocilia) exchange wavefronts and form a standing wave. Top shows a schematic of a SAW resonator with electrode fingers of opposite polarity; middle shows a top view of the sensing surface of the cochlea with its three rows of sensing cells; bottom shows the standing wave with 3 antinodes (AN) and two nodes, similar to a xylophone bar.

The SAW model calls for low velocity squirting waves to reverberate backwards and forwards between the rows of outer hair cells, in this way creating a standing wave and cycle-by-cycle gain. There is one wavelength between the first and third rows, forming a resonant structure like a guitar string or, more precisely, a xylophone bar. The three OHCs sit at three antinodes, with nodes inbetween, just like in a xylophone. The difference is that in the cochlea case, the wave motion is not bending waves in a metal bar, but squirting waves which represent the mass of fluid in the subtectorial gap resonating with the compliance of the reticula lamina, aptly called the plateau of Corti. The result is a resonance pumped by the motile activity of the OHCs.

Because the OHCs are presumed to contain a compressible element (Bell, 2008), the SAW system is sensitive to compression waves, and this is how each triplet of cells is responsive to a sound wave entering the cochlear fluids through the stapes. The eardrum and middle ear vibrate the stapes in the oval window, which pushes and pulls on the incompressible cochlear fluids and causes a fluctuating hydraulic pressure (a pressure wave). Because the cochlea is encased in hard bone, the sound energy is conveyed directly to the compressible OHCs.

How the SAW model specifies *Q*

Tuning in the SAW model relies largely on the distance between the three rows of OHCs, just like the tuning of a xylophone depends on the length of the bar.

The twist to this story is that the OHCs do not lie in a strict rectangular pattern; instead there is a slight tilt to the array of about 4º (Bell and Maddess, 2009), so that the box-like pattern ends up in a parallelogram (Figure 9). The tilted box has important ramifications for how the the resonance activity behaves. OHCs do not carry a single line of stereocilia; instead the stereocilia appear in a V-shaped tuft (Figure 8). Importantly, each of these two arms faces in a direction that supports wave interaction along the two diagonals of the parallelogram (Figure 9). The tilt means that one diagonal is slightly longer than the other, and this in turn means that every OHC experiences the effects of two somewhat different frequencies (that is, every OHC is connected to a long and a short reverberating path, see Figure 9).

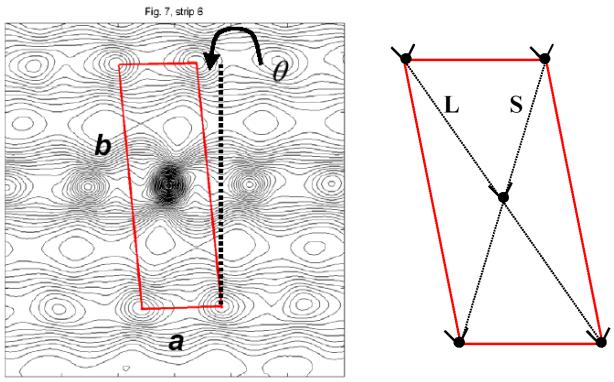
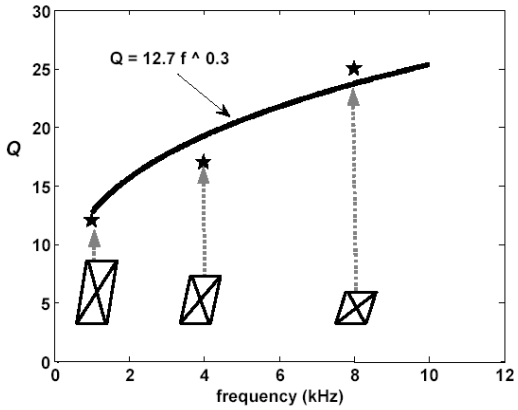


Figure 9. Tilt of the outer hair cell lattice. *Left*: Spatial autocorrelation analysis of the hair cell pattern in a monkey shows that the tilt of the lattice is about 4º. Dimension *a* is along the cochlea, *b* is the distance between the inner and outer rows (1 wavelength). *Right*: The tilt causes the diagonals to have different lengths (*L*, long; *S*, short).

Although a 4º tilt is not much, its effect is magnified because of the high dispersion of squirting waves: frequency is inversely proportional to the *cube* of the frequency. For example, taking a point on the cochlea near 1 kHz and referring to Figure 9, the *b*/*a* ratio here is about 3, so that a 4º tilt in the lattice translates to a ratio of diagonal lengths (*L*/*S*) of about 1.026. However, the ratio of frequencies supported by those lengths will be $(1.026)^3$, which is about 1.08. Put another way, the two frequencies will differ by 0.08, which in turn means a *Q* value of 1/0.08 = 12. In summary, we have established that the *Q* of the cochlea is set by the fixed geometry of the OHCs. Because every OHC sees itself subjected to two frequencies, the unit cell will appear to possess a certain *Q*, and this property is naturally reflected in the measured *Q* of the cochlea (otoacoustically and psychophysically).

Proceeding in the same way for other frequencies, Bell and Maddess (2009) calculate the effective *Q* values for other regions of the cochlea based on their unit cell geometry and find that the calculated values correspond well with Equation 1, and this is shown in Figure 10.

This concordance makes it straightforward to interpret the measured *Q* values of the cochlea as reflecting the inbuilt geometrical properties of the sensing surface. In brief, geometry determines *Q*, and in turn the spatial gradient of *Q* values determines the speed of an apparent travelling wave running along the system.



Source: (Bell, 2008) with permission

Figure 10. *Q* values (stars), calculated on the basis of actual measurements of the primate cochlea's tilted unit cell (shown schematically at the bottom), match the *Q* values (curve) derived by Shera et al. (2002) using psychophysical and otoacoustic measurements.

AN EXPLANATION FOR A PUZZLE: THE SAW MODEL AND COCHLEAR EMISSIONS

A remarkable thing happens when a sound is projected into the cochlea: after an appreciable time – typically 8 ms for a low-level 1 kHz tone, and shorter for higher frequencies (Kemp, 2010) – an echo returns to the ear canal. This echo, called an otoacoustic emission, can occur in response to a tone, a click, or as the distortion product of two tones (most clearly 2f1–f2 in response to f1 and f2).

In the case of the distortion product OAE, the response delay (at 2f1–f2) is difficult to measure but according to Konrad-Martin and Keefe (2005) it is about twice, but usually a bit less, that of each of the primary tones (f1 and f2), and this is curious. The traditional explanation is that the extra delay is the time it takes for a travelling wave to propagate from the overlapping region of f1 and f2 towards the apex, where 2f1–f2 has its characteristic frequency. But why should the presumed echo appear somewhat sooner than double the "one way" travel time?

Although other explanations have been presented, here another explanation, based on the simple understanding set out above, is offered. The explanation unifies the resonance picture I have constructed, but it also makes the theory amenable to experimental test. It is therefore adds a useful dimension to this paper.

Figure 11 illustrate how the extra delay in the DPOAE originates. It shows three resonators (white) placed upon the basilar membrane (BM, brown) and surrounded by fluid (blue). When two sounds (top arrows) enter the cochlea at 1000 Hz and 1100 Hz as pressure waves (period ≈ 1 ms), they excite the corresponding basilar membrane resonators (red, orange). For simplicity we assume a *Q* of 5, which means their amplitude peaks after 5 cycles or 5 ms (the waveform is shown on a superimposed downwards time axis).

Since these resonators are pressure sensors, this means, by reciprocity, that they also *produce* additional pressure in the cochlea as the cochlear amplifier performs its job. The pressure fluctuations created by these active and compressible sensors/motors, which are immersed in incompressible fluid,

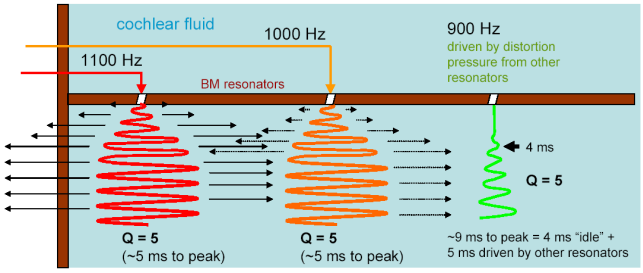


Figure 11. A model for how distortion product emissions appear from an idealised cochlea with a delay nearly double that of their primaries. A downwards time axis is added to the spatial layout of the basilar membrane. Thin black arrows represent fast pressure waves, which leave the cochlea as otoacoustic emissions, but also impinge on other pressure-sensitive resonators (for simplicity, these arrows are omitted from the 900 Hz resonator).

are shown by the thin black arrows, and this sound pressure passes to the ear canal (left) where they can be detected by a microphone as otoacoustic emissions.

The pressure fluctuations also impinge on all the other pressure-sensitive resonators, and one in particular, at 900 Hz (green), will respond to the lower distortion product (2f1–f2) of the pressure field being generated by the 1000 Hz and 1100 Hz resonators. It too, has a Q of 5 and takes 5 cycles (5 ms) to reach its peak. However, the 900 Hz resonator only starts resonating after 4–5 ms when the primary resonators have become fully active. Having a Q of 5, the 900 Hz oscillator therefore reaches a peak after 9–10 ms.

The feature to note is the double resonator chain. The 900 Hz resonator only begins to receive an appreciable stimulus once the other two resonators have approached their peaks. This means that there is a two-fold delay, once for the primary resonators and again for the distortion product resonator. Of course, the second resonator does not have double the time delay: it will be somewhat less than 2× in that it is given a bit of a 'head start' – the effective starting point is 1 or 2 ms before the other resonators reach their maximum amplitude, Perhaps for 3 or 4 ms, while the other resonators are building up to appreciable amplitude, the distortion product oscillator feels no effective pressure stimulus. For illustrative purposes, this effective starting point is shown as a 4 ms 'idle time' in Figure 10, and gives rise to an overall delay factor of 1.8×.

This analysis is simplified to illustrate the mechanism, and actual DPOAE measurements are complex, making it hard to give concrete figures. Konrad-Martin and Keefe (2005) were able to separate out multiple factors by measuring both SFOAEs and DPOAEs, and used gated stimuli and windowing analysis to separate the multiple components. They measured the latency of the primary resonators (red and orange in Figure 11) as about 4 ms at 2.7 kHz (their Fig. 2, using SFOAE methods) and the latency of the distortion product resonator (here in green) as 7 ms (their Fig. 7B), which demonstrates the 1.8 ratio.

Incidentally, they also found a distortion component originating with a shorter latency (3 ms), supporting the idea that distortion can originate from two sources, the primary oscillators and the secondary one. Normally when measurements are undertaken these two sources are blurred. Of even more interest, they found many instances of delays close to 0 ms, which they acknowledge may be due to "a reverse transmission path… through the cochlear fluid" (i.e., a pressure wave).

CONCLUSION

We have examined the response of a graded bank of independent resonating elements when simultaneously excited, and they have been found to give rise to an apparent travelling wave. The envelope of the maximum excursion of the individual elements varies with time as the oscillation of each element builds up and decays, governed simply by its Q, and the speed of this apparent wave has been found to be in the range 1 to 4 m/s over a large length of the cochlea, values directly comparable to travelling wave velocities.

We take this as an indication that the cochlea may well be operating on resonance principles, and that the associated "travelling wave" could be just a side-effect, an epiphenomenon, arising from that process. There need be no coupling between the elements, no serial excitation of the sensing cells, and no energy carried by the wave. Instead, we can assume that a fast compression wave could propagate through the cochlear fluids, exciting all the sensors – the pressure-senstive outer hair cells – virtually instantaneously.

Encouraged by this demonstration, an active, resonant model of the cochlea is proposed, one that pictures reverberation of waves between the parallel rows of outer hair cells, a biological form of the familiar surface acoustic wave (SAW) resonator. The waves are low velocity, high dispersion squirting waves which allow the system to be tuned from 20 Hz to 20 kHz using realistic parameters.

The model can explain how the Q of the resonating elements arises using simple considerations of the geometry of the OHC unit cell. In essence, the tilt of the OHC lattice broadens the allowable range of feedback frequencies that can reverberate between the rows, and in this way effects a certain Q. (If there were no tilt, reverberation between the parallel rows would continually build up cycle by cycle (with positive feedback gain) until an oscillation with effectively zero bandwidth (infinite Q) would be produced, which would not be useful in a cochlea context).

This work has sketched how cochlear mechanics can be explained in resonance terms. The OHC lattice geometry determines the Q, and the Q in turn determines the travelling wave velocity. No free parameters are used. The travelling wave is considered an epiphenomenon and is not needed as a causal mechanism to excite the cochlea's sensing elements.

"The resonance theory of Helmholtz is probably the most elegant of all theories of hearing", said Békésy (1960), and I agree.

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