

# Amplification in the Inner Ear: Bekesy's Paradox

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## Abstract:

This work provides a critical analysis of the amplification of quiet sounds according to the traveling wave theory. He points out the flaws and inconsistencies of Bekesy's theory with current knowledge. He presents his own modern sub molecular theory of hearing, which includes a new perspective on the amplification of quiet sounds at the molecular, intracellular level

**Keywords:** Bekesy's theory; Inner ear; Gambin

## Introduction

Hearing research by eminent professors of otolaryngology 100 years ago led to the conclusion that soft sounds on their way to the brain are amplified in some unknown way. In 1930, Wever and Bray published a study known as the Wever-Bray effect: "The action potential of the auditory nerve, generated in the cochlea, reflects the frequency of the sound acting on the ear." It has been confirmed that the inner ear converts the mechanical energy of sound waves into electrical signals in the auditory nerve, proportional to the energy of the sound waves [1]. Hallowell Davis (1896-1982) conducted pioneering research on the brain's electrical activity (a pioneer in EEG research) as well as on the electrical responses of the auditory nerve. He studied the auditory nerve impulses transmitted to the brain. He proposed a model of cochlear mechanics as two systems operating simultaneously [2]. The first system stimulates the OHC at sound levels above 40 dB. The second system is an active system, responsible for the reception and amplification of tones up to 60 dB (!). He observed a lack of correspondence between the amplitudes of auditory nerve potentials and the intensity of sound waves. He stated 100 years ago that "something provides new energy that amplifies the vibrations of the basilar membrane at a specific location. This mechanism depends on the OHC." Prof. Davis was a proponent of Helmholtz's theory, but his research was not incorporated into Bekesy's theory, because the observed mechanism of signal amplification in the inner ear was not explained. That didn't fit with Bekesy's theory. Bekesy did not acknowledge his competitor's pioneering research. Actin and myosin were discovered in the 1940s. Discovered in 1929, ATP laid the groundwork for the development of a new theory of signal amplification in the inner ear in later years. An OHC contraction, operating on the same principle as muscle contraction, causes increased movement of the basilar membrane, triggered by fluid flow in the inner ear. Increased maximum deflections of the basilar membrane in response to soft sounds stimulate IHC and transmit information to the brain. This leads to an amplified response of the inner ear to soft sounds. The energy for this amplification was supposed to come from ATP. Further research has shown that this mechanism is too slow to amplify high frequencies. Contractions of the OHC were observed during changes in electrolyte levels, primarily Ca<sup>++</sup> and K<sup>+</sup> ions; OHC contractions

also occurred in the absence of ATP. Ashmore presented a new version of signal amplification. A change in cell membrane potential causes a change in the electric field, which, according to Ashmore, leads to certain shifts in electrical charges in the cell membrane.

It was assumed that a kind of “molecular engine” was being formed. It is a macromolecule in the cell membrane in which shifts in charge cause conformational changes, leading to a change in the cell’s surface area by a few percent. In 1985, Brownell published research confirming OHC shortening by up to 5% of its length during cell depolarization [3]. Repolarization and hyperpolarization cause OHC lengthening. The molecular mechanism underlying intracellular amplification—as seen in other sensory organs — remained unknown.

The 1991 Nobel Prize in Physiology awarded to Erwin Neher and Bert Sakmann, and the 2003 Nobel Prize in Physics awarded to Roderick MacKinnon and Peter Agre, for explaining the function of ion channels in cells, brought audiologists closer to understanding the mechanisms involved in hearing. Ion channels play a very important role in hearing. In 2000, a study by Zheng and J. Hudspeth was published on prestin, a membrane protein of the outer hair cell, which is believed to be responsible for the contractions of the hair cell. It was accepted that prestin does not use ATP energy. The ability to perform work consisting of OHC contractions and driving the basilar membrane into vibration, effectively pulling it in accordance with frequency, requires a large amount of variable energy. This energy is believed to come from the electrochemical energy of the cell membrane, which varies during each OHC contraction. The pulling of the basilar membrane is thought to amplify soft sounds by 40–50 dB [4]. The problem with amplifying soft sounds is that this theory is based on Bekesy’s 100-year-old traveling-wave theory. Bekesy’s theory of hearing, based on Helmholtz’s theory from 1859, wave resonance, cochlear fluid flows encoding information transmitted to the receptor, and the recognition of the basilar membrane as the most important part of the inner ear, contains many ambiguities and even contradictions with the laws of physics. The adequate stimulus for the auditory receptor is the energy of a sound wave. In Bekesy’s theory, fluid flows and the basilar membrane act on the receptor through a tip-link mechanism. The signal’s path to the receptor is very long and involves multiple energy conversions, which increases the likelihood of errors in the transmission of information. In the new “submolecular theory of hearing,” the energy of the sound wave acts directly on the auditory receptor, without the mediation of the basilar membrane. The signal path to the receptor is simple, short, and fast. The receptor potential is generated within 1.5 ms – 1.8 ms. In Bekesy’s theory, this is impossible.

## Problems with Bekesy’s Theory

1. Bekesy assumed that the energy of the sound wave in the tympanic cavity is amplified, increasing 20 times, regardless of intensity, compared to the energy of the wave striking the tympanic membrane. An increase of 33 dB, as reported by other sources, corresponds to an amplification in the middle ear of 44 times. Laser Doppler vibrometry studies do not confirm this. A 90 dB sound wave = 500 nm in the external auditory canal has an amplitude of 11.7 nm at the stapes plate [5,6]. Calculations based on Bekesy’s methodology, performed by a professor at the University of Technology for a 10 dB tone at 10 kHz, showed an amplitude on the stapes plate of 0.000011757 nm (W. Gambin). We hear a tone of 10 dB and 10 kHz, which indicates that the calculations according to Bekesy’s model are incorrect.

2. The theory is based on the hydrodynamics of cochlear fluids. Fluid flows cannot simultaneously encode amplitude, frequency, phase shifts, harmonic components, length of sound, accent, and melody. These values are encoded by the sound wave traveling to the receptor. The cochlear fluid is the medium through which the sound wave travels. Only the particles of the medium move in accordance with the wave’s amplitude, from the central position forward in the direction of the wave and back again, in accordance with the frequency and amplitude of the sound wave. This is the displacement of sound pressure in fluid, just as it does in solids.

3. A challenging aspect of Bekesy’s theory is the resonance of the longitudinal wave in the cochlear fluid with the transverse wave of the basilar membrane’s natural vibrations. This resonance does not provide a complete transmission of auditory information. The force vectors of these waves act in perpendicular planes. The natural vibrations of the basilar membrane are incorrectly calculated. The basilar membrane, 0.1 mm wide at its base, cannot separate the 4.3 mm cochlear canals containing different fluid concentrations. The basilar membrane, which is connected to the organ of Corti, cannot vibrate on its own. It vibrates in unison with the massive organ of Corti, the hair cells and receptors, and the fluid adjacent to the basilar membrane. The entire vibrating assembly has high inertia. The fluid has excellent damping properties. Mammals that can hear up to 100 kHz are unable to resonate with the basilar membrane. We can hear near-threshold tones when the amplitude of the driving wave, in the range of 0.01 nm to 0.05 nm, has energy lower than the energy of the damping of the driven wave. In this configuration, resonance does not occur. We hear because the signal travels to the receptor via a different path, through bone, directly to the receptor of the hair cells [7].

4. The speed of a sound wave in fluid is 1450 m/s. The traveling wave speed on the basilar membrane

(according to Bekesy) is 50 m/s near the base of the cochlea and 2.9 ms near the cap. A speed difference ranging from 29 to 500 times makes it extremely difficult to transmit information. It is difficult to imagine cochlear fluid flows encoding such highly compressed information, which, in the case of soft sounds, is supposed to be amplified. A polytone with a series of harmonic components causes a series of maximum deflections on the basilar membrane, each at a different location and time. The maximum deflections are intended to produce a series of fluid wave streams that encode information and transmit it to the tip-link mechanism. This information encoding is thought to be maintained by cadherin filaments, which trigger the molecular mechanism responsible for the gating of mechano-dependent potassium ion channels in the hair cell.

5. Bekesy found that the pressure in the vestibular duct is 20 dB higher than the pressure in the tympanic duct. He also stated that traveling wave deflects maximally toward higher pressure, contrary to the laws of physics. The organ of Corti is attached to the basilar membrane; there is no free space between the basilar membrane and the organ of Corti. How did Bekesy observe this wave traveling on the basilar membrane? Was it through the organ of Corti?

6. Vibrometric studies have shown that the amplitude of a sound wave of 500 nm = 90 dB in the external auditory canal has an amplitude of 0.5 nm at the oval window. According to Bekesy's theory, the path of this signal is halfway to the receptor. We can hear (at the threshold of hearing) an amplitude of 0.01 nm at the input. This wave reaches the receptor via the cochlear fluids at approximately 0.0002 nm. It is impossible for a fluid flow with an amplitude a million times smaller than the diameter of a hair cell hair to bend or deflect the hair if the hair is embedded in the tectorial membrane. A wave that is several hundred times smaller than the diameter of the atoms of the basilar membrane cannot generate a traveling wave on that membrane. It is too small to generate fluid flows in the cochlea. Such a wave is incapable of transmitting all the information contained in a sound wave. We still hear this wave as very quiet – after amplification, according to mechanical theory, by 40-50 dB.

7. According to the theory, OHC contraction occurs after the depolarization of this cell. The ion channels of the cell wall and the conductivity of the cell membrane for Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>++</sup> and Cl<sup>-</sup> ions, as well as ion pumps and exchange transport mechanisms are responsible for the depolarization of the cell consistent with the information contained in the sound wave, initiated by the influx of K<sup>+</sup> ions into the cell through mechano-dependent channels. Ion channels have a limited operating cycle time of a few milliseconds. During the repolarization phase, voltage-dependent channels are inactivated and insensitive to stimulation by a new impulse. This is the

refractory period. During this time, the permeability to K<sup>+</sup> ions increases. The time it takes for the cell to return to an excitable state is several milliseconds [8]. Therefore, simultaneous depolarization of the OHC and simultaneous contraction of the entire cell up to 100 kHz is impossible. It is possible to consider the existence of a limited depolarization and a limited contraction of the cell. This rules out the amplification of soft sounds caused by the contracting OHCs pulling on the basilar membrane [9].

8. According to Bekesy's theory, the signal's path to the receptor is long, time-consuming, requires successive energy conversions, is energy-intensive, and leaves room for errors in information transmission. It is important to examine the signal travel time to the receptor in the case of loud and soft tones that require amplification [10].

9. In wave motion, there is velocity, acceleration, and mass. The inertia in the middle and inner ear must be calculated. This is particularly important when it is assumed that information is transmitted by the flow of cochlear fluids. In the middle ear, the auditory ossicles and the tympanic membrane undergo vibration. In the inner ear, the basilar membrane vibrates in unison with the organ of Corti and the fluids of the cochlea. The inertia in wave motion is directly proportional to the vibrating mass and to the amplitude of the wave, and is proportional to the square of the frequency. A sound wave has no mass and is not subject to inertia [11].

10. This point is important in explaining the lack of high-frequency conduction following stapedotomy.

11. In the case of a quiet sound received by a receptor and amplified by pulling on the basilar membrane, a receptor potential and an action potential are generated in approx. 1.5 ms. After the signal is amplified, a new, larger receptor potential and action potential must be generated within a few tenths of a millisecond. Do electrophysiological studies confirm that every quiet sound received by the receptor is transmitted twice to the brain?

## Bekesy's Paradox

Vibrometric studies have shown that the amplitude of a 90 dB, 1000 Hz sound wave decreases by a factor of 1000 from the external auditory canal to the oval window [5,6]. The greatest reduction in amplitude occurs in the vestibular canal, which is spirally coiled and narrows threefold from the oval window to the cap. Absorption damping, reflection damping, and interference damping occur here. Additionally, wave dispersion occurs in the cochlear fluid. The closer to the cap, the more the wave amplitude decreases. The loss of

wave energy is proportional to the square of the loss in wave amplitude. The amplitude of a sound wave in the middle ear between the external auditory canal and the stapes plate is reduced 50-fold. For a 90 dB wave, the amplitude decreases from 500 nm at the input to 11.7 nm at the stapes plate (Gambin, Presentations, Kraków 2018). Near the cap, the amplitude of this wave is about 1 nm — it decreases approximately 500-fold. The amplitude of a sound wave from the stapes plate to the cap decreases 42.73-fold for a 90 dB wave. Other sound wave intensities were not studied. At the beginning of the vestibular duct, the amplitude of the wave is similar to that of the stapes plate and then rapidly decreases toward the cap. If the amplitude of the wave on the stapes plate is reduced 50 times compared to the amplitude of the wave at the input, then the energy of that wave is reduced 2,500 times. The amplitude of the sound wave decreases by about 500 times near the cap. The energy of this wave decreases 250,000 times. According to Bekesy's theory, the resonance of a sound wave with the natural vibrations of the basilar membrane determines the location on the basilar membrane where specific frequencies are received.

Bekesy's paradox lies in making the perception of sound wave frequencies dependent on the structure of the basilar membrane. After stapedotomy, there is no perception of high frequencies, which are received near the oval window, where the energy loss of the wave is small. However, contrary to Bekesy's theory and the results of studies, after stapedotomy, low frequencies are received near the cap, when the energy loss for the lowest frequencies is approximately 1,826 times greater than the energy loss of high-frequency waves in the area of the oval window — which are not received. For the high-frequency signal path to the receptor, as determined by Bekesy and previously by Helmholtz — through the cochlear fluids and basilar membrane, the wave amplitude at the stapes plate was calculated for a 90 dB tone and frequencies of 1,000 Hz and 10,000 Hz. For a frequency of 1000 Hz, this is 11.17 nm. For 10,000 Hz, this is 0.11757 nm. The frequency increases 10 times — the wave amplitude decreases 100 times (Gambin).

Humans can hear sounds up to 20 kHz; other mammals can hear up to 100 kHz. There must be another mechanism for detecting high frequencies that is not related to wave resonance, cochlear fluid flows, or the structure of the basilar membrane. Inertia in wave motion plays an important role in hearing. It is assumed that a sound wave has no mass and is not subject to the law of inertia. At high frequencies, the mass of the vibrating particles that transmit the sound wave's energy can be significant. Vibrating particles have mass. Inertia is proportional to the square of the frequency, even for particles with the smallest mass. Inertia is directly proportional to the wave amplitude and the vibrating mass.

## Remarks on the Mechanical Amplification of the Sound Wave

1. A quiet sound that is below the threshold of excitability cannot be amplified.
2. The OHC has afferent innervation; the received information is transmitted to the brain immediately after the signal is processed in the hair cell.
3. There is no mechanism for assessing and adjusting the sound amplification level.
4. If each depolarization of the OHC causes the cell to shorten by 4 – 5% of its length, then each depolarization pulls on the basilar membrane, regardless of the sound intensity.
5. In the case of polytones or speech, where loud and soft sounds occur. Loud sounds are received, and the information is sent to the brain. Soft sounds are isolated and amplified. It takes time. Are they sent separately to the brain?
6. The OHC has no direct connection with the basilar membrane. How is the accuracy of encoding of the transmitted information ensured?
7. A sound wave in fluid travels at 1450 m/s, traveling through the vestibular duct. A sound wave does not travel directly along both sides of the basilar membrane, as Bekesy had assumed. What contact does the traveling wave in the vestibular duct have with the distant basilar membrane? Bekesy omitted Reissner's membrane from his calculations so that the sound wave could travel on both sides of the basilar membrane. It travels through the fluid of the cochlear duct, the tectorial membrane, and through the organ of Corti with the hearing receptors. The wave doesn't transmit information to the receptor, as its purpose is to reach the basilar membrane and generate a traveling wave, which is supposed to induce cochlear fluid flow to activate the tip-link mechanism. Bekesy really complicated things. During the time-consuming amplification of a soft sound, a new traveling wave encodes information that is not related to the wave being amplified. Two waves overlap: the wave present on the traveling wave and the wave being amplified. This creates a problem of how these waves are received, processed in the auditory cell, and transmitted to the brain. The very important issue remains the energy required to generate additional work, especially with low-intensity amplification of high frequencies. The prestin, designed to perform the work of executing OHC contractions and additionally amplifying soft sounds, does not possess

the energy of its own to do such work. The suggestion that prestin derives the energy for its conformational changes from the electrochemical potential of the hair cell is not acceptable. Conformational changes in prestin are thought to be responsible for the shortening and lengthening of the OHC. It is necessary to calculate the required energy for amplifying a wave, e.g. 4000 Hz, from 20 dB by 40 dB, taking into account that the entire vibrating element has a mass of approximately 250 mg (OHC, basilar membrane, organ of Corti with fluid spaces, blood vessels, and nerves). All of this vibrates even more during this amplification — it requires additional energy.

An excitable cell, such as a hair cell, will degrade or die if it loses even a portion of its electrochemical energy. A cell's electrochemical energy is the difference between the various ions on either side of the cell membrane, which has different membrane permeability to individual ions. This energy cannot be transferred to prestin.

## Intracellular Signal Amplification at the Molecular Level

In all senses, there is an intracellular, regulated, molecular amplification. Intracellular amplification involves a complex set of factors, including phosphorylation and dephosphorylation of ion channels responsible for cell membrane conductance, ATP concentration, cAMP and cGMP levels, cell pH, osmotic pressure, the presence of ligands, and the activity of the sodium-potassium pump and Ca<sup>++</sup>ATPases. Ion pumps associated with the cell membrane play a significant role in maintaining fluctuating electrolyte levels within the cell. Intracellular amplification is associated with the activity of calcium-binding proteins, where calmodulin plays a significant role, influencing the production and degradation of cAMP and cGMP. It activates protein kinases and protein phosphatases and regulates the function of the calcium pump. It affects the contraction of muscle and non-muscle cells by activating cAMP-independent myosin light chain kinase. Calmodulin also influences transmitter exocytosis. The process of enzyme production or the rate of their breakdown is regulated within the cell. Calcium is a second messenger in the cell, acting faster than other second messengers: cAMP, cGMP, DAG, and IP<sub>3</sub>, which are produced in response to increased calcium levels or activated by G proteins [12]. The stage of second messenger production is one of several mechanisms of intracellular amplification. One enzyme molecule can produce several hundred second messengers. For proper hair cell function, a balance must be maintained between the influx of calcium into the cell through ion channels in accordance with the electrochemical potential and the rapid release of calcium out of the cell through ion channels, ion pumps, and ion exchangers. The signal, in the form of a sound

wave — that is, usable energy — reaches the receptors of the hair cells, which are specifically sensitive to the frequency of the wave, to a given sound wavelength. The hair cells are arranged along the basilar membrane according to the principle of tonotopy. Information is transmitted to the brain. Hair cells in the initial section of the basilar membrane detect high frequencies. As we approach the cap, lower and lower frequencies are detected. The location of the frequency reception site is transmitted to the central nervous system with great precision. Based on this, an assessment and analysis of frequency is conducted. The size and location of the receptor fields play a significant role, as they transmit impulses in the form of excitatory potentials to the nerve cell of the spiral ganglion. The transmission of information to the receptor must accurately reflect the sound wave at the input to the system. The conversion of the encoded sound wave energy into receptor potential takes place at the level of molecular transformations in the receptor. A signal in the form of mechanical energy from a sound wave, detected by the sound-sensitive molecules of the receptor, triggers conformational changes in the subsequent molecules responsible for gating the mechano-dependent potassium channels that regulate the flow of positive K<sup>+</sup> ions from the endolymph into the hair cell. The influx of positive ions into the cell, in accordance with the information contained in the sound wave, causes its depolarization and triggers a cascade of chemical reactions within the cell. Process regulation involves the constitutive system, responsible for processes related to cell life, as in normal cells. The second system, the regulatory system, is responsible for processes related to the reception, processing, and transmission of auditory information. One of the most important regulated processes is the production of the transmitter, its packaging, transport, and exocytosis to the synapses on the basal and lateral walls of the hair cell. The transmitters produced and released into the synapse cause the conversion of the chemical energy of the encoded information transmitter into electrical energy in the form of a postsynaptic excitatory potential, which in the nerve cell of the spiral ganglion is converted into an action potential, transmitted via the auditory nerves to the brain.

Throughout the signal pathway from the receptor to the brain, the received signals, which are too weak to reach the brain, are amplified. The energy of the signal transmitted by the auditory nerve is amplified by depolarization at each Ranvier node. Spatial summation and temporal summation, as well as presynaptic inhibition and postsynaptic inhibition, play a role. At high intensities, adaptation plays an important role.

## References

1. Wever EG, Bray CW. (1930). Auditory nerve impulses. *Science*. 71: 215.
2. Davis H. (1968). Mechanism of the inner ear. *Physiol. Laryngol.* 77: 644.
3. Brownell WE, Bador CR, Bertrand D, Diribanpierre Y. (1985). Evoked mechanical responses of isolated cochlear outer hair cells. *Science*. 227: 194.
4. He D, Lovan S, Ai Y, Li Y, Baisel K. (2014). Prestin at Year 14: Progress and Prospect. *Hear Res.* 311: 25-35.
5. Wysocki J, Kwacz M, Mrówka M, Skarżyński H. (2011). Comparison of round window membrana mechanics before and after experimental stapedotomy. *The Laryngoscop.* 121: 1958-1964.
6. Kwacz M, Marek P, Borkowski P, Mrówka M. (2013). A three-dimensional finite element model of round window membrane vibration before and after stapedotomy surgery. *Biomed. Model Mechanobiol.* 12: 1243-1261.
7. Myjkowski J. (2025). The secrets of hearing. *J Cur Tre Clin Case Rep.* 6: 1-3.
8. Miękisz St, Hendrich A. (1998). Selected topics in biophysics. Wrocław: Volumed. 91-96.
9. Piela L. (2022). Idee chemii kwantowej. PWN, Warsaw. 1300.
10. Myjkowski J. (2023). Submolecular Theory of Hearing, *HSOA J. Otolaryngology, Head and Neck Surgery.* 8: 69.
11. Myjkowski J. (2025). Inertia in the Ear, *WebLong Journal of Surgery.*
12. Myjkowski J. (2004). Transforming and transmitting auditory information]. *Otolaryngol Pol.* 58: 377-383.

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