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Changes in the Theory of Hearing

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Introduction

Bekesy's traveling wave theory is still propagated in textbooks, on the internet, and at the university level. There are many indications that the theory announced in 1928, corrected and supplemented, still does not meet the requirements of 21st century science. The basic assumptions of this theory dating back to the mid-19th century and announced in 1859 by Helmholtz have not changed. This concerns the hydrodynamics of cochlear fluids, the resonance of the longitudinal wave in the fluid with the transverse wave of the basilar membrane's natural vibrations, the transmission and coding of auditory information by cochlear fluids, and the importance of the basilar membrane in the reception and transmission of information. This theory recognizes the only signal route to the hair cell receptor is through the cochlear fluids and the basilar membrane. In the 1980s, this theory was supplemented with a questionable mechanism for amplifying quiet sounds by 40-50 dB. The mechanism is simple and involves the pulling of the basilar membrane by the contracting outer hair cells. "The submolecular theory of hearing", a new and modern theory of hearing, does not contain the errors of the previous theory and attempts to explain all hearing mechanisms in accordance with the scientific knowledge of various disciplines of the 21st century. This theory, first announced in 2003, has strong evidence collected from various research centers around the world.

Differences between the "Old" and "New" Theories of Hearing

According to the new sub-molecular theory of hearing, the energy of the sound wave absorbed by the auricle undergoes constructive interference with the energy transmitted to the skull bone from the tympanic membrane, in accordance with Huygens' principle that every point in a medium reached by a sound wave becomes the source of a new spherical wave. The wave energy from the ossicles of the middle ear is partially transmitted to the skull bone and the bony casing of the cochlea [1].

Each subsequent interference of the waves causes the summation of the sound wave energy, which directly reaches the receptor through the bone, without involving the basilar membrane and the fluids of the cochlea. The wave energy from the tympanic membrane to the oval window is conducted by the vibrations of the ossicles, up to a certain frequency. Above 3-4 kHz, sound waves are conducted through the ossicles without their vibration. The ossicles provide a medium through which sound waves are conducted as pressure changes. The transition from wave transmission as vibrations of the medium's mass to wave transmission without vibrations of the medium is determined by inertia in wave motion. The wave energy necessary to generate a sound wave is equal to the inertia of the vibrating mass transmitting the energy. Inertia in wave motion is directly proportional to the oscillating mass and to the wave amplitude, and proportional to the square of the increase in frequency [2].

With the same oscillating mass of the middle ear of 70 mg,

For 20 dB and a frequency of 100 Hz the inertia is 1.38×10^{-19}J.

For 20 dB and a frequency of 2000 Hz the inertia is 5.52×10^{-17}J.

For 40 dB and a frequency of 4000 Hz the inertia is 2.21×10^{-16}J.

For 20 dB, a frequency of 10,000 Hz the inertia is 1.38×10^{-15}J [3].

The inertia is 10,000 times greater for a wave frequency that is 100 times higher in the case of vibrations of the mass of the vibrating element. It follows that above a certain frequency; the sound wave is conducted through the ossicles without vibrations of the mass of the ossicles. A sound wave, having no mass, is not subject to inertia.

The sound wave in the middle ear is not subject to the amplification described in the traveling wave theory.

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Inertia F = ω^2xmassxamplitude. ω=2πxf, f = frequency. ω = radian/s., π = 3.14159. J (joule) is the energy needed to lift 100g to a height of 1 m, or 1 J = 1Nx1 meter. 1 Newton = 1 kgxm/s^2.

High frequencies transmitted through the ossicles without their vibrations cause rocking movements of the stapes, which were already observed by Bekesy. The cause of the rocking movements of the stapes is the uneven structure of the tympanic membrane on both sides of the malleus, which results in unequal transmission of energy [4]. Rocking movements are movements of the stapes plate, that are consistent with the frequency of the sound wave, along the transverse or longitudinal axis of the stapes plate. When one part of the plate generates forward wave motion, the other part of the plate generates reverse motion. This disrupts the transmission of wave energy due to friction between waves traveling in opposite directions, as well as destructive interference of the waves.

A complete transmission of auditory information carried by these waves is impossible. These degraded high-frequency waves are supposed to undergo resonance with the transverse wave of the basilar membrane's natural vibrations. Hearing high frequencies conducted to the receptor via the path marked by Bekesy or earlier by Helmholtz is impossible.

We can hear sounds up to 20 kHz, other mammals can hear up to 100 kHz, so there must be a different path for high frequency signals to reach the receptor. There is a quick and easy pathway through the bone and bony casing of the cochlea. Successive constructive interference of the sound wave from the auricle, tympanic membrane, malleus, and incus sums the energy of the sound wave with the energy transmitted from the stapes plate to the temporal bone. The formation of a receptor potential within 1.5 ms confirms the speed of the information transmission path.

Low-frequency vibrations are transmitted from the stapes plate to the cochlear fluid in the vestibular duct. They are conducted through the fluid and tissue continuity to the receptor, just as the baby's ear receptor in the mother's womb - from the second half of pregnancy - receives the sounds of the mother's voice or other internal sounds, e.g. the mother's heartbeat [5].

Vibrations from the stapes plate transmitted into the human inner ear cause changes in fluid pressure, i.e., a sound wave is generated that travels from the oval window to the cap and further to the round window. There is no fluid flow greater than the amplitude of the sound wave. According to Bekesy, the fluid pressure in the vestibular duct is 20 dB higher than the pressure in the tympanic duct. Bekesy assumed that the pressure difference on both sides of the basilar membrane is the source of the traveling wave on the basilar membrane. Bekesy's paradox is that the deflections on the basilar membrane arise in the direction of higher pressure, i.e., they arise in the organ of Corti itself, adjacent to the basilar membrane. Deflection of the basilar membrane does not stimulate the flow of cochlear fluid between the basilar membrane and the tectorial membrane to bend the hairs of the hair cells, because the organ of Corti is adjacent to the basilar membrane.

The greatest reduction in the wave amplitude in the cochlea (energy loss) occurs in the vestibular canal, which is spirally coiled and narrows three times from the oval window to the cap. Absorptive attenuation, reflective attenuation, and interference attenuation 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 of wave amplitude. The amplitude of the sound wave in the middle ear between the external auditory canal and the stapes plate decreases approximately 50 times. For a 90 dB wave, the amplitude decreases from 500 nm at the input to 11.7 nm at the stapes plate (Gambin, Prezentacje, Kraków 2018). In the area of the cap, the amplitude of this wave is about 1 nm - it decreases about 500 times. The amplitude of the sound wave from the stapes plate to the cap decreases 42.73 times for a 90 dB wave. Other sound wave intensities were not tested. [6,7].

At the beginning of the vestibular duct, the wave amplitude is similar to the amplitude of the stapes plate and decreases rapidly towards the cap. If the wave amplitude at the stapes plate decreases 50 times compared with the wave amplitude at the input, then the energy of this wave decreases 2500 times. The amplitude of the sound wave in the area of the cap decreases approximately 500 times. The energy of this wave decreases 250,000 times. The speed of sound wave in the vestibular duct is 1450 m/s. The speed of a traveling wave on the basilar membrane, according to Bekesy, is 50 m/s at the base of the cochlea and 2.9 m/s in the cap area. The difference in speed ranges from 29 times to 500 times. The wave speed is variable at each point of the basilar membrane, depending on the wave frequency.

Each wave frequency is expected to produce the greatest basilar membrane deflection at a different location and at a different time. The sound wave in the vestibular duct does not have direct contact with the basilar membrane. How is auditory information encoded in the sound wave transmitted with such accuracy, over a distance, with such enormous information compression? How many maximum deflections are generated on the basilar membrane given such a large variation in the resonance location on the basilar membrane and such density of information? The problem concerns polytones received at the same time. How, according to Bekesy, is information transmitted to the cochlear fluid? Cochlear fluid is absent along the basilar membrane, which is connected to the organ of Corti along the entire length of the basilar membrane.

There are major doubts regarding the calculation of the natural vibrations of the basilar membrane [8]. The basilar membrane vibrates together with the organ of Corti and is immersed in the cochlear fluid. There is significant energy attenuation. We hear a threshold tone with an amplitude of 0.01 nm when the energy of the forcing wave - the longitudinal wave in the fluid, is smaller than the energy of the forced wave - the transverse wave of the basilar membrane. Resonance cannot occur. A wave this small is received by the receptor. There must be another signal pathway to the receptor. In mammals and birds that hear from 20 kHz to 100 kHz, the natural vibrations of the basilar membrane have no significance, there is no resonance of the wave in the fluid with the natural vibrations of the basilar membrane. The human hearing organ uses the same mechanisms. The energy of the sound wave, not the fluid flow and wave resonance, is the adequate stimulus for the auditory receptor.

Billions of creatures on Earth lack cochlear fluids and a basilar membrane, and they hear perfectly well. The sound wave acts directly on the hearing receptor. Soft sounds received are amplified molecularly within the hair cell itself.

Wave Amplification in the Inner Ear

Already in the 1940s, Davis (1896-1982), while studying the functional currents of the auditory nerve, noticed a lack of

correspondence between the amplitude of the auditory nerve potentials and the intensity of sound waves in the external auditory canal [9].

The discovery of actin and myosin in the 1940s, and the discovery of ATP in 1929, provided the basis for a new theory of signal amplification in the inner ear in later years. Contractile proteins were thought to be responsible for OHC contraction, working on the same principle as muscle contraction. It was assumed that they enhance the movement of the basilar membrane caused by fluid flow in the inner ear. The increased maximum deflection of the basilar membrane for soft sounds is supposed to stimulate the IHC and transmit information to the brain. This leads to an amplification of the inner ear's response to soft sounds. The energy for the amplification was to come from ATP. The idea behind this amplification is still the same. The problem is to provide the necessary high energy to stimulate the basilar membrane to increase vibrations, especially for high frequencies.

Further research showed that this mechanism is too slow for high frequency amplification. OHC contractions were observed during changes in the electrolyte levels on the hair cell membrane (electrochemical potential), mainly Ca^{++} and K^{+} ions; OHC contractions also occurred without the presence of ATP.

Ashmore has introduced a new version of signal amplification. A change in the cell membrane potential causes a change in the electric field, which leads - according to Ashmore – to certain shifts of electrical charges in the cell membrane.

It was assumed that a kind of "molecular motor" was being created. It is a macromolecule in the cell membrane in which charge shifts cause its conformational changes, leading to a change in the cell surface by several percent.

In 1985, Brownell published research confirming that the OHC shortens by up to 5% of its length during cell depolarization. Repolarization and hyperpolarization cause OHC lengthening. The molecular mechanism of intracellular amplification – as it exists in other sense organs – was still unknown.

In 2000, a new protein was identified in the OHC wall, which was named prestin [10]. It has been assumed that prestin is responsible for OHC contractions and is a molecular motor that does not use ATP energy, because OHC contractions have been observed in the absence of ATP. This is a questionable statement, because ATP is necessary for the operation of ion pumps in the cell wall - it is the energy that powers ion pumps, which are involved in the generation of membrane potential along with the variable permeability of the cell membrane to ions on both sides of the cell membrane. Without ATP there is no electrochemical potential.

It was assumed that the conformational changes in prestin responsible for cell shortening and elongation are powered by the electrochemical energy of the cell wall.

An excitable cell such as a hair cell cannot lose such a large amount of energy during each contraction. Loss of cell energy causes disruption of its functioning or leads to its death. Losing so much energy in each wave cycle is impossible.

Prestin does not have its own potential or kinetic energy to perform the work of pulling the basilar membrane up by 1 nm (40 dB) in accordance with the varying frequency of the oscillating mass. Please note that the basilar membrane does not vibrate on its own. Its close connection with the organ of Corti makes the vibrating element the entire conglomerate of the basilar membrane with the organ of Corti and the fluids of the cochlea. The speed, acceleration and mass of the vibrating element determine the inertia of the system.

The energy necessary to generate a new wave on the basilar membrane is equal to the inertia of the vibrating system (a pseudo-resistive force). Inertia in wave motion is calculated using the formula: $(2\pi xf)^2 xmxA$ in Newtons, or J – joules. f – frequency, m – vibrating mass, A – amplitude.

For example, the vibrating mass of the inner ear was assumed to be 250 mg for calculations: (basilar membrane with connective tissue on the lower surface, organ of Corti with fluid spaces, fluid of the cochlear canal to move the hairs of the hair cells, cadherin fibers). Everything vibrates during the transmission of information to the receptor – along a path determined by Bekesy's traveling wave theory.

Inertia for selected intensities and frequencies: Amplitude 0.1 nm = 20 dB. Amplitude 1 nm = 40 dB, Amplitude 10 nm = 60 dB – according to AI.

250 mg - 20 dB = 0.1 nm - 100 Hz ---- $9.87x10^{-9}N$

250 mg - 20 dB = 0.1 nm - 1000 Hz --- $9.87x10^{-7}N$

250 mg - 20 dB = 0.1 nm - 100000 Hz - $9.87x10^{-5}N$

250 mg - 40 dB = 1 nm --- 100 Hz ---- $98.7x10^{-8}N$

250 mg - 40 dB = 1 nm ---1000 Hz --- $9.88x10^{-6}N$

250 mg - 40 dB = 1 nm --- 10,000 Hz -- $98.7x10^{-4}N$

250 mg – 60 dB = 10 nm - 100 Hz --- $9.87x10^{-7}N$

250 mg – 60 dB = 10 nm - 1000 Hz -- $9.87x10^{-5}N$

250 mg – 60 dB – 10 nm – 10,000 Hz - $9.87x10^{-3}N$

This is a 40 dB to 60 dB amplification of the 20 dB silent wave.

Conclusion: Inertia in wave motion is directly proportional to the vibrating mass and amplitude, and is proportional to the square of the frequency of the sound wave.

Amplitude or mass increases 10 times – inertia increases 10 times.

Frequency increases 10 times – inertia increases 100 times.

The second problem with mechanically amplifying soft sounds by pulling on the basilar membrane is that waves below the excitability threshold cannot be amplified because they do not cause OHC depolarization. The received silent wave causes the formation of a receptor potential, depolarization of the OHC, and the formation of an action potential, transmitted to the brain. We hear the quiet sounds of rustling leaves, or soft, pleasant whispers in our ears, which fortunately are not amplified.

Amplifying quiet sounds with this invented mechanical method is time-consuming, and operates with a millisecond delay after the quiet signal is received by the OHC. In 1 millisecond, a sound wave in the fluid travels 1450 mm. During this time, the wave on the basilar membrane travels 50-2.9 mm, compressing the information transmitted to the brain during this time, slowing down the transmission from 29 to 500 times, depending on the frequency.

Pulling on the basilar membrane, even a tenth of a millisecond

after the silent wave is received by the OHC, causes the amplification of unknown wave, which is at that moment "condensed" on the basilar membrane. The frequency of this wave and its intensity are unknown. The wave that encodes certain information is disturbed. What is the point of amplifying a wave of a different frequency and different, perhaps higher, intensity?

What happens next to both waves?

The pulling up of the basilar membrane connected to the organ of Corti causes the organ of Corti to be pulled up along with the contracting cell and its hairs.

It is difficult to agree that hair cells and their hairs vibrate continuously in accordance with the frequency and amplitude of the sound wave – just like the basilar membrane.

It is highly questionable whether electrochemical energy is sufficient to set such a large oscillating mass into motion in order to amplify soft sounds, especially at high frequencies, where inertia increases proportionally to the square of the increase in frequency.

Another problem concerns the coding of information transmitted by sound waves. What information, and in what way, is transmitted by OHC contraction through pulling on the basilar membrane? The transmission of information concerns amplitude, but also frequency, harmonic components, phase shifts, length of sound, and accent.

Another problem is that soft sounds from musical instruments also contain harmonic components ranging from a few to several dozen for a single tone. Each harmonic component has a different frequency, which means that the energy required to amplify that frequency is different for each component. There is no mechanism for adjusting the amplification level of the fundamental tone and subsequent harmonic components. How is the location of resonance and the greatest deflection of the basilar membrane connected to the organ of Corti transmitted?

The traveling wave theory accepts contradictory information regarding OHC contractions and the amplification of soft tones. OHC contractions depend on cell depolarization, which is dependent on the functioning of ion channels and the permeability of the cell membrane [11].

The operating cycle of voltage-gated channels in the cell membrane is limited to a few milliseconds. With simultaneous depolarization of all ion channels and simultaneous OHC contraction, it is impossible for the cell contraction frequency to reach 100 kHz. The sub-molecular theory of hearing suggests the existence of limited depolarization of the OHC wall and limited contraction at the site of depolarization. It can be assumed that this is related to the large number of afferent and efferent synapses on the hair cell. Limited OHC contractions do not produce as much energy as needed to generate and amplify soft sounds. The contraction of the entire cell after electrical stimulation - as evidence of contractility of the whole cell - is a misconception. The ion channels responsible for cell depolarization are turned off.

Intracellular signal amplification at the molecular level

Intracellular amplification at the molecular level involves a whole range of mechanisms, such as: phosphorylation and dephosphorylation of cell wall ion channels, ATP concentration, cAMP, cGMP, cell *pH*, osmotic pressure, sodium-potassium pump activity, Ca^{++}ATPases. Intracellular amplification is related to the activity of calcium-binding proteins, where calmodulin plays an important role, influencing the production and breakdown of cAMP and cGMP. It activates protein kinases and phosphatases, and regulates the functioning of the calcium pump. It influences the contraction of muscle and non-muscle cells by activating cAMP-independent myosin light chain kinase. Calmodulin also affects transmitter exocytosis. The process of enzyme production or the rate of their breakdown is regulated in the cell. Calcium is the second messenger of information in the cell, acting faster than the other second messengers: cAMP, cGMP, DAG, IP3, which are produced in connection with the increase in calcium levels or activated by G protein.

The step of producing second messengers is one of several mechanisms of intracellular amplification. One enzyme molecule can produce several hundred-second messengers.

The signal, in the form of a sound wave, i.e. useful energy, reaches the receptors of the hair cells that have a specific sensitivity to the wave frequency, to a given sound wave length.

The information is transmitted to the brain. The hair cells in the initial section of the basilar membrane detect high frequencies. As one gets closer to the cap, lower and lower frequencies are received [12].

The location of the frequency reception point is transmitted to the central nervous system with great precision. Based on this, frequency evaluation and analysis are performed. An important role is played by the size and location of the receptor fields from which impulses in the form of excitatory potential reach the nerve cell of the spiral ganglion.

The transmission of information to the receptor must be faithful to 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. The signal in the form of mechanical energy of the sound wave, received by the sound-sensitive molecules of the receptor, causes conformational changes in subsequent molecules responsible for gating mechano-dependent potassium channels, regulating the flow of positive K+ ions from the endolymph into the interior of 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.

Conclusions

In the second half of the 20th century, scientists noticed a disproportion between the measured auditory nerve potential and the intensity of the wave delivered to the external auditory canal. They correctly concluded that there was an unknown force in the inner ear that amplifies the signal transmitted to the ear. Without knowing the mechanisms of reception and transmission of auditory information, a simple mechanical method of amplification - through pulling on the basilar membrane - was adopted as a plausible explanation at that time.

This mechanism resulted from the belief in the infallibility of Bekesy's traveling wave theory, which had been awarded the Nobel Prize just a few years earlier. However, over the past few decades, Science has made enormous progress. In light of current scientific knowledge, the theory of mechanical amplification, like the traveling wave theory itself, contains many ambiguities and inconsistencies with present-day understanding of hearing. It is time to replace the paradigm of hearing theory with a new paradigm [13].

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