

Comparison of Two Theories of Hearing

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Received: 14 Feb 2023

Accepted: 31 Mar 2023

Published: 07 Apr 2023

JShortName: ACMCR

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Citation:

Myjkowski J, Comparison of Two Theories of Hearing. Ann Clin Med Case Rep. 2023; V10(16): 1-4

1. Theoretical Study

A comparison of two theories of hearing:

Theory No. 1 – the old theory by Bekesy, published in 1928.

Theory No. 2 – a new, modern theory, Submolecular Theory of Hearing –.

Author of the theory: Jan Myjkowski.

Theory No. 1 – does not take into account the value of inertia in the ear and does not calculate it properly.

Theory 2 – recognizes it and calculates the value of inertia in the ear by using the formula: $(2\pi \times \text{frequency})^2 \times \text{amplitude} \times \text{mass} / \text{mm}^2$. For a threshold tone of 1,000 Hz and 0 dB, the inertia of the middle ear is 0.009859 g/mm². For a tone of 10,000 Hz and 100 dB, the inertia of the middle ear is 98596 g/mm². For each frequency, the value of inertia is different and proportional to the square of the frequency. To these values must be added the inertia of the inner ear. A sound wave travelling to the receptor through the bony housing of the cochlea has no mass and no inertia.

Theory No. 1 – does not recognize the existing fading of energy on its way to the receptor; instead, it recognizes the mechanical amplification of quiet tones of 40 – 50 dB through an OHC contraction, and we still perceive those tones as quiet.

Theory No. 2 – a tone below the auditory threshold cannot be amplified by an OHC contraction. An amplification occurs in the auditory cell at the molecular level, but only in the case of signals received by the receptor, and too small to reach the center.

Theory No. 1 – the basilemma is responsible for the frequency resolution.

Theory No. 2 – a receptor in the form of auditory cells with a specific ability to receive sound waves of a given frequency is responsible for the frequency resolution. A piece of evidence is the immobilization of the basilemma which does not affect the recognition of both a tone and a timbre.

Theory No. 1 – is based on the calculation of the basilemma's natural oscillations, calculated according to Bekesy's methodology.

Theory No. 2 – those calculations are incorrect, the basilemma vibrates along with the entire organ of Corti, the fluid spaces and the connective tissue on the lower surface of the basilemma, all being embedded in a fluid that has some suppressive properties. Bekesy would calculate proper vibrations in the air for a very thin and narrow strip of connective tissue.

Theory No. 1 – resonance is the basis for the transmission of sound wave vibrations to the basilemma.

Theory No. 2 – resonance is a process of energy transfer over time, with frequency compatibility or high similarity between the forcing and forced waves. The rate of receptor potential formation lies within tenths of a millisecond – which is in conflict with resonance. A sound wave is a longitudinal wave, whereas a wave on the basilemma is a transverse wave. The speed of the wave in the fluid is 1,450 m/s and the speed of the wave on the basilemma – from 1.9 m/s to a few dozen or so m/s.

Theory No. 1 – the tip-links mechanism is responsible for the gating of mechanosensitive channels.

Theory No. 2 – the mechanical energy of the sound wave is responsible for gating the mechanosensitive potassium channels of the auditory hair cells. This takes place on the atom, particle and

molecular level. Further evidence is a low energy – since on the pathway of fluids of both the cochlea and basilemma tip-links mechanism is feasible because it disappears. It reaches the receptor by a different route, viz. through the bone housing of the cochlea.

Theory No.1 – cadherin fibers, tensioned up due to the bending of the auditory cell hairs, will tighten the cell membrane of the hair, and this is sufficient to gate the ion channel located next to the cadherin attachment to the cell membrane.

Theory No. 2 – it is impossible that such a simple mechanism could be responsible for the transmission of complex information. It is impossible to open and close a channel with a lumen of 0.3 nanometers to 1 nm. The fiber tip springs described above are not connected to the channel gating mechanism. Molecular motors – myosins – are supposed to be responsible for closing an ion channel. These are too slow to handle channels operating at frequencies of up to 200 kHz. It is difficult to accept the encoding of multi-tones that have numerous harmonics by means of pulling on the cell membrane itself, without contact with the mechanosensitive channel itself.

Theory No. 1 – does not explain all the molecular processes that make up the conversion of receptor potential to action potential in the auditory nerve.

Theory No. 2 – describes subsequent processes within the auditory cell related to proteins, enzymes, calcium, intracellular information transmitters, transmitter production, its transport and secretion, synapse operation, intracellular amplification, summation, presynaptic and postsynaptic inhibition, the importance of afferent and efferent innervation, etc. A detailed description can be found in the paper entitled: “Przetwarzanie i przekazywanie informacji słuchowych” (Auditory information processing and transmission). – *Otolaryngologia Polska* Nr 2, 2004, pp. 377–383.

Theory No. 1 – assumes a displacement of cochlear fluid masses in line with the sound wave in a range of amplitudes, according to the amplitude of the transverse basilemma wave, for the inclination of the auditory cell hairs and the functioning of the tip-links.

Theory No. 2 – a displacement of a sound wave in solids and in a fluid is not related to the mass displacement of the environment. There is only a displacement of the environmental particles according to the amplitude of the wave in both directions, with no change in position with respect to the axis of excursion. A pressure wave moves forward, has no mass and is not subject to the law of inertia.

Theory No. 1 has it, an OHC, provided with afferent innervation, does not use it, and signals to the center are retransmitted exclusively through the IHC.

Theory No.2 – the afferent innervation of the OHC performs well. Otherwise, it has no right at all to exist.

Theory No. 1 – multi-tones of different loudness are separated, quiet tones are amplified and loud tones are transmitted directly to the center. There is no description of the transmission of the information of the quiet tones, as well as after what time and with what subsequent waves does the transmission occur.

Theory No.2 – believes that a signal cannot be split and the information of quiet sounds cannot be transmitted in delay with other information.

Theory No. 1 – cannot explain the lack of high frequency transmission in stapedotomy operations.

Theory No.2 – an operation shuts down the transmission of wave energy to the bone housing of the cochlea for a direct and rapid signal transmission to the receptor. A sound wave, for having no mass, is not subject to inertia. It can be transmitted up to 20 kHz in humans and up to 200 kHz in bats. Up to 100 kHz in mice. Swinging movements of the stapes play a role in the transmission of high frequencies. Those movements are excluded in stapedotomy.

Theory No.1 – does not explain directional hearing. The problem is the timing of the receptor potential and the disappearance of energy on the way to the receptor through the cochlear fluids and the basilemma, as well as an amplification of quiet tones.

Theory 2 – the basis for directional hearing is the interaural distance in binaural hearing. This size of ears spacing in humans, other mammals and birds results in different distances between the ear and the sound source, deflected from a line straight ahead. An angle of hearing will be formed. The difference in the distance to the respective ear produces a difference in the receptor excitation time, the difference in the path also causes a reduction in the energy of the incoming sound wave. In humans, the time difference of the waves reaching the respective ear is 0.0006 seconds! There is a very small difference in intensity.

Theory No. 1 – cannot explain the preservation of existing partial hearing in the case of cochlear implant surgery when the basilemma is immobilized.

Theory No.2 – the sound wave runs through the bony housing of the cochlea, regardless of the immobilization of the basilemma – which is corroborated by hearing. This provides ossicular hearing and ‘boneless ossicular’ hearing, i.e. the conduction of sound waves through the soft tissues from the eardrum cavity to the receptor.

Theory No. 1 – there is a threshold of excitability and mechanical amplification of a signal by pulling the basilemma by OHC contractions.

Theory 2 – low intensity and high frequency amplitudes are heard by us, but not via the cochlear and basilemma fluid pathway. They are below the hearing threshold, do not cause OHC depolarization, and therefore cannot be mechanically amplified.

Theory No. 1 – uses an incorrect name ‘auditory cell cilia’. Also incorrect is the name ‘stereocilia’, viz. rigid cilia.

Theory 2—there are no cilia on the auditory cells. There are some protrusions of the auditory cells formed into small hairs. The cilia are found in the respiratory tracts, have a completely different cell structure from the auditory cell hairs, and have an ability to move.

Theory No. 1—Does not differentiate between quiet and loud tone response times, does not analyse the hearing of mammals and birds that have the same hearing system.

Theory No. 2—is based on studies on the time of the signal path to the receptor and on the study of hearing thresholds. In humans, the receptor potential arises after tenths of a millisecond. According to calculations **consistent with the traveling wave theory**, for quiet tones this time is about 3 milliseconds. A human hears a threshold tone having in the external auditory canal a wave amplitude of 0.01 nm = 10 picometres.

Theory No. 1— does not explain the hearing of short tones whose duration time is as long as tenths of a millisecond and which are perceived by the receptor.

Theory No. 2 – the signal pathway through the cochlear fluids requires some resonance action. Resonance within tenths of a millisecond is not possible, and yet, the signal reaches the receptor. Therefore, there is another signal pathway to the receptor.

Theory No. 1—cannot explain the encoding of information transmitted by the transverse wave of the basilemma, by cochlear fluid flows, by bending the auditory cell hairs, by the cadherin junctions of auditory cell hairs, by the springs of the lower cadherin terminals, by the stretched cell membrane of a shorter hair. Multiple tones of varying intensity, with numerous harmonic tones, will pose a problem.

Theory No. 2 – promotes a signal pathway through the cochlear bony housing, which means that such encoding is superfluous. The energy of the sound wave reaches the receptor directly, quickly without any change, with no superfluous coding transformations

Theory No. 1 – fails to analyse and to account for multiple exchanges of energy for encoding information on the way to the receptor.

Theory No. 2 – postulates that the amount of energy conversions on the way to the receptor, assuming a pathway through the cochlear fluids and the basilemma, is far too high and can influence a distortion of information. A longitudinal wave in fluids is converted into a transverse, slow wave on the basilemma. A transverse wave in fluids does not work, so it is converted into a longitudinal wave. This wave is supposed to move the hairs of the auditory cells according to the wave’s amplitude and frequency. The hairs bend in a fluid that has attenuating properties and transmit their information to the cadherin fibers. At the lower end of the hairs are

arranged some springs—acting as links; in operation are also molecular motors—myosins, whose task is to close the K⁺ mechanosensitive channels. This pathway will be subject to inertia.

Theory No 1 – mechanosensitive channels are calcium channels and such may also be potassium channels, or both at the same time.

Theory 2—assumes that the mechanosensitive channels in the ear are potassium channels. The rationale is that there are very high levels of potassium in the endolymph produced by a large input of ATP energy by the potassium pumps in the vascular striatum.

Theory No. 1 – describes a single mechanosensitive ion channel on the auditory cell capillaries, and in addition only in the inferior rows of capillaries.

Theory 2 – this thesis is false. The cell membrane of the auditory cell capillaries constitutes an extension of the cell membrane of the whole cell. This membrane has on it several square micrometers a large number of sodium ion channels, tension-related potassium ion channels, calcium-dependent and ligand-dependent calcium channels of 3 types with different conductance and different sensitivity to depolarisation. There are also chloride channels and water pores.

Theory No. 1—the tip-links mechanism is essential for the transmission of sound wave energy to the receptor.

Theory No. 2—Mammals have the ability to perceive sound wave energy over a wide range, and some mammals use echolocation, others make use of electromagnetic fields and also electric fields generated by other organisms. The energy and encoded information are conveyed by a sound wave directly to the specific receptor. Here, the potential energy of a sound wave is converted into the chemical potential of the atomic bonds of the sound-sensitive molecules. There are changes in the vibrations of atoms and molecules, changes in atomic bond lengths, changes in oscillations, changes in valence angles, changes in electron spheres. This causes conformational changes in the molecules, changes in their dimensions and creates an opportunity to perform work, viz. controlling the openness of mechanosensitive potassium ion channels—that is, dependent on the information contained in the sound wave. This process is referred to as ion channel gating.

Theory No. 1—based on tests with electric current, OHC cells have an ability to contract up to 70000/s. This provides for OHCs an opportunity of amplifying quiet sounds.

Theory No. 2—investigations into contractions of an auditory cell by using electric current is incorrect and unreliable. Depolarization and contractions of the OHC depend on ion channels, and these have their own cycle of action, viz.: excitation, opening, closing and a period of refraction with no sensitivity to stimulation, to a change in potential. This process takes place over a period of time that cannot be reduced almost to zero.

Theory No. 1 – the energy of a sound wave in bone conductivity is transferred to the cochlear fluid and the subsequent signal path is the same as in air conduction with the origin of the wave running on the basilemma.

Theory 2 – believes that such a ratiocination is **incorrect**: The round window has 20 times more elasticity than the oval window and, in the event of a break in the middle ear ossicular chain, a wave on the basilemma may possibly be generated from the round window – viz. ‘reverse’ on the basilemma. If both windows are blocked, ossicular hearing is preserved in the absence of any wave on the basilemma.

Theory No. 1 – does not explain the mechanism of resonance of the longitudinal sound wave with the transverse wave on the basilemma. Neither does it explain why a simple, while being pushed on the side at right angles will not increase the amplitude of the swing excursion.

Theory No. 2 – has no such problems!