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Sound Wave Amplification in The Inner Ear

Abstract:

The first part of this paper briefly describes some of the processes occurring in the inner ear at the sub molecular level that make up the reception and transmission of auditory information. An accurate and rapid transmission of information to the receptor depends on these processes. The importance of molecular changes in the auditory receptor and in the auditory cell itself has been highlighted.

The second part presents an intracellular amplification mechanism such as exists in other sense organs. The molecular level ensures the speed and accuracy of amplification of information received, too weak to reach the center where it is analyzed..

Key words: sound wave; auditory cells

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Introduction:

Synopsis:

The first part of this paper briefly describes some of the processes occurring in the inner ear at the submolecular level that make up the reception and transmission of auditory information. An accurate and rapid transmission of information to the receptor depends on these processes. The importance of molecular changes in the auditory receptor and in the auditory cell itself has been highlighted.

The second part presents an intracellular amplification mechanism such as exists in other sense organs. The molecular level ensures the speed and accuracy of amplification of information received, too weak to reach the center where it is analyzed.

Mechanisms of hearing:

A sound wave reaching the inner ear transmits encoded information to the hearing receptor. An auditory stimulus is the energy of the sound wave acting on the sound-sensitive molecules of the auditory cells. These molecules transfer information in the form of energy to neighboring molecules undergoing conformation and the resulting conformers constitute a part of the mechanism that regulates the gating of mechanosensitive potassium ion channels. The energy of the sound waves controls the openness of the ion channel by acting on the activation or inactivation gate. According to the signal energy, the channel lumen allows between 0 and 6000 potassium ions/ms to pass through. The influx of positive potassium ions into the auditory cell causes it to depolarize. So that the depolarization may develop, a certain threshold of the number of ions entering the cell must be exceeded. Molecular changes take place inside the auditory cell leading to the release of transmitter into the synapse and the formation of an action potential, which is conducted to the center. The receptor potential is generated in 1.5-1.9 ms. The signal reaches the center with a delay due to synaptic delays on the pathway [1].

A serious issue will arise at the level of depolarization of the auditory cell, viz. it is difficult to accept the view that any signal, regardless of its intensity, causes maximum depolarization of the entire auditory cell. Such a situation arises in the nerve cell and the result is a maximum action potential.

In the auditory cell, depolarization triggers molecular processes in the auditory cell which lead to the production, transport and secretion of a transmitter into the chemical synapse, where generated is a postsynaptic excitatory potential, conducted to

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the spiral ganglion nerve cells, where an action potential is formed. The secretion of transmitter into the synapse is dependent upon the signal energy of the sound wave inducing depolarization of the cell. It is believed to be local depolarization with the transmission of information to the corresponding synapse which is the origin of the afferent nerve, conducting information to the center.

Another major argument against simultaneous depolarization of the auditory cell is the time required for a full cycle of subsequent depolarization and repolarization. Depolarization depends on the operation of the ion channels of the auditory cell wall, When the sodium channels are in an inactivated state, inducing a repolarization is impossible. This is the refraction period after each depolarization of the entire auditory cell. The ion channel goes from an inactivated state to a closed state. It takes approximately 0.4 ms to return to the active state. The times, respectively, of inactivation, relative and absolute refraction mean that the frequency of depolarization of the whole auditory cell cannot exceed 2,500/s. [2]. This also excludes an amplification of quiet high-frequency tones - by the physical method, or OHC contractions.

Yet another problem is that with the OHC shrinking method that theoretically amplified can be only quiet tones, already received, when the information has already been transmitted to the center. What is the point of amplifying such a signal? Pulling at the basilemma will interfere with the transmission of the information currently being conducted by the travelling wave. We do not perceive straight harmonic waves which could be amplified, because the successive waves are invariable.

The time difference between the quiet wave that supposedly needs to be amplified by 40-50 dB, and the wave on the basilemma is about 0.4 - 0.5 ms. During this time, approximately 580 mm of sound wave runs in the cochlear fluid, encoding various information. An extraneous wave is amplified by the pull of the basilar membrane. If the existence of a simultaneous maximal depolarization of the cell is to be assumed, the contraction of the cell pulling at the basilemma will be maximal regardless of the intensity of the sound. If there is no mechanism to regulate the strength of the contraction, then loud tones will be also amplified by 40-50 dB [3,4].

If prestin is thought to be responsible for OHC contraction and sound amplification, then it will not use the universal energy source ATP [5]. The driving force (energy source) of prestin is supposed to be the electrochemical potential which depends on the levels of chlorine ions and HCO3 on either side of the cell membrane. Prestin is provided with sensors for Cl and HCO3- ion levels on both the surfaces of the cell membrane. The intracellular Cl- current is involved in the cell repolarization , increasing thus the chlorine level in the cell above 4 mM, whereas the chlorine level outside the cell is 114 mM. The level of Cl ions outside the cell is almost unchanged. Chlorine ions play a stabilizing role in the membrane potential. The Cl- equilibrium potential is close to the equilibrium membrane potential dependent, in turn, on sodium and potassium ions [6]. It is difficult to expect such a large energy transfer for prestin doing a large amount of work related to shortening and lengthening the OHC length by about 4 %. An auditory cell with a length of 250 µ is shortened by 1000 nm. If one assumes that one end of the cell shortens by 500 nm, then as a result of pulling at the basilemma the amplitude of the travelling wave excursion will be increased by an amplitude equal to 90 dB. This quiet tone at the input, amplified in the middle ear by 33 dB, will be then amplified in the inner ear by 90 dB? Can a completely different extraneous tone be amplified by 40-50 dB (90 dB?). We still hear a quiet tone perceived as a quiet tone. A middle ear amplification is also questionable, as there is no 99.9% loss of energy of the wave falling on the cochlear fluid. The sound wave falls on the elastic, energy-absorbing eardrum. On the cochlear fluid energy is transferred from the stapes with a comparable impedance value, resulting in no such energy loss

Should prestin be formed into a globular protein and cover the walls of the OHC, then what conformational changes will cause it to contract only along the OHC long axis? The electrochemical potential acts across the cell membrane; hence, the force vector acts on the prestin atoms in a line perpendicular to the length of the auditory cell. If the auditory cell decreases in length, this will increase in cross-section; the volume of the cell will not change. The auditory cell has no direct connection to the basilemma. There is no explanation of how a shrinking auditory cell encodes all the information transmitted to the receptor. This is inconsistent with the principles of quantum physics.

If it is assumed that the sound wave to the receptor does not run through the cochlear fluids and the amplification of quiet tones occurs in the auditory cell, what role will prestin play? Perhaps it only increases the pressure in the cell during exocytosis of the transmitter to the synapse, which facilitates and accelerates the emptying of synaptic vesicles. In the case of multi-tones containing quiet and loud sounds – will the quiet ones be separated from the loud ones for amplification? And with a delay are they sent to the center? Along what route? Together with information from other waves already on the way to the receptor? Is there any constructive interference created? Will 2 separate streams of fluid flowing from the basilemma to the tip-links mechanism arise? These 2 fluid streams become separated as one heads for the OHC and the enhanced one for the IHC. How do the fluid streams of the cochlea simultaneously encode amplitude, frequency, aliquots, phase shifts and sound length? Is the transfer of energy encoding information continuous - according to classical physics? Or quantised? - according to physics or quantum chemistry [7].

If the threshold signal of a young person at the entrance into the external auditory canal has a wave amplitude of 0.01 nm, and if on its way through the cochlear fluids the amplitude of this wave decreases by more than 100 times, its amplitude will be 0.0001nm. And can such a wave produce a travelling wave? or fluid flows in the cochlea? It causes the hairs of the auditory cells, more than 1,000,000 times thicker than the amplitude of the sound wave, to tilt or bend. A wave not received by the receptor cannot be amplified.

There must be a different signal path to the receptor, because so tine a signal is received. A barn owl can receive sounds with an amplitude at the input of 0.001 nm, when the amplitude of the wave is about 100 times smaller than the diameter of the atoms that make up the basilemma structure. How is a travelling wave formed? The owl can recognize the frequency and

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direction from which the sound is arriving.

High-frequency sounds cause swaying movements of the stapes. Also in the case of quiet tones. There is a hinging movement of the stapes plate either along the transverse or longitudinal axis of the plate. One part of the plate generates a forward movement of the fluid - already described by Bekesy - while at the same time, the other part of the plate generates a backward movement. There is the possibility of destructive interference. How is auditory information encoded? How is a travelling wave formed? Especially, since the speed of the sound wave in the cochlear fluid is 1450 m/s, while the speed of the travelling wave on the basilemma is 2.9-50 m/s, is variable at each point of the basilemma and depends on the frequency of the sound wave. According to the said Bekesy principle, will a multi-tone of different frequencies produce on the basilemma a series of wave peaks corresponding to the transmitted frequencies? Do the waves undergo superposition, and is there generated a single maximum excursion - which will encode all the information? Each traveling wave peak generates its own fluid stream encoding information heading towards the tip-links. This is an unacceptable, but still recognized, concept, consistent with travelling wave theory.

Signal amplification:

In all senses there is an intracellular, regulated, molecular amplification. Most chemical reactions and energy transfer between small molecules will take place in 10^{-14} s. Those are reactions at the atomic and electron level. 'Difficult' reactions occur 1,000 times slower, but it is still 10^{-11} s.

Intracellular amplification constitutes a whole complex of factors such as phosphorylation and dephosphorylation of ion channels responsible for cell membrane conductance, ATP concentration, cAMP levels, cGMP, cell pH, osmotic pressure, presence of ligands, and operation of Ca++ATPase pumps. These membrane-associated pumps play a major role in maintaining fluctuating calcium levels in the cell. Intracellular potentiation is also related to the action of calcium-binding proteins where calmodulin, which influences the production and breakdown of cAMP and cGMP, plays an important role. It activates protein kinases and phosphatases and regulates the calcium pump. Influences the contraction of muscle and non-muscle cells through the activation of light myosin chains of cAMP-independent kinase. Calmodulin also affects the transmitter's exocytosis. The binding of 4 calcium atoms by calmodulin, increases its action 1000-fold. Enzyme production or the rate of enzyme degradation is subject to regulation.

Calcium is the second messenger of information in the cell, acting faster than the other second messengers: cAMP, cGMP, DAG, IP3, produced in association with an increase in calcium levels or activated by G protein. The production stage of the second messengers is one of several mechanisms of intracellular amplification. One enzyme molecule can produce several hundred-second messengers. Received tones, whose energy is too low to reach the center, are amplified.

Intracellular signal amplification is one of the main pillars of the 'Submolecular Theory of Hearing' [8].

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