Editorial Article

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Analysis of Amplification of a Sound Wave

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According to the theory of traveling wave, silent sounds are amplified by 40-50 dB due to OHC contractions and the pulling at the basilemma towards the tectorial membrane, which amplifies a signal for the IHC. The OHC is only an amplifier for the IHC. This is the dogma of the theory.

Let us analyze the mechanism of such an amplification.

1. The threshold tone cannot be amplified this way, since the signal will disappear on the way to the receptor and is insufficient to depolarize the auditory cells. If a 90 dB and 800 Hz tone, while traveling to from the external auditory meatus to the oval window, will decrease from 500 nm to 0.5 nm, then the threshold signal of an amplitude of 0.008 nm will also fade several hundred times.

2. A 20 dB tone (silent tone) has a wave amplitude of 0,8 nm. At a frequency of 100 Hz, the length of the wave in cochlear fluids is 1,450 cm. The length of the basilemma is 35 mm. A wave travels in cochlear fluids at a speed of 1,450 m/s. Vibrations of the basilemma, according to the traveling wave theory, will appear due to a resonance of the sound wave and a spot on the basilemma whose proper frequencies are either identical or very close to the frequency of the sound wave. The transmission of energy of the forcing wave to the wave being forced takes place in a determined time, and the transmission is accurate only in the case of conformity of the wave frequency and direction. In this case there is a big incompatibility of wave directions. A sound wave is a longitudinal wave, whereas the wave on the basilemma is a transverse wave whose direction of deflections is perpendicular to the sound wave. The effect of transmission of information is like in the case of a swing pushed from the direction the perpendicular to the line of deflections It is necessary to take into consideration that the peak of deflection of a sound wave (potential energy) that conveys energy (wave peak) occurs once per 1 wave period. It takes place at 1,450 cm intervals! Assuming theoretically that each section of the basilemma has a specific value of proper vibrations, it should be accepted, although with a large margin, that 1 mm of basilemma has a frequency compatible with a 100 Hz wave, in such a case a sound wave travels over that 1 mm of basilemma in a time of 145 μs, and the situation occurs repeatedly at 1 s intervals. In such a time no resonance is possible. After 1 second, over the basilemma there is a completely different wave, perhaps of a high amplitude, or several waves simultaneously with harmonics

3. A sound wave travels in the vestibule duct to the cupula, and then in the tympanic duct to the round window. In his calculations, Bekesy assumed erroneously that a sound wave travels on both the sides of the basilemma – not only directly but also in a straight tube, not in a cochlea. He left out the presence of Reissner's membrane and connected the vestibular duct to the cochlear tube so that a sound wave may be in resonance with the basilemma. In natural conditions, a sound wave is separated from the basilemma by Reissner's membrane, fluids in the cochlear duct and the organ of Corti. Such a resonance is not that simple. Another difficulty consists in that Bekesy and other researchers investigated into the resonance by using simple harmonic tones of continuous intensity. In Nature such simple harmonic tones do not occur. Simple tones, if any, may occur only in labs of audiology. We receive multitones of variable intensity and variable frequency, and in addition, with a package of harmonic tones.

4. While looking at a large, colorful pictures we can discern all colors simultaneously. A similar situation is with our hearing. While listening to the Large Symphony Orchestra, we can hear all sounds at the same time. Not only loud, but also silent and the most silent ones. If such silent sounds should need a time-consuming mechanical amplification; information about such silent sounds would reach the center in considerable delay. Successively, further information is conveyed. There would be generated unacceptable chaos. Such a split of signal with a package of information is unlikely to occur.

5. It is assumed that an OHC's contraction generates a sound amplification of 40-50 dB. A 20 dB sound, of an amplitude of 0.8 nm, after an amplification by 50 dB, will reach 70 dB and is still heard as silent – but is 7 million times louder than the threshold tone. Should be assumed that no amplification under 50 dB has been generated, it means that the wave amplitude is 100,000 times higher than the threshold value. It is still heard as a silent wave.

6. If silent tones should be amplified, there must be a mechanism to evaluate which tones ought to be amplified and which not.

Moreover, upon receiving multitones with numerus harmonics with different frequencies, single time resonances must occur at very many spots on the basilemma. There is concern whether resonance sections do not interfere one with another or suppress adjacent sections. Can quickly changing frequencies generate resonance and transmit information accurately? How is loud information encoded when mixed up with a piece of silent, delayed or amplified one?

7. The energy of a sound wave is transmitted to the receptor and quantified in such a form and received and conveyed to the gating mechanism of mechanosensitive channels. Hence a question: in what way is an OHC's contraction quantified? By pulling at the basilemma? By flows of fluids? Inclination of auditory cell hairs? Operation of cadherin fibers? It is important in view of precision, speed and the mechanism of coding auditory information.

8. So that a silent signal may be amplified mechanically through an OHC's contraction and basilemma pull-up, it must first reach a receptor, then cross the excitability threshold, and eventually cause positive potassium ions to flow into the negative interior of an auditory cell, which triggers off OHC's depolarization. The result of depolarization is an inflow of Ca^{++} to the cell and further transformations leading to the production, transport and excretion of a transmitter into a synapse, which results in the generation of post-synaptic excitation potential. This signal is led to the nervous cells of the auditory ganglion, where action potential is generated and conveyed to the central nervous system through the auditory nerve.

Therefore, the center receives information on a silent signal. The OHC has afferent and efferent innervation. In the case of a doubtful procedure – in compliance with the theory of traveling wave – viz. amplifications of such a signal, will the center receive another delayed information with completely new information? It is illogic and rather unlikely. Impossible. Nature would not accept it.

9. The methods for calculating proper vibrations as postulated by Bekesy is very problematic and are in conflict with the anatomy of the internal ear and physics, too. Bekesy assumed that the basilemma is an independent formation vibrating in the air. Bekesy prepared a very thin film of basilemma, changed its dimensions, cut into millimeter-sized sections and tested its elasticity with a blunt needle loaded with 1 ml of water. Basing thereupon he assumed that human basilemma has proper vibrations in an interval from 16 Hz to 20 kHz. But mammals can hear sounds up to 100 kHz, and bats – even up to 200 kHz. Objective investigations into proper vibrations of human tissues do not exceed 100 Hz. The basilemma is loaded with the organ of Corti with fluid spaces, nerves and a strip of connective tissue from the tympanic duct. Vibrations in water are intensively suppressed.

10. Each vibrating mass that has a speed and acceleration is subject to the law of inertia. The vibrating parts of the middle and internal ear are not excluded from this law of physics. Inertia is proportional to squared frequency, which is crucial in the reception of auditory information on the path through cochlear fluids, the basilemma, motions of fluids, inclination of auditory cell hairs and pulling at cadherin fibers in the tip-links mechanism. A sound wave has no mass, and thus, it is to subject to the law of inertia. If one should assume the path of a sound wave to the receptor through the bony housing of the cochlea, there will be no problem with the cochlea' inertia nor with a quantified transmission of auditory information encoded in a sound wave.

11. The theory assumes that in the time of depolarization, the OHC is reduced by 3-5%.The length of auditory cells is diversified, viz. from 20 µm in the basilemma's region to up to 100 µm nearby the cupula. If all cells during the depolarization contract on an average by 4%, low tones are amplified 5 times more than high tones. Moreover, the contraction of the whole cell needs depolarization of the entire cell, viz. a simultaneous action of all tension-dependent cell channels is decisive for depolarization. Depolarization will need time, a contraction occurs in microseconds, but in the cycle ion channels, apart from the excitation time will have a refraction time in which case they are insensitive to excitation. In short, an OHC's contraction to amplify a silent tone will need some time when both the receptor and an auditory cell are insensitive to other sound waves that arrive unceasingly. One should ask what happens to that lost information? The time in which a cell will regain in full the ability to generate new action potential is around 10 ms. Ion channels become activated gradually, since there is absolute refraction when the channels are sensitive to no stimulus and relative refraction when the channels are insensitive to a strong stimulus. So, a continuous quantified transmission and reception of information by a receptor is rather out of the question. It can be supposed that there is no simultaneous contraction of the entire auditory cell. A signal transmitted to afferent synapses must be compatible with that in a sound wave. The working cycle of tension-dependent ion channels excludes one-time OHC frequency of contractions up to 100,000 /s [mammals) and continuous, precise transmission of quantified energy of a signal.

Silent signal is amplified, but according to intracellular amplification and upon reception of a signal by the receptor. Quantified sound wave energy is received by a receptor's molecules susceptive to the action of sound wave energy (sound sensitive molecules). This is a group of molecules, mainly protein ones, consisting of thousands of atoms. Each atom is provided with bond having different lengths and oscillations, a sphere of electrons and vibrations around their own axes. There are also bonds among those and other molecules. A molecule's energy is a sum of energies of all atoms. At rest, a molecule assumes the lowest quantified level of energy. External energy of a sound wave, according to the resonance principle, increases a molecule's energy stepwise (energy is quantified) by one or more levels. This energy, within a time of 10^{-12} s , is transmitted on to an adjacent molecule which, while increasing its energy, is subject to conformation changes which, in turn, are able to perform a work consisting in a regulation of channels of mechanosensitive potassium ions. The maximum opening of a channel causes 6,000 potassium ions to pass from endolymph into an acoustic cell. Positive potassium ions bring about depolarization of the negative interior of the auditory cell. Tension-dependent sodium, potassium, calcium and chlorine ion channels in the cell wall will start to open. Very important are not only calcium ions which flow into a cell at the depolarization time, but also those released

from the stores of the endoplasmic reticulum, mitochondria and the karyon. The role of calcium in an auditory cell is very diversified. Calcium is a regulator of many intracellular mechanisms, but its most important tasks include participation in the transmission of intracellular information, its amplification and distribution. The role of calcium in transmitting information consists in that Ca^{2+} influences such enzymes as adenylyl cyclase, phosphodiesterase A_2 phospholipase and protein kinase. A [PKA]. Calcium ions are another transmitter and they also participate in the generation of other transmitters, like cAMP, cGMP, IP_3 and DAG. **Intracellular amplification** acts through the other transmitters and Ca^{2+} - sensing receptor proteins. When bonded with calcium, those proteins change their biological properties, influence other proteins through cellular reactions. There is a large number of Ca^{2+}- sensing receptor proteins, but a particular role is played by calmodulin. This protein, made of 148 amino acids, has four spots to bond Ca^{2+} ions. The addition of successive ions to the bonding domains triggers off conformation changes in a calmodulin molecule, increasing its bonding capacity with enzyme particles. Enzymes are activated after addition of at least three Ca^{2+} ions. At a calcium level in the cell of an order of 10^{-6} mol/l, the activity of calmodulin complex with calcium is 100,000 higher that the activity of calmodulin itself. This is one of items on the intracellular amplification. This complex works directly upon enzymes, or directly through stimulating calmodulin dependent protein kinases which, through phosphorylation of enzymatic proteins, render them active. The stage of information transmission in an auditory cell, connected with calmodulin, causes a signal splitting in various directions.

An amplified signal travels on towards in the concentric direction, but simultaneously calmodulin activates a large number of the so-called constitutive processes viz. occurring in a cell not excited, like: The effect upon the production and decomposition of cAMP and cGMP, through the activation of phosphodiesterase or adenylyl cyclase according to the calcium level in a cell. Activation of kinases of protein phosphatases. Regulation of the calcium level through the activation of the calcium pump. Effect upon the cellular metabolism through activation of phosphorylase kinase A and activation of glycogen synthase kinase. Effect upon contractions exerted by muscular and non-muscular cells through the activation of cAMP-independent kinase of light chains of myosin.

Due to phosphorylation of specific membrane proteins, calmodulin influences the cellular excretion (exocytosis).

Other cellular proteins whose action depends on the calcium level in the cell are gelsolin, troponin C and parvolbumin. When bonded with calcium, they activate other cellular proteins. Also, the activity of certain enzymes intensifies in the presence of calcium ions. This group includes mitochondrial enzymes, like pyruvate dehydrogenase and alpha-ketoglutaric acid dehydrogenase. If a signal is too weak to reach the central nervous system, it is amplified on the molecular level at the very auditory cell, on the auditory nerve and successive synapses where information is decoded, new information is added, where information is encoded and transmitted further. Along the path, amplification draws the energy from ATP for the operation of channels of sodium and potassium ions.