



Analyses Related to the Hearing

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Opinion

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Opinion

I intend to present a few interesting issues for analysis: We deliver a 20 dB 100 Hz tone for 20 milliseconds. The ear receives it; a low tone has to be enhanced, since such a signal energy is too weak to reach the centre. There are doubts whether here active is only a mechanical amplification through an OHC contraction.

For simplification's sake, assuming 1,500 m/s as the sound speed in fluid, this means two periods of 100 Hz wave within a 20 msec. time, the wave length being 15m.

The resonance of the basilaris membrana for such a frequency occurs, in the best case, nearby the cupula. In what way can such a wave length fits on the basilaris membrana (approx. 37 mm). Resonance in any place of the basilaris membrana is unlikely to occur. Actually, the sound wave will disappear on the way to the cupula.

It was proved that the sound pressure in the vestibule duct is higher than the pressure in the eardrum meatus, which is to thought of to generate a traveling wave. But the pressure in the vestibule is considerably higher than in the eardrum, and the first wave is shown with its crest to the vestibule. It should change the distance from the basilaris membrana to the tectorial membrane and alter the fluid flow in the infratectorial space. This is the physical mechanism, responsible for the bending hairs of the auditory cells. This mechanism has to encode precisely all information contained in a sound wave. If a sound wave clearly diminishes from the oval window to the cupula, why does the traveling wave on the basilar membrane increase on the way to the cupula in the absence of resonance? A sound wave in the fluid decreases 500 times, whereas a traveling wave increases.

This 100 Hz wave does not go into resonance with the basilar membrane on the 3/4th of length from the oval window, since the basilar membrane parameters are completely different and proper vibrations of this membrane do not comply with the wave length and sound wave vibrations. Moreover, there are objections to the calculations of basilar membrane free vibrations. It is very doubtful whether the entire organ of Corti, lying on the basilar membrane which rests on a thick stratum of connective tissue, and in addition, the entire vibrating conglomerate immersed in a fluid with sound attenuation properties, might have such a resonance compliance? The entire calculations for the inner ear were made for a cochlea straightened to a straight tube, which changes the physics of the inner ear. Prepared was also the basilar membrane without the organ of Corti lying thereupon and the connective surface on the inferior surface, and for such a thin formation determined were proper vibrations. The entire conglomerate vibrates. There is no explanation why after reaching the maximum of the traveling wave on the basilar membrane for 100 Hz the transverse wave suddenly disappears. What happens to the energy of this wave? What causes such a sudden loss of the wave energy? How does the wave change its phase during the course? Can 2 wave periods so much increase the amplitude of the basilar membrane transverse wave? Instead, at sounds lasting for less than 1/10 ms there occurs only one wave period. The ear receives a signal, recognizes the frequency. One wave period is unable to cause either resonance or a traveling wave [1].

If the sound wave energy is amplified due to resonance, only the basilar membrane transverse wave is to move the cochlear fluids, to bring the tectorial membrane closer and farther, to bend hair cells, to cause OHC's depolarization and OHC's contraction, to pull the basilar membrane, to increase the amplitude of the transverse wave, to bring the tectorial membrane closer again, to cause movements of fluids maintaining the information coding, to bend IHC hairs and

to bring about IHC's depolarization. Only now will occur the action potential which is conveyed to the center.

What purpose does an amplification of the basilar membrane vibrations serve, concerning that this primary wave with auditory sensory information is no longer on the basilar membrane? At a speed of wave in fluid equal to 1,500 m/sec, such a wave travels a few dozen meters during the procedures. The wave signal lasts for 20 ms. And if the signal lasts for 1/10 ms and is audible, how long does the signal amplification last? This mechanical wave amplification lasts many times longer. A sound wave, primary, does not wait for the energy of amplifications. It is hard to explain how that amplified signal reaches the receptor and the center for being audible. OHC do not have afferent innervation (according to the theory of traveling wave), they serve only as an amplifier for the IHC. In line with this theory, a silent sound is not received by IHC, and must be amplified by OHC.

Is it physically possible that OHC which change their lengths might pull and push the basilar membrane? Is it possible, since auditory cells do not lie directly on the basilar membrane? They cushion the support cells.

Mechanical amplification is simpler in the case of transmitting simple tones which need amplifying. It looks different in the case of sounds of various frequencies and intensities. Here is also the problem with the basilar membrane, viz. how it copes with the resolution of frequencies, and then with the amplification of respective, various tones, simultaneously quiet and loud. Moreover, in the case of continuous sounds amplified is another alien wave appearing on the basilar membrane and causing the largest displacement after the said elapsing milliseconds of mechanical amplification. When tones are silent and loud, are all of them amplified? Is there any segregation? Who manages this? They write this is a compression of the amplification, but who manages this? Hopf bifurcation? Rather unlikely. The basilar membrane is not provided with any instruments for the regulation of amplification.

How is a split signal transmitted to the center? Only in the brain is it combined. So amplified tones are perhaps conveyed together with other tones? A uniform signal is transmitted to the center with a time delay. How will the routes of transmission, intermediate synapses cope with this transmission? The acknowledgement of existence of a perfect system of intracellular amplification allows avoiding problems, perhaps artificial, superfluous, related to the signal amplification. And so, it happens in the twin organ of sight, where the energy of 1 photon can already generate the receptor's reaction.

A photon's energy is so small that in order for it to reach

the center it has to be amplified more than 1,000 times on the way. But not mechanically. Molecular biology was not known at all or little was known about it when amplification in the hearing organ was ascertained, and therefore, mechanics itself was invented. Nowadays it is very difficult to withdraw therefrom

The second issue, not dealt with at all, concerns the hearing in the case when the tympanic membrane is maintained, but the chain of middle ear ossicles is broken, or without the tympanic membrane with the broken chain of middle ear ossicles. An interruption of the chain of ossicles or the lack of the tympanic membrane causes a hearing loss of 50-60 dB. The question is the hearing mechanism in such a case, the signal path to the receptor. It is assumed that air vibrations of the tympanic membrane transmit the energy to the stapes plate, and then the process of hearing is identical as in the case of air conduction. But is this really true?

There is no amplification typical of the inner ear. The impedance of the bone, viz. the stapes plate exceeds the impedance of water where it is assumed that 99.9% of energy will be reflected. Hence, a conclusion that in the absence of the inner ear ossicles more than 99.9% of energy of a sound wave is reflected from the stapes plate.

Moreover, even if this 1/1000th part of energy is conveyed to the ventricle duct, it will be subject to the same fading rule on its path to the cupula as other sound waves, amplified in the middle ear. If on the way from the external auditory meatus to the round window the amplitude of the 8000 Hz and 90 dB wave decreases 1,000 times, the energy will decrease 1,000,000 times. Therefore, on its path to the cupula (halfway) the amplitude will disappear 500 times. Can't such an energy of the wave of the 1/1000th part falling on the stapes plate (without middle ear ossicles) and disappearing approx. 500 times in the cochlear fluids reach an OHC receptor? It is unlikely to generate a traveling wave on the basilar membrane, to put the OHC cells out of dynamic balance and to trigger off OHC depolarization. This mechanism, according to the physics rules, excludes the hearing of silent tones. We can hear silent whispers pleasing to the ear, wind rustling and singing birds at dawn. There must be some logical solution.

There is one solution described in the 'Submolecular Theory of Hearing'. It is known that a sound wave in the air of the tympanic cavity acts not only upon the oval window, but also upon the round window. This susceptibility to vibrations of the round window is 20 times higher than of the oval window. Therefore, what is the principle of conveying the energy to the inner ear through the oval, not through the round window? In this case, does the traveling wave run from the round to the oval window? It fades exactly

the same according to the rule. It is hard to explain what the amplification looks like here. How is the receptor stimulated? What about the resolution of sounds? No explanation available. Nevertheless, this is heard.

In the explanation helpful is the hearing of a fetus in its mother's womb, where the child can hear as soon as from the 2nd half of gestation and react to voices from outside. The most pleasant is its mother's voice; the fetus can hear not only the voice but also the mother's heartbeat, vesicular murmurs, resonance of swallowing or peristalsis.

The kid's external ear is inactive, the inner ear is inactive, filled with fluid, the head does not touch the bones better conducting sounds. It hears and reacts, remembers also its mother's voice after birth. Its can also hear its father's voice but on condition the father lays his palm of the hand or head accurately on the pregnant abdomen, while speaking. The child also reacts to such a voice.

In what way does the energy of those waves reach the inner ear - the receptor? There is no other possibility, but through tissues with various conductivities and fluids surrounding the child's head, and eventually the cranial bone. This is an irrefutable proof of sound transmission through tissues.

The tympanic membrane in the healthy ear vibrates, which is beyond any doubt. This membrane is fastened in a ring fixing it to the bone. This second element to carry vibrations from the tympanic membrane to the bone is the

muscle, viz. the tympanic membrane tensor, quite massif and adhering to the bone. The third element may be ossicles, when the continuity is interrupted, but the incus and the malleus are connected with the walls through osseous ligaments. The distances of those ossicles from the bony wall are very short.

Can such distances be compared to the distances and obstacles in the fetus's hearing in the mother's womb? The wave energy is conveyed to the bone of the labyrinth housing, where sound waves travel at a speed of 3000-4000 m/s. But here is a problem – fundamental theoretical differences. It is hard to understand why the energy containing encoded information does not pursue the goal, viz. the receptor? And all to be done now is to generate a traveling wave on the basilar membrane, to induce movements of the tectorial membrane, flows of cochlear fluids, bending the auditory cell hairs and their tightening for opening and closing the mechanically activated potassium channels? What purpose does those complex ion channels of the auditory receptor are mechanically activated receptors. The energy of a sound wave is mechanical, and for millions of years organisms became specialized in the reception of this energy in receptor cells of the hearing organ, like in the twin organ of sight receiving portions of light energy as high as even of one photon. Decisive are molecular processes, not the mechanics itself.

Reference

1. Jan Myjkowski (2021) Submolecular Theory of Hearing. The New American J Med 2(2): 1-3.

