



Fallacies in Bekesy's Travelling Wave Theory

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Abstract

Improved and complemented over decades, the travelling wave theory proposed in 1928 by Bekesy still contains ambiguities and logical inconsistencies. The 1961 Nobel Prize awarded to Bekesy for explaining the mechanisms of hearing provides no justification for further existence of the travelling wave theory in its present form.

This paper presents reservations made regarding key points of the theory of hearing that continues to be recognized. Discrepancies concerning the reception, processing, and transmission of auditory information are discussed. A different path of the signal to the receptor is suggested. This entails a change in the amplification of the signal and the conversion of sound wave energy to an auditory cell response.

The transfer of sound wave energy coding auditory information is progressive according to physics and quantum chemistry. The transfer of auditory information to the receptor by means of a travelling wave, the cochlear fluid and the tip-links mechanism is subjected to critical assessment.

Keywords: Impedance; Resonance; Coding; Signal Amplification

Analysis of the mechanisms of hearing

Upon hitting any object, sound waves are reflected, absorbed, or transmitted. The angle of reflection is equal to the angle of incidence at which they hit a given surface. Energy absorption is determined by the angle of incidence. The human auricle has a diverse, cavernous surface that is conducive to wave energy absorption. Reflected waves are dispersed with only a small part of the reflected rays directed to the external auditory canal [1].

The waves absorbed by the auricle are transmitted by the skin of the auricle to the auricular cartilage. The material constant of the cartilage called specific acoustic resistance is lower for the cartilage than for the skin and the connective tissue. Lower resistance means that a wave is transmitted quicker. On its way from the air to the auricle, a sound wave changes direction in which the wave propagates due to the difference in the velocity of the wave in that environment. The frequency of the transferred wave remains unchanged.

The energy of the acoustic wave absorbed by the auricle is transmitted onto the surroundings, the temporal bone, in line with the law of acoustics, "each point reached by the sound wave becomes the source of a new sound wave".

The vibrations transmitted by the auditory ossicles of the middle ear are channelled by ligaments and joints to the bone capsule of the tympanic membrane. The most significant impact on the transmission of sound wave energy onto the bone labyrinth of the cochlea is exerted by the stapedial footplate vibrating in the oval window.

According to vibrometric studies, a 90 dB (amplitude of 500 nm) sound wave hitting the tympanic membrane on the side of the tympanic cavity has the amplitude of 80 dB (amplitude of 100 nm). It is hard to agree with the thesis that a wave that hits the tympanic membrane or a wave that is transmitted by the ossicles of

the tympanic cavity upon reaching the fluid of the vestibular duct is amplified 44 times = 33 dB [2]. The questionability of this thesis for such amplification is evidenced by vibrometric studies of wave amplitude on the stapedial footplate on the side of the inner ear and in the initial section of the fluid of the vestibular duct.

The studies were conducted for a 90 dB (500 nm) input wave [3,4]

Frequency—the base ----- the vestibule
 1000 Hz-----5.09 nm-----0.275 nm
 4000 Hz-----1.37 nm-----0.00886 nm
 8000 Hz-----0.0905 nm-----0.00153 nm

With constant intensity of a wave that is hitting the tympanic membrane, a change in frequency to high causes a drastic decrease in high-frequency wave energy measured in the cochlear fluid. The reason behind this disproportion lies in the structure of the oval window, the mechanics of the annular ligament, and the rocking motion of the stapes at high frequencies. During rocking motion of the stapes, vibrations of the stapedial footplate transmit sound wave energy via the annular ligament to the bone of the oval window capsule. High frequencies trigger rocking motion of the malleus caused by the structure of the tympanic membrane that are transmitted to the stapes. The sound wave energy transferred onto the bone labyrinth of the cochlea is subjected to constructive interference with the energy of waves previously transmitted from the auricle and the ossicles of the middle ear onto the bone. The combined wave energy is heading straight to the receptor at the speed of 4000 m/s.

The proof lies in the time for this distance, 1.5 ms, from the external auditory canal to the point at which the EcoG measurement was taken.

Bekesy assumed that upon hitting water, sound wave energy is reflected in 99.9%. According to his supposition, a sound wave that is heading from the air to the cochlear fluid is reflected to the same extent. A sound wave inside the ear does not hit water directly. It hits the flexible tympanic membrane of low impedance that absorbs and transfers up to 80% of the incident sound wave energy. This fact is confirmed by laser Doppler vibrometry. For a wave of 90 dB, 3 kHz (amplitude of 500 nm) hitting the tympanic membrane, testing on the tympanic cavity side showed a wave with the amplitude of 100 nm, corresponding to 80 dB. It is hard to agree that in the middle ear, this wave is amplified 44 times, that is, by 33 dB. The difference in the area of the tympanic membrane and

the stapedial footplate, with the former being 17 times bigger, allegedly amplifies the wave energy 17 times. In stapedotomy, there is a difference in the area of the tympanic membrane and the active surface of the piston whose diameter is 0.4 mm, with the former being 100 times bigger, and no wave amplification occurs. With the piston diameter of 0.6 mm, the area is 50 times bigger and there is no wave amplification either [5].

Sound wave energy is proportional to the wave amplitude squared. If the lever mechanism of the middle ear reduces wave amplitude in a ratio of 1.3 : 1, then it can increase the strength but it does not increase the amplitude of the wave, it does not increase the energy transmitted to the sound wave.

Up to the frequency of ca. 2400 Hz, sound wave energy transferred onto the stapedial footplate generates piston motion of the stapedial footplate and excellent transmission of low frequency sounds. At medium frequencies, the footplate vibrates, thus generating rocking motion along the transverse axis of the footplate. At high frequencies, the footplate vibrates along the longitudinal axis of the footplate. This is rocking motion of the footplate.

Please note that during rocking motion, at the same time, half of the footplate is generating a forward wave movement while the other half of the footplate is generating backward movement, often of the same intensity, creating a wave that propagates in the opposite direction. These waves can be subjected to destructive interference. They cannot further transfer the information encoded in the wave properly, as it becomes destroyed, friction and dampening of the wave energy occurs. The resulting wave cannot generate a proper travelling wave on the basilar membrane. The proof supporting this thesis is the absence of transmission of high-frequency sounds following stapedotomy since the piston is responsible solely for transmitting low frequency sounds down to a certain threshold.

There are no rocking motions of the stapedial footplate that would transfer high-frequency sounds onto the bone labyrinth of the cochlea.

Another questionable thesis in Bekesy's theory claims that there is resonance of the longitudinal wave in the cochlear fluid and the transverse wave of the basilar membrane in the transfer of auditory information. Due to inconsistent wave direction and frequency, particularly in other mammals able to hear sounds up to 100 kHz and in birds, which stems from the frequency of natural vibrations of basilar membranes, the wave resonance cannot occur.

Inside the human ear, there is a high-degree discrepancy regarding the velocity of both waves. The longitudinal wave that propagates at the speed of 1450 m/s in 0.1 ms covers the distance of 1450 mm and transmits the auditory information onto the wave that runs on the basilar membrane at the speed of 2-50 m/s, covering the distance of 0.9-5 mm in 0.1 ms.

The wave velocity in the fluid is constant, whereas the travelling wave velocity for each frequency is different. For low frequency sounds, the wave on the basilar membrane is over 1000 times slower than the incident wave. This high decompression of transmitted information and varying velocity depending on frequency renders precise transmission of the information to the receptor impossible. Additionally, according to the travelling wave theory, this transmission is disturbed by the basilar membrane being pulled at by OHC contractions that amplify soft tones [6].

There is some uncertainty – are OHC contractions that are not related to soft tone amplification pulling at the basilar membrane during a contraction, too?

There is no explanation as to where the peak of a running wave is formed in the case of multi-tones with numerous harmonics. Phase shifts and the quantitative must be transmitted too. This information must be transmitted to the receptor. It is difficult to agree with the thesis that further towards the receptor, this information is coded by means of flows of the cochlear fluid or the bending of hair cells in the tip-links mechanism. Another issue arises when this information transmitted by a travelling wave overlaps with the information of a previously received low-tone wave amplified by OHC contractions which, likewise, has harmonics and phase shifts. A conflict of interests takes place – which wave should be further propagated when these waves overlap.

Resonance is conditioned on the principle that the energy of the incident wave is greater than the energy of the dampening of the reflected wave. Weighted by the organ of Corti, with fluid spaces and connective tissue on the lower surface of the basilar membrane, vibrating in the cochlear fluid, the basilar membrane is subjected to a significant dampening of the wave energy. This dampening is several times greater than the threshold wave energy that reaches the receptor, and is audible. The ear receives short sounds that last for a tenth of a millisecond [7,8].

Resonance is a process that takes place in time. One period of a wave cannot effectively transfer information by means of resonance.

Due to all these facts, it is hard to accept that information transfer occurs in this manner, as it pertains not only to amplitude and frequency but also to the harmonics, phase shifts and the quantitative. All the more so given that the transfer of the information contained in a sound wave is a transfer of differences in pressure in an environment, without the environment itself shifting or moving in any way. An energy transfer occurs progressively in line with physics and quantum chemistry [9]. A continuous transfer, which is consistent with classical physics, does not allow auditory information to be fully transferred.

In the case of a cochlear implant due to partial deafness the basilar membrane is either immobilized or its movements are disabled, which has no effect on the transfer of the information to the receptor. Resonance of the longitudinal wave with a transverse wave of the basilar membrane is impossible. There is no travelling wave or flow of the cochlear fluid. The hair cells are not bent; the tip-links mechanism is not working. The auditory information is received by the receptor. Logic indicates that there is a different path that an auditory signal takes to reach the receptor other than the one described in the theory. It is the path via the bone labyrinth of the cochlea.

According to the theory, soft tones are amplified by OHC contractions ranging from 40 to 50 dB. This claim is highly unlikely for a number of reasons. Mechanical amplification by means of this method pertains solely to received waves when the information is already heading towards the centre. During this amplification another wave is already on the basilar membrane that is being pulled up by an OHC contraction; the energy of that wave is distorted, altered. It is difficult to determine which bit of information is forwarded to the receptor. Is it the amplified wave? Or, is it the distorted wave overlapping with the amplified wave? If the problem pertains to a multitone, then loud tones are received and transmitted to the centre whereas soft tones are separated, amplified and transferred in a package along with the other data to the centre with a delay. According to the theory, soft tones such as 20 dB are amplified 44 times (by 33 dB) in the middle ear, then amplified further by 40-50 dB in the inner ear and, consequently, they are audible as soft tones 20 dB, and additionally, they are audible along with loud tones. It is an illogical concept that is unacceptable.

If the sound intensity of 10 dB with the amplitude of 0.05 nm is increased by 40 dB, then the amplitude becomes 100 times higher and the sound intensity becomes 10.000 times higher. The power

of this sound increases from 10^{-11} W/m^2 to 10^{-8} W/m^2 . It is difficult to explain the fact that these soft tones amplified 10,000 times are still heard as soft tones, equal to 10 dB.

There is an auditory signal amplification mechanism which is intracellular [10], molecular; the very same as in other sense organs. Inside an auditory cell, the energy that encodes auditory information that is too weak for this information to reach the centre becomes amplified. The mechanism of this amplification is well-known and has been described.

One of the foundations of the travelling wave theory is the hydrodynamics of the cochlear fluid. There are descriptions of the flows of the fluid, whirls in the fluid (?), typical of turbulent flows, information transfer through the waves of the cochlear fluid that move hair cells [11].

Vibrometric studies have shown that a sound wave propagated through the cochlear fluid towards the round window fades. The energy loss is determined by the frequency and intensity of the wave. For 1000 Hz and 90 dB with the input amplitude of 500 nm, the amplitude of the wave measured at the round window is 0.5 nm. Assuming that the path to the receptor does not lead through the round window, the part of the path to the cochlear cupula for soft tones is the path to the receptor. The energy loss on this section can be assumed as 100-200 times.

A healthy ear receives threshold sounds of the amplitude equal to 0.01 nm. If this wave energy fades 100 times on its way towards the receptor, then the amplitude of this wave is 0.0001 nm. According to the theory, the wave received by the receptor by means of the tip-links mechanism allegedly bends or leans hair cells whose hair diameter is 100 nm. A million times smaller sound wave (bo nie przepływ płynu) bends or leans thick hair cells, and additionally transfers all the information contained in the sound wave. It is highly unlikely that a 1 cm thick twig could be used for bending or leaning a tree whose diameter is 10 m at the frequency of up to 100 Hz, with any transfer of encoded auditory information or any melody additionally related to this tree bending.

Conclusions

These fallacies are not part of the submolecular theory of hearing [12]. The essence of this theory lies in recognizing that the route of the signal to the receptor runs through the bone labyrinth of the cochlea. The auditory information is transferred by means of encoded energy in a sound wave transmitted in connective tissue,

bones, or the cochlear fluid. The mechanical energy encoded in the sound wave acts directly on the particles of the auditory receptor called sound-sensitive molecules, sensitive to that energy which is a stimulus adequate for the hearing organ receptor. Conformational changes in molecules take place leading to the formation of conformers [9,13]. Owing to the change in the dimensions of these molecules they can carry out mechanically-activated potassium ion gating of the walls of an auditory cell. The cellular membrane of hair cells is the membrane of an auditory cell. The inflow of positive potassium ions controlled with sound wave energy initiates depolarization of the auditory cell and further chain reaction of molecular alterations in the auditory cell, leading to synthesising and secreting a transmitter to synapses with dendrite terminations of the spiral ganglion cells, where the action potential of the vestibulocochlear nerve transmitted to the centre is created. All energy conversions in the receptor, the auditory cell, and the synapses take place on a molecular level and an electron level. The mechanism for amplifying the low tone signal whose energy level is too low to ensure a transfer to the centre operates in the auditory cell itself; it is well-known and has been described. The transfer of auditory information is related to the phenomenon of temporal summation and spatial summation, as well as pre-synaptic inhibition and centrifugal inhibition. Receptor fields are significant. The submolecular theory acknowledges tonotopy that has been known for 100 years. It does not acknowledge the significance of the basilar membrane, the travelling wave and the functioning of the tip-links mechanisms.

Billions of beings on Earth have no cochlear fluid, basilar membrane and the tip-links mechanism and they can hear perfectly regardless. This is unquestionable evidence that there is a molecular mechanism that transfers the sound wave energy into auditory cell response and the action potential of the vestibulocochlear nerve containing the exact same information as in a sound wave.

Bibliography

1. Szymański M., *et al.* "Vibration of the human tympanic membrane measured with Laser Doppler Vibrometer". *Otolaryngologia Polska* 69.2 (2009): 182-185.
2. Śliwińska-Kowalska M. *Audiologia Kliniczna*, Mediton, Łódź (2005): 32-33.
3. Kwacz M., *et al.* "A three-dimensional finite element model of round window membrane vibration before and after stapedotomy surgery". *Biomed Model Mechanobiology* 12.6 (2013): 1243-1261.

4. Wysocki J., *et al.* "Comparison of round window membrana mechanics before and after experimental stapedotomy". *The Laryngoscope* 121 (9 (2011): 1958-1964.
5. Kaźmierczak W., *et al.* "The result of operational otosclerosis treatment after stapedotomy". *Otolaryngologia Polska* 67 (2013): 164-169.
6. Dong W and Olson E. "Detection of Cochlear Amplification and Its Activation". *Bio Physical Journal* 105.4 (2013): 1067-1078.
7. Martinson K., *et al.* "Dyskryminacja czasu trwania ultrakrótkich impulsów akustycznych". *Postępy Akustyki, Otwarte Seminarium Akustyki, Instytut Fizyki Jądrowej, Kraków* (2018).
8. Majka M., *et al.* "Subsekundowe impulsy akustyczne: Wysokość skuteczna i prawo Webera-Fechnera w różnicowaniu czasów trwania". *Instytut Fizyki Jądrowej PAN, Kraków* (2014).
9. Pielak L. "Idee chemii kwantowej 2022". *PWN Warsaw* (2022): 1300.
10. Myjkowski J. "Transforming and transmitting auditory information". *Otolaryngologia Polska* 58.2 (2004): 377-383.
11. Fettiplace R. "Hair cell transduction, tuning and synaptic transmission in the mammalian cochlea". *Comprehensive Physiology* 7.4 (2017): 1197-1227.
12. Myjkowski J. "Submolecular Theory of Hearing". *HSOA Journal of Otolaryngology, Head and Neck Surgery* 8 (2023): 069.
13. Myjkowski J. "Mechanoreceptor of the Hearing Organ". *American Journal of Biomedical Science and Research* 1 (2024).