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The vulnerabilities of Bekesy's travelling wave theory

Abstract

The theory of hearing declared in 1928 by George von Bekesy under the name of 'travelling wave theory' fails to explain all the processes related to hearing in accordance with the current state of knowledge, albeit complemented on many occasions. Perfect new research methods of various Centres around the world as well as analyses of published studies allow one to form a new philosophy of hearing. Many beliefs presented as part of the former theory have become a thing of the past. A new vision of hearing has been put forward in a published paper titled *Submolecular Theory of Hearing.*

Keywords: submolecular theory of hearing, sound wave frequency, receptor, Bekesy's methodology

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Introduction

Rocking motions of the stapes

Up to the sound wave frequency of ca. 2400 Hz, the vibrations of the malleus that transfers vibrations to the stapes are consistent with the vibrations of the tympanic membrane.[1] The stapedial footplate generates a piston motion in the oval window. Higher frequencies are transferred by the auditory ossicles the malleus and the incus to the stapes. Differences in the amplitude of vibrations of the tympanic membrane on both sides of the manubrium of the malleus generate rocking motions of the malleus, which are then transferred onto the stapedial footplate via the incus and the ball-and-socket incudostapedial joint. Up to ca. 6000 Hz, the stapedial footplate vibrates along the transverse axis of the footplate. Above this threshold, the footplate vibrates along the longitudinal axis of the footplate. At low frequencies, the piston motion transfers the sound wave energy freely to the cochlear fluid.

Soft tones easily pass through liquids, soft tissues, and bones to the receptor. The occurrence of a rocking motion of the stapedial footplate under high frequencies disturbs the transfer of waves onto the cochlear fluid. When one half of the plate is generating forward motion, in the very same time, the other half of the plate is generating reversed motion of the sound wave with the same frequency. The wave motions with opposite directions and the same frequency undergo destructive interference or a standing wave is formed.

The transfer of auditory information to the centre is distorted. A problem arises as to the formation of a travelling wave on the basilar membrane. The emerging deformed wave is unable to code quantized energy heading to the centre. The inertia of the ossicles of the middle ear plays a role in this process. Vibrating elements that transfer the energy of a sound wave, which have mass in the wave motion are subject to the law of inertia. The stapedectomy procedure eliminates the rocking motion of the stapes that ensure we can hear high-frequency sounds via air-bone conduction by means of the osseous chamber of the cochlea. Since only the piston mechanism is working, hearing of high-frequency sounds does not occur following a stapedectomy.[2]

Fading of wave energy on the way to the receptor

The sound wave that hits on the flexible tympanic membrane is reflected in ca. 20%, whereas ca. 80% is absorbed and transferred onto the ossicles of the middle ear. A sound of 90 dB with the wave amplitude of 500 nm in the outer ear canal was examined. On the side of the tympanic cavity, pressure was recorded at 80 dB – the wave amplitude at 100 nm. The strengthening of the midle ear due to the difference in the surface of the tympanic membrane,[3,4] and the surface of the stapes in the ratio of 55 : 3.2 and the leverage mechanism is supposed to amplify the sound wave by 33 dB compared to the wave that hits the tympanic membrane, which is unacceptable.

In stapedoctomy, the difference in the area of the tympanic membrane and the surface of the piston is 100 : 1 and there is no signal amplification. A tone of 90 dB tested on the entry by means of laser Doppler vibrometry on the stapedial footplate on the side of the vestibulum shows 11.7 nm for 1000 Hz. At 10,000 Hz, the amplitude of the stapes falls to 0.117 nm. The sound wave generated by the stapedial footplate 'runs' through the cochlear fluid to the cochlear cupula via the vestibular duct and next via the tympanic duct to the round window. A wave was examined in the outer ear canal, 90 dB and 800 Hz. = 500 nm. On the round window, the wave amplitude was recorded at 0.5 nm.[5] When the tested wave showed 30 dB on the entry, no wave was recorded on the round window.

Based on wave modelling in a straightened-out cochlea, in line with Bekesy's methodology, a wave in the vestibular duct was calculated with a wave in the outer ear canal at 10 dB and 10 kHz. The wave was 0.00000008747nm. This wave is supposed to be heading to the receptor, making auditory hair cells with a diameter of 100 nm lean on its way. Such a wave cannot trigger depolarization of a hearing cell. Young people hear such a wave. This indicates that there is a different simple path a signal takes to reach the receptor.

Resonance of longitudinal wave with transverse wave of the basilar membrane

The resonance of waves is formed under the following conditions:

a. Consistent frequencies

b. Similar consistence in the plane of operation

c. The energy of the incident wave is greater than the dampening of the reflected wave.

In the ear, these conditions are not fulfilled, particularly in mammals that hear sounds up to 100 kHz that have the same auditory mechanism as a human has. It is not possible for natural vibrations of the basilar membrane in these animals to be lower than 100 kHz. The sound waves in the cochlear fluid are a longitudinal wave. The natural vibration waves of the basilar membrane are a transverse wave in a plane perpendicular to longitudinal waves. The basilar membrane is

weighted with the organ of Corti and connective tissue on the lower surface of the basilar membrane. The entire massive conglomerate vibrates in the cochlear fluid showing significant damping properties. Transmission and coding of all the information contained in a sound wave into a transverse wave is rendered impossible. An issue arises as to the formation of a travelling wave and further transmission of the information to the centre.

Difference in the wave speed on the way to the centre

The sound wave in the cochlear fluid is transmitted at the speed of 1450 m/s. A transverse wave on the basilar membrane is believed by Bekesy to be moving from the oval window to the cochlear cupula at a variable speed ranging from 50 m/s in the area of the base of the cochlea to 2.9 m/s in the cochlear cupula area.[6] The wave frequency and the damping of the sound wave energy reduce the speed for low-frequency tones. If one assumes 10 m/s down to certain low frequency, the speed is 145 times lower than the speed of the sound wave in the fluid. It is impossible for the information contained a 145 cm long longitudinal wave to be recorded on a 1 cm long transverse travelling wave on the basilar membrane. In what manner is this information quantized, coded, and further propagated? Can the energy of a longitudinal wave be compressed 145 times? Is that hearable? Comprehendible?

Discontinued path to the receptor according to Bekesy's theory

In cochlear implant surgery, due to partial deafness,[7] the introduction of electrodes into the tympanic duct immobilizes the basilar membrane. Hearing in the part of the scale from before the surgery remains unaltered. The basilar membrane is inactive, the travelling wave does not occur, the auditory hair cells do not lean, and the auditory signal reaches the receptor. This indicates the existence of a different path that a signal takes to reach the receptor by-passing the cochlear fluid, the basilar membrane, and the tip-link mechanism. This path runs from the auricle through the middle ear to the bone chamber of the cochlea, where the receptor is reached at the speed of 4000 m/s. It involves air and bone conduction.

Difference in the time it takes a signal to reach the receptor

In auditory response testing, one studies the time from when a signal is provided to the outer ear canal to the reading of the wave in electrocochleographic testing (ECoG) or the auditory brainstem responses (ABR). In ECoG testing, the latency time is 1.5 ms.[8] The test can be done by means of an intra-tympanic method and an extra-tympanic method. The result indicates the time after which a response occurred.

In the ABR test, the latency time generated in the distal part of the vestibulocochlear nerve as wave I is 1.9 ms. The time results for all the sections of the path a signal takes to reach a receptor totalled 5-6 ms or more for amplified soft tones.

Splitting of soft and loud tones in the course of amplification

Mechanical amplification of soft tones ranging 40-50 dB by means of the OHC contraction and the pull on the basilar membrane is time consuming. When it comes to multitones comprising soft and loud tones, loud tones are received, and the information is transferred to the centre. Soft tones requiring amplification are split to the path of time-consuming amplification. During this amplification, a different alien sound wave at the speed of 1450 m/s provides a new unknown information of varying amplitude. The amplified soft sounds superimposed on new waves disrupt the coding and the further transfer of the information of two different waves. How does the basilar membrane code these overlapping waves that can have harmonics, phase shifts?

Auditory threshold

The acoustic pressure of sound waves audible to a human ranges from 2 x 10-5Pa to 28 Pa. It is the pressure deviation from the atmospheric pressure of 100,000 Pa. Acoustic pressure – the threshold – for a 1000 Hz wave audible to a young man is 2 x 10-5 Pa. When we convert acoustic pressure to the sound wave amplitude, we get 8 x 10-12 m = 0.008 nm. It is the amplitude of a sound wave that acts on the tympanic membrane in the outer ear canal. If we assume the path of a signal to the receptor involves resonance, the basilar membrane, and the cochlear fluid, then the amplitude of that wave on its way to the cochlear cupula is from 100 to 200 times smaller. The energy of the wave is proportional to the wave deviation squared. A wave amplitude that is many times smaller than the diameter of a hydrogen atom cannot cause a travelling wave. It cannot generate the described movements of the cochlear fluid. It cannot make the auditory hair cells bow nor bend, the diameter of which is from 10,000 to one million times bigger than the amplitude of the sound wave supposed to make these hair cells lean down. The paradox of this situation lies in the fact that the signal whose amplitude is 8 pm reaches the receptor. This is another simple path that a sound wave travels from the middle ear through the cochlear bone chamber to the auditory cell receptor.

Quantization of sound wave energy

The sound wave energy transferred to the receptor by means of the mechanosensitive potassium ion channel gating mechanism cannot have a continuous form in rises and falls of energy. Energy conversion is transferred in portions of energy that is a multiple of the total number of quants of mechanical energy of the sound wave.[9] The energy coded in the sound wave acts on sound-sensitive molecules of the receptor, and causes conformational changes in particles responsible for potassium ion channel gating. The flow of potassium ions to the auditory cell is regulated. The inflow of positive ions into the cell starts depolarizing it if an excitability threshold is exceeded at ca.10 mV of the forming response. Further energy conversions pertain to the auditory cell, synapses, and the path of a signal to the centre. Energy quantization that pertains to the amplitude of the wave, the frequency, the harmonics, phase shifts, and the quantitative of the travelling wave, the flow of liquids, auditory hair cells, and cadherin tip links is impossible. It is possible on the path to the receptor described in Submolecular Theory of Hearing.[10]

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None.

Conflicts of interest

The author declares that there is no conflict of interest to disclose.

References

1. Pruszewicz A. Outline of clinical audiology. Publishing house of the medical university of Warsaw. Karol Marcinkowski in Poznań. 2000;65.
2. Kaźmierczak W, Janiak-Kiszka J, Polak-Osińska K, et al. The result of operational otosclerosis treatment after stapedotomy. *Otolaryngol Pol*. 2013;67(3):164–169.

3. Gillspie P, Muller U. Mechanotransduction by hair cells models molecules and mechanisms. *PMC.* 2009;139(1): 33–44.

4. Hudspeth AJ. Hearing: mechanical amplification in the ear. *Neuron.* 2008;59(4)530–542.

5. Kwacz M, Marek P, Borkowski P, et al. A three-dimensional finite element model of round window membrane vibration before and after stapedotomy surgery. *Biomech Model Mechanobiol*. 2013;12 (6):1243–1261.

6. Fettiplace R. Hair cell transduction, tuning and synaptic transmission in the mammalian cochlear. *Compr Physiol*. 2017;7(4):1197–1227.

7. Skarżyński H. Cochlear implants, pediatric otolaryngology by Danuta Gryczyńska. *Alfa Media Press*. 2005;142–154.

8. Kochanek K, Śliwińska-Kowalska Mariola. Auditory evoked potentials. *Clinical Audiology*. Mediton, Warsaw. 2005;21:163–176.

9. Piela L. New auditory signal pathway to receptor. *PWN Warsaw*. 2022;1300.

10. Myjkowski J. Submolecular theory of hearing. *HSOA J. Otolatyng Head Neck Surg.* 2023;8:69.