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A New Vision of Hearing

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Abbreviations:

Hz: Hertz; kHz: 1000; Hz: kiloHz; Pa: Pascal; pm – pikometr = 10^{-12}m; OHC: Other Cel Hair; IHC: Inner Cel Hair; nm: nanometr = 10^{-9}m; ms: milisekunda = 10^{-3}sekundy; ABR: Auditory Brainstem Respons; BERA: Study of the Auditory Braistem Potentals

1. Abstract

The hearing theory announced in the first half of the 20th century by Georg von Bekesy requires reviewing and updating. There has been a huge advancement in the sciences ever since, which allows to deepen the understanding of processes responsible for reception, processing and transmission of acoustic information. The study paid attention to several issues related to hearing, which require a deeper analysis. A bold hypothesis was stated that the signal in the form of a sound wave does not necessarily have to run through the cochlear fluids and the basilar membrane. It may reach the hearing cells through the bone of the cochlear enclosure. Attention was draw to the meaning of swinging motions of the stirrup and of the inertia in the middle ear and the inner ear. Hearing sounds on the border of the auditory threshold is discused in the paper, Attension was paid to the problems of mechanical amplification of soft tones of different intensity and frequensy, A nev mechanism of signal amplification has been proposed, taking place intercellularly ad the molecular lewel.

The problem of the basement membranę resonanse during the receptione of very short of low frequencywas painted out.

The mechanism of the auditory cells has been described.

The role in hearing sound wave conduction trough soft time and bon eis emphasized.

2. Our Hearing

Hearing is the only sense organ used to perceive and contact the outside world that has not been explored and described closely so far. There is a hearing theory developed in the first half of the 20th century by Prof. Georg von Bekesy [1]. A long time has passed since the announcement of the theory and the science has taken a huge step, which warrants the revision of the age-old hearing theory. Scientific achievements and the possibilities offered by modern studies allow us to see many details which went unnoticed in the past. Analyses lead to formulation of a new picture of the human hearing. The current theory, consolidated for decades, cannot explain many issues regarding the reception, processing and transmission of hearing information. A good example of such issues is the fact that the mechanism of threshold hearing has not been explained. A young individual hears a sound of 1000 Hz with the pressure amplitude of ca. 2.0 x 10^{-5} Pa. The pressure amplitude is proportional to the displacement amplitude. The wave intensity is proportional to the square of the amplitude and the square of the pressure. When the pressure amplitude is converted into the wave displacement amplitude, the value of 8 x 10^{-12} m = 8 pm is achieved. This is a quantity that is many times smaller than the diameter of atoms comprising the basilar membrane. This is the amplitude of the sound wave in the external acoustic meatus. Neglecting impedance and amplification of wave in the middle ear, the amplitude of that wave as transmitted to the vestibular fluid is lower compared to that in the external acoustic meatus, because of inter alia the fact that the leverage in the middle ear reduces the wave amplitude at the ratio of 1.3 to 1.

A reduced wave in the cochlear fluid is to lead to according to the

theory a difference in pressure on both sides of the basilar membrane, which, as Bekesy claimed, is the source of formation of the travelling wave on the basilar membrane. The theory holds that quiet sounds are amplified by 40 dB. There is a contradiction here as signal amplification according to the theory is conditional on receptor excitation and OHC contraction [2]. As a result, there should be pulling of the basilar membrane, increase in the flow of the cochlear fluids, inclination of the hearing cell hairs and, eventually, the excitation of IHC [3]. The theory fails to explain how such a low wave amplitude creates pressure difference on both sides of the basilar membrane. If there is not pressure difference, there is no travelling wave, there is no amplification.

New studies on laser Doppler vibrometry gave a clear answer. The studies were repeated numerous times (Prof. Monika Kwacz with her team) [4]. On preparations of fresh human temporal bones, a 90 dB sound at various frequencies was fed into the external acoustic meatus. The wave amplitude was investigated on the round window, which is loose and flabby and 20 times more prone to vibration than the oval window. For the wave of 90 dB, which corresponds to the wave amplitude of ca. 500 nm, the amplitude of 0.5 nm was found on the round window: the amplitude decreased 1000 times and energy decreased a million times. For a wave of 30 dB, not even a trace of amplitude was found on the round window. This is hard evidence that the energy of the wave in the cochlea disappears abruptly thanks to absorptive damping, reflexive damping, interferential damping and dispersion in the perilymph fluid [5]. On this basis it should be stated that a sound wave having 0.01 nanometre in the external acoustic meatus cannot generate any difference in pressure on both sides of the basilar membrane and form a travelling wave. Going further, the signal cannot be amplified in any way whatsoever because it cannot reach and excite the OHC receptor [6]. This is where a substantial problem emerges as a young individual can hear that sound intensity. There must be a different way of the signal to the receptor, without loss of energy, which bypasses the cochlear fluids. Such a way can only be an osteopneumatic one. The signal from the tympanic membrane is transferred onto the ossicles of the middle ear, which – through the ligaments of those ossicles and the plate of stirrup in the oval window – transfer the signal onto the osseous enclosure of the cochlea; from there, the signal reaches the receptors of the hearing cells in the organ of Corti, which are distributed along the cochlear canals on the basilar membrane. The cells located near the base of the cochlea receive high frequencies. The nearer they are to the cochlear apex, the lower sounds they receive. The hairs of those cells have different length and thickness, different sensitivity to the given frequency. This is conditioned genetically, similarly to the eye, where the sensitivity of cones to the given length of light wave. Conduction of vibrations from the tympanic membrane, the ligaments of the ossicles in the middle ear, especially from the plate of stirrup to the enclosure bone of the cochlea, is possible. Soft tissues conduct sound vibrations. This is proven by the fact that the child hears already at mid-pregnancy. The child hears its mother's voice, heartbeat or peristaltic movement despite the fact that its external acoustic meatus and its middle ear are inactive.

The level of potassium in endolymph is higher than in hearing cells. Negative charges of proteins and a deficit of positive ions in the cell caused by the function of sodium-potassium pumps generates a high electrochemical potential on the cellular membrane. Contrary to the Bekesy's theory, where the opening of ion channels is controlled by pulling cadherin fibrils, it is much more probable that the activation and inactivation gates are controlled by the energy of the sound wave. Channel gates are made from protein molecules, or sound-sensitive molecules. The number of potassium ions moving to the hearing cell depends on the energy coded in the sound wave. Up to 6000 K+ ions can move to the cells during 1 millisecond, causing its depolarisation. If depolarisation crosses the threshold of ca. 10 mV, voltage-dependent calcium and sodium channels located on lateral walls of the hearing cell start to work. Depolarisation increases, calcium flows into the cell, which results in the release of the calcium accumulated in the endoplasmic reticulum, the mitochondria and the cellular nucleus. The calcium binds to calcium-dependent proteins, e.g. calmodulin. After binding to calcium, those proteins increase their activity by a number of times. Calcium, along with cAMP, cGMP, IP3 and DAG, is a transmitter of intracellular information. Calcium has an effect on production, transport and secretion of the transmitter. Cellular depolarisation has an effect on prestin, which is responsible for OHC contraction. The hearing cell functions at 2 levels: the constitutive level, responsible for normal operation of each cell, and the other, regulated level, related to the production, transport and secretion of the transmitter. Those levels cooperate. Along with calmodulin and other calcium-dependent proteins, signal transmitters, calcium participates in intracellular signal amplification. The amplification occurs after the receptor captures the signal.

It is not the basilar membrane that decides on the necessity to amplify. The basilar membrane does not have afferent, efferent or autonomic innervation. It is a connective and supportive tissue, which stems from a different germ layer than the organ of Corti. After every signal, the level of calcium in the cell decreases to the minimum. Calcium pump and ion exchangers are working and calcium is being transferred to the endoplasmic reticulum, the mitochondria and the nucleus. The lower the level of calcium in a cell, the stronger the cell reacts to a new excitation.

Another issue in the hearing theory emerged upon introduction of cochlear implantation surgery. In the case of strong hearing impairment of a half of a sound scale, procedures are performed which entail introduction of 20 electrodes into the tympanic canal through the round window. The electrodes are up to 25 mm long and immobilise the basilar membrane completely. Hearing is im-

proved in the nearly-deaf spectrum without damaging the remaining hearing despite the fact that the basilar membrane becomes deactivated. It indicates that the path of the signal to the receptor is not related to the tympanic membrane. A wave running on the basilar membrane is not significant for hearing at all.

Moreover, Bekesy could not predict that advancements in operative treatment of conductive hearing loss will testify against his theory. Specifically, it is about stapedotomy. It is most often performed in the case of otosclerosis or otospongiosis, with immobilisation of the plate of stirrup in the oval window. A small opening is made in the plate of stirrup and a small prosthesis connected with the long crus of the incus is placed, which transfers vibrations to the vestibular canal. Theoretically the hearing should improve fully. Unfortunately, the improvement in frequency of up to 2 kHz is most often achieved: sometimes it is up to 4 kHz and rarely up to 6 kHz. The procedure, performed perfectly and according to the theory, does not give the expected effect because it is contrary to physiology. A healthy young individual hears up to 20 kHz. Why, then, the same young individual hears up to 2 kHz only after the procedure? This is so as the Mother Nature created a better mechanism allowing direction of higher sounds directly to the receptor, bypassing the cochlear fluids. It might be suspected that this is related to the phenomenon of inertia in the internal middle and inner ear. The inertia does not allow hearing high frequencies with the said method. The fact that there is a different mechanism is proved by the presence of the incudostapedial joint, which is a spheroid joint. Therefore, movements on various planes occur which are not copied by the prosthesis. Furthermore, the plate of stirrup makes only piston-like movements up to 2 kHz. Around 4 kHz, it makes rocking movements along the transverse axis of the plate. Above 6 kHz, the plate makes rocking movements along the longitudinal axis of the plate. The prosthesis becomes inactive: when its half moves forwards, increasing fluid pressure, the other half of the plate reduces that pressure. The inertia of fluids, the basilar membrane, the mass of the organ of Corti and hearing cell hairs excludes the transmission of signal through vibrating elements that have a mass. A sound wave without a mass is not subject to the law of inertia and can be transferred to the receptor through a different way. Only a path through bone remains and bone is a good conductor of sound waves [7]. The conduction speed of a sound wave in bone is ca. 4000 m/s.

The inertia in the wave motion is calculated according to the following formula: $(2n \times \text{wave displacement})^2 \times \text{frequency} \times \text{mass}$ in g/mm x s^2. Wave displacement at one moment equals the amplitude. If we substitute values for high frequencies and intensity into the formula, we will obtain very high values. It is rather hard to assume that such pseudo-forces act in the ear. Therefore, it is highly probable that high frequencies and intensities are transferred to the receptor without any intermediate elements with a mass. Assuming the vibrating mass of the middle ear is 70 mg, then for 80 dB and 10,000 Hz the inertia will be 27,606,880 g/mm x s^2. For 20 dB and 1000 Hz, inertia is 276 g/mm x s^2. In the case of the inner ear, the result will be multiple times higher with a considerably higher mass. There is no information whether the vibrating mass of the middle ear sums up with the vibrating mass of the inner ear with the same sound [8].

There is yet another aspect confirmed by numerous experiments which indicates inconsistencies with the travelling-wave theory. Electrophysiological tests – ABR and BERA – indicated that the time required for the signal to get from the external acoustic meatus to the trunk of the acoustic nerve is 1.5 to 1.9 ms. On the other hand, when all sections of the way which the signal covers according to Bekesy are added, the resulting time is 2-3 times longer. If mechanical amplification through OHC contraction is taken into account, the time becomes even longer.

The experimental evidence is irrefutable [9].

Bekesy makes some combinations that are inconsistent with physiology to prove his own theory. He straightened out the cochlea and did calculations for such a deformed organ. The inner ear developed in the form of the cochlea only in mammals, so there must be a reason behind it. It might be the elongation of the cochlea in a smaller space or possibility to annihilate the energy reaching the ear without pause which cannot be accumulated. Straightening out the cochlea significantly changes its physiology. Furthermore, Bekesy, willing to achieve wave flow on both sides of the basilar membrane, connected the vestibular duct and the cochlear duct, eliminating Reissner's membrane. This contradicts physiology as these ducts have a different electrolyte makeup and the cochlear duct is blind-ended and does not communicate with the tympanic duct.

Bekesy made faulty assumptions to calculate own vibrations of the basilar membrane. If the width of the vestibular duct and the tympanic duct at the base of the cochlea is 3-4 mm, the width of the basilar membrane at that base cannot be 0.1 mm and its thickness is 0.025 mm. On the bottom of the surface of that membrane, there is a thick layer of connective tissue and the organ of Corti deadens vibrations from the top. Vibrations occur in a fluid of certain viscosity. Bekesy calculated that own vibrations of that membrane have the frequency from 16 Hz to 20,000 Hz. However, dogs hear frequencies up to 50 kHz, young cats and mice up to 100 kHz and bats up to 200 kHz. Can the basilar membrane have such a spread in own vibrations? Experiments have shown that own vibrations of various human tissues range from 5 to 100 Hz. Such calculations were required to prove the resonance of sound wave and basilar membrane. However, there are problems here as well: the velocity of sound wave in the cochlear fluids is 1450 m/s and the velocity of the travelling wave is 1.9 through 100 m/s, depending on the frequency and site on the basilar membrane. With such a low velocity of wave, the reaction time on the basilar membrane stretches and is does not comply with electrophysiological testing [10].

3. Conclusions

Effort has to be made to learn about and explain hearing according to physiology. An important issue here is the explanation of formation and magnitude of swinging motions of the stirrup. It it dependent on the structure and properties of the tympanic membrane which receives acoustic information and transfers it onto the ossicles? Difference in the structure, thickness, tension and attachment to the handle of malleus and the tympanic ring may be of significance. Rocking motions of the handle of malleus are generated, which are transmitted by the incus to the stirrup through the spheroid incudostapedial joint. Another significant issue is the need to explain the osteopneumatic conduction. There are possibilities of ever more detailed vibrometric and electrophysiological testing. In stapedotomies, if one could direct high frequencies to the osseous enclosure of the cochlea instead of the prosthesis only – as is physiologically correct – hearing could be improved for high frequencies [11]. Good knowledge of all hearing mechanisms may lead to improvement of pharmacological treatment outcomes.

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