



# Remarks to the Theory of Hearing – A Traveling Wave

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## Abstract

The process of hearing is determined by physical, chemical and molecular mechanisms. Not all of them have been so far known and described. As an example, inertia in the middle and external ears may be taken. In the ear there is speed, acceleration and mass on the path of information transmission; hence, there is also the phenomenon of inertia. This phenomenon is described in the wave motion, too. Too little attention is devoted to the acoustic cell itself in relation to all transformations occurring in the cell, connected with the reception and processing of auditory information. The acoustic cell is also responsible for amplifying the signal on the path to the center. In the paper, attention was also paid to hearing nearby the threshold hearing and the reception of short sounds whose duration time lies under 1/10 ms. Described was the significance of the disappearance of the sound wave energy on the way from the external auditory meatus to the round window. Indicated were some doubts about the basilar membrane proper vibrations and its resonance potential. Attention was paid to the temporal inconsistency of the auditory response, especially, between the generation of the receptor potential and the time of the signal traveling through the cochlear fluids and the basilar membrane. It concerns, in particular, silent tones which need an amplification through OHC's contractions. Described is the mechanism to explain how the sound wave energy acts upon the auditory receptor. Indicated was the lack of any description of the mechanism for the encoding of auditory information through numerous elements of the signal path to the center in the presence of so many energy transformations.

**Keywords:** Acoustic cells, amplification, protein molecules, receptor, Stapedotomy; Ionic canals

## Abbreviations

ATP: Adenosine Triphosphate; cAMP: Cyclic Adenosine Triphosphate; cGMP: Cyclic Guanosine Monophosphate; DAG: Diacylglycerole; Hz: Hertz; kHz – 1000 Hz – kilo Hz; Pa: Pascal; pm: Picometre = 10<sup>-12</sup> m; OHC: Outer Cell Hair; IHC: Inner Cell Hair; nm: Nanometer = 10<sup>-9</sup> m; ms: Millisecond = 10<sup>-3</sup> second; ABR: Auditory Brainstem Response; BERA: Study of the Auditory Brainstem Potentials; BM: Basilemma = Basilar Membrane

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## Introduction

### Inertia in the ear

In the middle and the internal ears there are speeds, acceleration and mass; hence, there is inertia, too. Inertia in the wave motion is calculated from the formula:  $(2\mu \times \text{frequency})^2 \times \text{amplitude} \times \text{mass g/mm} \times \text{s}^2$ . Middle ear: Malleus 25 mg, incus 30 mg, stapes 3 mg, supplemented by the eardrum, mucous membrane, a part of ligaments, which together with ossicles amounts to a total mass of 70 mg.

For an amplitude 0.1 nm (20 dB) and frequency 1000 Hz – the inertia value of the middle ear is  $276 \text{ g/mm} \times \text{s}^2$ .

For an amplitude of 100 nm (80 dB) and frequency 1000 Hz - the inertia value of the middle ear is  $276068 \text{ g/mm} \times \text{s}^2$ .

Amplitude 100 nm (80 dB) - and frequency 10,000 Hz - the inertia value of the middle ear -  $27606880 \text{ g/mm} \times \text{s}^2$ .

Basing thereupon it can be judged that the inertia in the parts of the inner ear will increase even more, since there appears also an additional vibrating mass. Each increase in the vibrating mass will increase inertia and will lower the limits to which the wave energy may be conducted through vibrations of the elements of both the inner and the middle ears. There is one question: what is the limit of inertia so that energy may be freely conducted? And what happens when this limit is exceeded? A sound wave has no mass and may be freely and quickly transmitted to a receptor

through the bone of the cochlear housing.

### Operation of an acoustic cell

In all senses embedded is a perfect controllable molecular mechanism for signal amplification [1]. Intracellular amplification means a whole complex of factors, like: Phosphorylation and dephosphorylation of ionic canals responsible for the conduction of cell membranes, control of the ATP concentration, cAMP and cGMP levels, cell's pH, osmotic pressure, presence of ligands, the work of  $Ca^{++}$ ATPase pumps. Those pumps, related to the cell membrane, play an important role in keeping a variable calcium level in a cell. An intracellular amplification is also connected with the work of calcium dependent proteins, where an important role is played by calmodulin influencing the production and disintegration of cAMP and cGMP. Calmodulin activates kinases and protein phosphatases which control the work of the calcium pump. It also affects contractions of muscular and non-muscular cells through activating the kinase of light myosin chains independent of cAMP. Calmodulin affects exocytosis. Saturation of 4 calmodulin domains increases its action up to 100,000 times. Regulated is also the process of generation of enzymes or the rate of their decay. Calcium is the second information transmitter in a cell, acting faster than other transmitters, *viz.* cAMP, cGMP, DAG, IP3 which are produced due to increased calcium level or to G protein activation. The stage of producing second transmitters constitutes one of several mechanisms of intracellular amplification. One enzyme particle may produce several hundred-second transmitters. To guarantee a correct work of an acoustic cell, there must be equilibrium between the calcium flowing into a cell through ionic canals in compliance with the electrochemical potential and a fast release of calcium outside the cell through ionic canals, ionic pumps and ion exchangers.

### Signal amplification

The pressure amplitude of a sound wave, the quietest one a young healthy man can hear at a frequency of 1,000 Hz, is  $2.0 \times 10^{-5}$  Pa, and after conversion into the amplitude in meters it amounts to  $8.0 \times 10^{-12}$  m = 0.008 nm = 8 pm. This is a size 5 times smaller than the hydrogen diameter. The leverage ratio 1.3:1 causes the wave amplitude to decrease. The signal energy to the receptor conveyed through cochlear fluids and the basilar membrane and decreasing a few hundred times, is not very reliable and too slow. Such a wave cannot bend the hairs of acoustic cells whose diameter is 10,000 times larger. So that a signal may be amplified with an OHC's contraction, according to the traveling wave theory, the signal is to be received by the receptor. Sub-threshold tones cannot be amplified with this method. Complex tones are loud and silent tones, with various frequencies. Loud sounds are received and a signal is conveyed directly to the center [2]. Silent tones are subject to time-consuming mechanical amplification (according to the traveling sound theory) which is superimposed onto new waves traveling towards the ear at this time. The signal becomes split, silent information is sent with a delay. An OHC's contraction increases a Basilar Membrane [BM] displacement, but of a new wave, not the primary and quiet one which needed amplifying.

### Resonance of very short tones

The organ of hearing receives sounds of a duration time under 0.1 ms. At such a short time of the driving force of a sound wave no resonance of the basilar membrane is possible. One or two wave periods are unable to transmit encoded energy of a sound wave. In what way is the frequency of such a sound recognized?

### Disappearance of sound wave energy

Research has proved that for input values, respectively, of 90 dB and 8000 Hz, *viz.* for an amplitude value of 500 nm, on the round window is ascertained an amplitude of 0.5 nm. The fading of this sound wave in cochlear fluids, as well as of an 8 pm threshold wave, takes place in the cochlea in such a proportion. Wave energy is proportional to the amplitude squared. This signal cannot be amplified since it does not reach the receptor [3,4]. It implies another signal path towards the receptor. That is why it should be supposed that a sound wave from the stapes base is conveyed to the bone of cochlear housing and without energy disappearance it reaches the crista spinalis at a speed of 4,000 m/s, and continues directly to the receptor of acoustic cells.

An important role is played by the rocking plates of the stapes, especially in the range of high frequencies and high sound intensities. Under 4 kHz those are motions in the short axis of the stapes. Above 6 kHz motions take place in the long axis of the stapes, instead.

### Basilar membrane parameters

The period of vibrations of a vibrating solid body is directly proportional to the square root of the solid body mass. It depends on the density and elasticity. The bigger the vibrating mass, the longer the vibration period and the lower the frequency. There are many indications that this is due to the law of inertia which is rather disregarded in the ear. For the calculations of the basilar membrane proper vibrations, it was assumed that the basilar membrane width at the cochlear base is 0.1 mm. Instead, the width of the septum between the cochlear duct and the eardrum canal is 4.3 mm near the oval window. Nearby the cupula, the basilar membrane width is 1.7 mm, whereas for the calculations the value assumed was 0.5 mm. It is difficult to agree that the thickness of the basilar membrane at its base is 0.0075 mm, and at the apex only 0.0025 mm [5]. Such dimensions were taken for the calculations as it is known that the basilar membrane is loaded from the top by the mass of the organ of Corti, and from the bottom the basilar membrane rests on a strip of connective tissue. Moreover, the whole conglomerate is immersed in the cochlear fluids which have suppressive properties.

### Mechanism of action of a sound wave upon a receptor

The hypothesis regards merely the mechanism transforming the sound wave energy into receptor potential of acoustic cells. It occurs without participation of the basilar membrane and the cochlear fluids. Mechanical energy of a sound wave acts upon protein molecules (sound sensitive molecules), responsible for generating the motion for the gating mechanism which opens  $K^{+}$  mechanosensitive ion channels. The energy of a sound wave acts upon ionic and covalent bonds, changes the angles of bonds as well as vibrations of particles and atoms. It also changes the activity of enzymes, causes a slight modification of temperature and pressure in the cell together with a modification to the properties of the cell membrane. Conformational changes of proteins may support the gating mechanisms. Proteins will be stretched and rolled up, which gives rise to a motion of molecules. Information transferred by a sound wave may be precisely conveyed without delay and exposure to deformations. Energy of a sound wave may reach a receptor on a roundabout path through cochlear fluids and the basilar membrane, but it also can reach it considerably faster through the osseous housing of the cochlea, without multiple changes of energy and without disappearance and deformations [6,7].

### Hearing in case of disrupting the ossicles

An interruption of the chain of ossicles or the lack of the

tympenic membrane causes a hearing loss of 50 dB to 60 dB. It is assumed that air vibrations of the tympanic membrane transmit the energy to the stapes footplate, and further path of the signal to the receptor complies with the theory of traveling wave. Nevertheless, a sound wave acts at the same time on the round window whose susceptibility to vibrations of the round window is 20 times higher than of the oval window. A wave is generated faster in the tympanic duct and travels to the oval window. There is a problem with the generation of a traveling wave on the basilar membrane. A higher wave pressure is in the tympanic duct, not in the cochlear duct; any way the resolution of frequency and intensity is maintained. There is no signal amplification through an OHC contraction. For the reception of information responsible are receptor fields of acoustic cells, not the basilar membrane with a traveling wave. According to the theory, information is transmitted through the ossicles exclusively to cochlear fluids. Instead, many investigations have proved that human soft tissues also convey sound waves. An irrefutable proof is the hearing of a fetus in its mother's womb as soon as from the 2<sup>nd</sup> half of gestation. There is no direct conduction through the bone, and the child can hear its mother's voice, her heartbeat, or peristalsis. It 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. Such research was done. The vibrating tympanic membrane is fixed in the tympanic ring and is able to convey vibrations to the bone. Also the auditory ossicles are connected with the walls of the tympanic cavity; sound waves are conducted through ligaments. The stapes is connected with a strong stapedial muscle with the osseous wall. The stapedial plate is, instead, connected with a flexible annular ligament with the housing of the oval window housing. During vibrations, the energy of waves is conveyed to the osseous cochlear housing. Hence, the theorem that the energy of waves is transmitted only and exclusively to cochlear fluids is unjustified.

#### Rate of generation of the receptor potential

A signal covers each segment of the path towards the receptor in a specified time, and those times are added up. Performed were some

experiments and diagnostic tests which accurately determined in what time after a stimulation receptor potential would be generated in the external auditory meatus, and in what time a signal is examined in the auditory nerve, the nuclei of the auditory nerve - the brainstem or in the cerebral cortex. It was ascertained that receptor potential is generated in a time almost indeterminable, as short as decimal parts of millisecond, whereas in the auditory nerve a signal is detected after 1.5 ms to 1.9 ms. Should it be assumed that a quite tone is subject to a time-consuming mechanical amplification, so on what path and in what way can this signal reach the receptor in such a short time?

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