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The Baby's Hearing in the Womb

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Abstract

The paper presents a brief description of developmental stages of the human hearing organ. Attention was brought to the mechanism of receiving and processing auditory information in a child in the womb. Bekesy's travelling wave theory does not provide a sufficient explanation of these processes. There are no descriptions of auditory mechanisms on a molecular level. Further in the study, attention is drawn to the beneficial effect of early stimulation of the child's auditory receptor from the second half of pregnancy and after childbirth.

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Development of the Hearing Organ

Early in week 4 of foetal life (FL), a primordium of both bony labyrinths emerges in the form of a swelling of the ectodermal epithelium in the rhombencephalon area. Around day 25 of FL, the swelling sinks into the mesenchyme, creating the auditory vesicle filled with a liquid. In week 6 of FL, a straight cochlear duct is formed, which then becomes coiled and reaches its final form of 2.5 coils mid-pregnancy. In week 10 of FL, the tympanic duct and then the vestibular duct are formed, and in week 17 of FL the helicotrema is formed. The epithelium of the organ of Corti is differentiated from week 12 of FL and it becomes fully developed by mid-gestation. In the same time, the spiral ganglion and the neural links with brain centres are being formed. The tympanic membrane is formed ca. week 12 of FL. The ossicles of the middle ear are ultimately developed in week 36 of FL [1].

Hearing Mechanism in Foetal Life

In the womb, the child can hear as early as from the beginning of the second half of pregnancy. However, there is no precise explanation for the mechanism of hearing in these conditions. The theory of hearing developed by Bekesy is not fulfilling its role here [2]. Air conduction is not in place. What is in place is liquid conduction, soft tissue conduction, and cartilage and bone conduction. A myxoid liquid is accumulated in the tympanic cavity. In the inner ear, the immobile stapedial footplate generates no sound wave in the cochlear fluid. The blockade of the round window due to the presence of liquid in the tympanic cavity prevents the travelling wave from being formed on the basilar membrane. Nonetheless, the energy of the sound wave reaches the receptor that is capable of receiving and processing auditory information from the second half of gestation. These circumstances indicate that wave energy does not have to generate wave motion of the basilar membrane that supposedly, which according to Bekesy's theory reduces the distance between the basilar membrane and the tectorial membrane, to cause flow of the cochlear fluid that makes hair cells bend. By passing through different environments sound wave energy reaches the bone labyrinth and the receptors of auditory cells of the organ of Corti directly. This is a pathway taken by the signal to reach the receptor different to the one described by Bekesy [3].

As the inner ear develops, a fast-paced formation of the cerebral cortex is taking place. Its two layers became completely replaced whereas in other layers, 50-90% of neurons are replaced. The renewal of the cerebral cortex consists in old neurons being replaced with a more perfect network of new neurons. The neuron replacement process occurs mainly in foetal life, but it also occurs long after the child is born. It is neuron proliferation. Ultimately, we have about 10 billion neurons and 1000 times as many synapses. A significant role is played by a process called neuron migration. Neurogenesis aside, there is also the process of synaptogenesis, that is, the forming of new synaptic connections between neurons. Synaptogenesis starts in early stages of foetal life and takes place to about 2 years after birth in sensory areas and up to teenage years in prefrontal areas, as well as temporal and parietal areas.

One can assume that the brain continues to develop and mature up to middle age and that the minimum decrease in the number of neurons is observed before the age of 55 years. Albeit limited,

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neurogenesis in an adult brain is possible.

Learning and ongoing training of the brain stimulate neurogenesis. This pertains not solely to the formation of new cells but also dendritic arborization, that is, the creation of new lateral connections between neurons. Learning increases the number of grey matter and improves brain plasticity by means of new lateral synaptic connections. Mature astrocytes of the hippocampus regulate neurogenesis by issuing relevant commands to stem cells of an adult mammalian brain so that they can be formed as neurons. Astrocytes regulate the environment surrounding neurons by releasing numerous neural and growth factors, they partake in regulating synapse formation and regulate synaptic transmission. In line with the information of neuroinformatics experts the total amount of the data processed on all our sensory receptors is as high as 100 billion bits per second! Of this entire sum, only 100 b/s reach our consciousness. This means that for every single bit of the information that reaches our consciousness, one billion bits of data are processed without using our consciousness [4].

In the foetal stage, the beginning forms of neurons of the developing brain divide at an extreme pace. The efficiency of this process can reach up to 15 million in one hour. This process is slowing down on an ongoing basis. However, the process of synaptogenesis intensifies significantly after birth, which is related to the information from the outer world being received by all sensory organs. There is a reversed principle, namely, the absence of signals reaching a given sensory organ inhibits neurogenesis and synaptogenesis of cortical and subcortical centres of a given organ.

Synaptogenesis is the development of trillions of synaptic connections between neurons of the developing child brain. The timescale of that process is long and falls to a period from the sixth month of pregnancy to the third year of the child's life. We can grow our lateral connections of neurons throughout our lives by learning and training the brain in various ways, which is referred to as adult brain plasticity. Neurogenesis consists in neurons fighting one another to the death. A neuron that fails to connect to receptor cells and link to other neurons is doomed. The apoptosis mode is triggered automatically. The same apoptosis mode can be triggered by detrimental factors, toxins, and xenobiotics that affect a young brain in particular. Likewise, many medications that are in use have an adverse effect on both neurogenesis and synaptogenesis. In many areas on a neuron, the so-called spines are formed, with axons growing through to reach places that are sometimes distant, such as sensory organs or ganglia, where they form synaptic connections. A neural spine is where a growth cone is formed, first described by Ramóny Cajal (1852-1934). A growth cone is formed by microtubule populations growing in one direction causing the emergence of axons. A growth cone creates numerous microspines that initiate the formation of new axons. This way, one neuron can have 100 axons. A neuron that fails to connect to obtain stimulation, signals from target cells deteriorates by means of apoptosis since it is not receiving trophic supply essential for an axon and a neuron to survive. Target cells produce neurotrophins that act on axions via tyrosine kinase receptors. A typical trophic factor is TNF nerve growth factor. The absence of the NGF factor leads to the death of a cell dependent on this factor. There are more of such factors. Some kinases pass to the nucleus of a neuron where they activate transcription factors. Neurons communicate with one another by means of electric potentials and signalling using transmitters. The arising groups of neurons with shared interests create separate centres that are linked to specific sensory organs or ganglia. Specific centres form connections with one another. A network of synapse connections is formed.

Conclusions

Why is it so important? Because in foetal life, there is no visual, gustatory, or olfactory information. The information that reaches the receptors is the touch and vast majority of auditory information. Auditory information stimulates development of the nervous system. From mid-pregnancy, the child can hear the mother's heartbeat, reacts to changes in the heartbeat, hears eating-related sounds, hears intestinal movements, and the noise coming from the respiratory system. It starts hearing the mother's voice when sound energy is transmitted via soft tissues and the skeletal system. Initially, the child in the womb recognizes only simple sounds but over time, it can distinguish vocal timbre and tone. These sounds are received and memorized, as evidenced by the ability to recognize the mother's voice after birth. **A child** in the womb hears the mother's voice better than the father's voice for two reasons. Firstly, the tonality of the mother's voice is higher and better understood owing to enhanced pronunciation of consonants. It is transmitted directly through the mother's body. Secondly, the father's voice is resisted by the impedance of the abdominal wall significantly reducing the energy that reaches the ears of the foetus. This is facilitated by the father putting his head against the pregnant woman's abdomen, laying his hands on the pregnant woman's abdomen. In that period, stimulating auditory receptors is very important for the development of the nervous system and the mental development of the child. It is highly beneficial if the child hears conversations, listens to music often. The child reacts by becoming either more relaxed or excited depending on the type of music. Accustomed to listening to the sounds that come from the cardiovascular system, the respiratory system and the digestive system of the mother all the time, after birth, the crying child is soothed when a hairdryer or a vacuum cleaner is turned on. After birth, the child should listen to good, not overly loud music often.

Bekesy's travelling wave theory fails to explain the mechanisms for receiving and processing auditory information by the child in the womb [5]. The inner ear is not functioning, there is no possibility for a travelling wave to be formed on the basilar membrane. Receptors of the organ of Corti receive signals transmitted by the mechanical energy of a sound wave that come to the ear from the world outside though bodily tissues and the surrounding liquid. This energy acts on protein (sound-sensitive) molecules by means of changes in their bonds, shifts in the bond angles, affects particle rotation, the arrangement of protein molecules, enzyme activity, conformational changes in proteins responsible for ion channel gating. As many as 85 genes determine the composition of channel proteins of the mechanically-activated potassium channel. The sensitivity of sound-sensitive molecules to a specific sound wave frequency is genetically conditioned. Sound receptors on the hair cells are situated along the basilar membrane from the highest frequencies close to the oval window to the lowest frequencies in the vicinity of the cupula. The basilar membrane serves as a support for the organ of Corti and also separates two liquid spaces of different concentration values. There is no afferent or efferent innervation, it stems from the mesenchyme. There is no capacity to alter the tension. Natural frequencies of basilar membranes in mammals that receive frequencies up to 100 kHz rule out resonance with a sound wave in the cochlear fluid. Sound wave

in the cochlear fluid is a longitudinal wave of the speed of 1450 m/s, whereas the wave on the basilar membrane is a transverse wave, with a slow pace of 0.9-50 m/s. Encoding quantified [6] energy of a sound wave transmitted on the travelling wave at compression this high is impossible. Vibrations of the basilar membrane in the fluid are severely dampened. If the dampening is greater than the energy of the incident wave, resonance is impossible. This situation takes place during listening to the threshold tones and slightly above-threshold tones. These tones are received by the receptor. Frequency resolution is determined by tonotopy amplified by the effect of receptor fields. Signal amplification takes place on a molecular level in an auditory cell. This pertains to received tones whose energy is too low for the information to reach the centre.

Finally, a fundamental question arises, namely, is it possible that Nature replaced the hearing mechanisms present at the initial stage of life with the mechanisms described in Bekesy's travelling wave theory, though at times, the latter go against logic and physics? Is it possible that the hearing mechanisms described in the *Submolecular theory of hearing* [7] are a simple continuation of the hearing mechanisms of a child in the womb in line with the laws of Nature?

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