Journal of Spine & Neurosurgery

Editorial A SCITECHNOL JOURNAL

Biological Mechanism of Cochlear Implant Technology in Humans

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Received date: 07 December, 2021, Manuscript No. JSNS-22-56390;

Editor assigned date: 09 December, 2021, PreQC No. JSNS-22-56390 (PQ);

Reviewed date: 23 December, 2021, QC No JSNS-22-56390;

Revised date: 28 December, 2021, Manuscript No. JSNS-22-56390 (R);

Published date: 07 January, 2022, DOI: 10.4172/2325-9701.1000e101

Editorial Note

The World Health Organization reports that hearing loss was the most common form of sensory impairment in humans, affecting 360 million persons worldwide, with a prevalence of 183 million adult males and 145 million adult females. The most common source of hearing loss is sensor neural hearing loss, characterized by disfunctions of the sensory organ: the cochlea and its associated structures. These dysfunctions may be genetic or acquired. In the latter case, it can be due to environmental factors such as chemical agents or noise exposure, or to age related senescence. In patients with sensorineural hearing loss, the functions of the cochlear cells and tissues are lost. Nevertheless, some auditory neurons survive, and the role of the cochlear implants is to stimulate them directly by shunting the cochlea. In this case, the hearing of patients with profound hearing loss can be successfully rehabilitated with cochlear implants capable of encoding and delivering the spectral and the temporal information of sound to the surviving auditory neurons. In this review, we summarize the physiological mechanisms involved in hearing loss and hair cell apoptosis, the role of cochlear implants in cochlear neuron stimulation, and the clinical advantages and disadvantages related to this cochlear device implantation.

Biology of Hearing Loss

The mammalian inner ear is a sensory organ capable of perceiving sound over a range of pressures and differentiating both infrasonic and ultrasonic frequencies in different species. In human hearing, sound pressure waves travel down to the ear canal and cause the vibration of the eardrum. These vibrations are transmitted to the cochlea via 3 small bones: the malleus, the incus, and the stapes, all located in the middle ear. The movement of these bones allows the oval window to move and to conduct the movement into the cochlea. The cochlea is then responsible for transducing the mechanical vibration into action potential that will propagate towards the part of the brain responsible for hearing which allows perceiving a sound.

The cochlea is a spiral structure divided along its length by a membrane, called the basilar membrane. It is large and flexible at its apex, and narrow and stiff at its base. This longitudinal stiffness gradient makes the basilar membrane react differently depending on the frequency of the incoming sound. For sounds with energy in the low-frequency range, vibrations are maximal at the apex, while for sounds with energy in the high-frequency range, vibrations are maximal at the base. This results in a tonotopic organization of the acoustic input along the cochlea [1]. The organ of corti, located on the basilar membrane, houses two different subtypes of sensory cells arranged along the duct: three rows of outer hair cells and one row of inner hair cells, those are the true sensory hair cells [2]. Each one possesses dozens of hairs which bend back and forth with the vibration of the basilar membrane. This bending of the hair depolarizes the cell, which releases neurotransmitters onto the afferent nerve fibers, provoking an action potential transmitted to the auditory brain structures. More than 90% of the afferent fibers originate at the inner hair cells. Each fiber has synaptic contact with one inner hair cell which is innervated by around 10-20 fibers. The outer hair cells are innervated by only 10% of the afferent nerve fibers, and many outer hair cells converge on a single fiber. These dendrites forming synaptic contact with hair cells compose the spiral ganglion, a nervous structure that transmits electrical signals from the cochlea to the central nervous system. One tiny change in one of these structures or systems can lead to Sensorineural Hearing Loss (SNHL).

Implant Technology

As mentioned before, in 2012, the World Health Organization reported that hearing loss was the most common form of sensory impairment in humans, affecting 360 million persons worldwide [3]. The most common source of hearing loss is sensorineural hearing loss, which accounts for about 90% of reported hearing loss and emerges from disfunctions of the sensory organ: the cochlea and its associated structures. These disfunctions may be genetic; 40 genes have been identified to cause deafness [4] or acquired. In this case, it can be due to environmental factors such as chemical agents or noise exposure, or to age related senescence [5].

It is well established that mitochondria are responsible for ATP production, and that this process induces an increase of Reactive Oxygen Species (ROS) such as superoxide anion, hydrogen peroxide, and hydroxyl radical, playing an essential role in cell signaling [6]. Under normal conditions, ROS produced by the mitochondria are easily metabolized by endogenous antioxidant mechanisms such as catalase, superoxide dismutase, glutathione, and balance cell homeostasis. However, the aging process, pharmacological treatment, or external factors, can alter this balance. This imbalance is called oxidative stress [7]. Several publications confirm that the mitochondrial ROS overproduction plays a key role in hearing loss by activating hair cell apoptotic pathways [8]. More precisely, intracellular damage caused by noise, or ototoxic agents such as aminoglycosides or cisplatin, seems to share a final common pathway via the cytochrome c translocation and caspase activation, leading to hair cell death [9].

In patients with SNHL, the function of the basilar membrane and the sensory cells is lost. Nevertheless, some auditory neurons survive, and the role of the Cochlear Implants (CI) is to stimulate them directly by shunting the cochlea. In this case, the hearing of patients with profound hearing loss secondary to ototoxic agents can be rehabilitated successfully with CI capable of encoding and delivering the spectral and the temporal information of sound to the surviving auditory neurons [10].

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