Inner ear damage affects brain chemistry

Our ability to hear is brought about by the function of our ears. Besides the visible external ears, we have middle and inner ears. Our left and right inner ears, called cochleas (singular cochlea), translate sound vibrations into nerve signals, which are carried to the brain by the auditory, or cochlear nerves (Fig. 1.). Where the cochlear nerves enter the brain on each side, they transmit their signals to nerve cells located in structures called the cochlear nuclei (singular cochlear nucleus). The signal transmission is accomplished by release of chemicals that affect the activity of nerve cells at junctions called synapses. Many chemical transmitter substances are amino acids, the same chemicals that also serve as the building blocks of proteins. Current evidence indicates that the main chemical transmitter substance used by the cochlear nerve to activate nerve cells in the cochlear nucleus is the amino acid glutamate. The cochlear nuclei are just the first stop in our brain connections that enable us to hear. Once the nerve cells in the cochlear nucleus have received the information about sounds from the cochlear nerves, they analyze this information and send signals on to higher brain centers, where further analysis occurs, until information finally reaches the cerebral cortex.

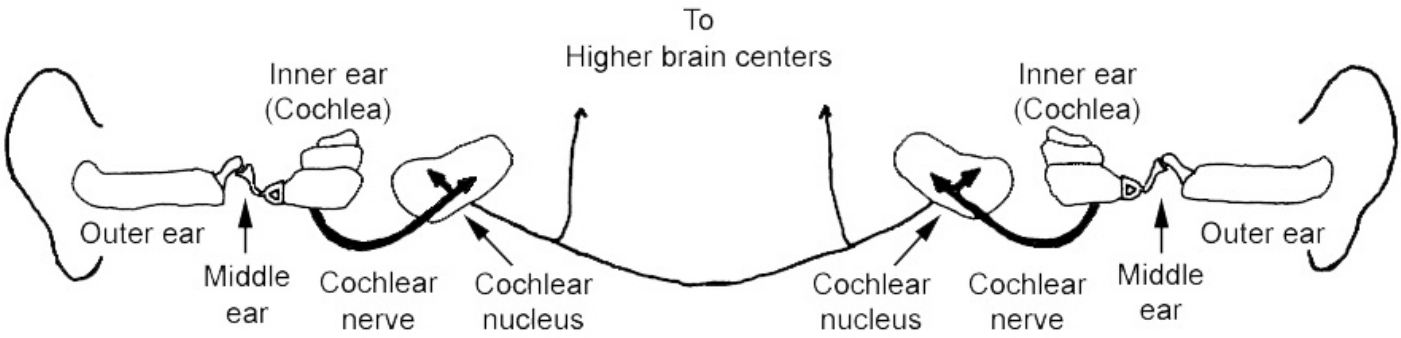

Fig. 1. Hearing system, from ears to cochlear nuclei.

Our cochleas and cochlear nerves are susceptible to damage by loud sounds, some drugs, blows to the head, and brain tumors or other illnesses. These damages can lead to changes in the brain parts of our hearing system, which are currently not well understood, and result in loss of hearing, distorted hearing, and tinnitus (hearing a monotonous, usually ringing, sound that is not actually present). By experiments on animals, we can study changes in the brain after damage to the cochlea or cochlear nerve. A logical place to initially study is the first brain center for hearing, the cochlear nucleus.

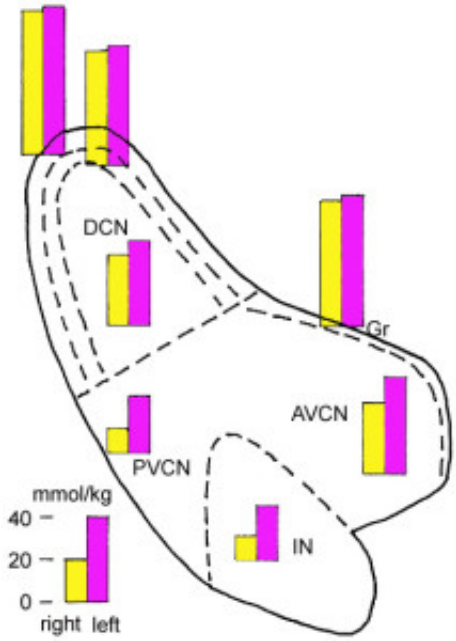

Fig. 2. Average glutamate levels, expressed as heights of bars, in right and left cochlear nuclei one month after destruction of the right cochlea, in regions that receive much (AVCN, IN, and PVCN) or little (DCN outer layers and Gr) input from the cochlear nerve.

We studied changes in amino acid levels in the right and left cochlear nuclei after destruction of the right cochlea. There were large decreases in levels of glutamate in the right cochlear nucleus parts where the cochlear nerve makes most of its synapses with cochlear nucleus nerve cells but little change in other parts of the right cochlear nucleus or in the left cochlear nucleus (Fig. 2.). These decreases of glutamate levels probably resulted from degeneration of the auditory nerve after destruction of the cochlea. They were noticeable 2 weeks after cochlear destruction and continued for 3 months or more. The levels of most other amino acids did not change very much. Our findings are consistent with an important role for glutamate in transmission of information from the cochlear nerve to the cochlear nucleus.

Currently, there are no treatments that can restore normal hearing after damage to the cochlea or cochlear nerve, but researchers are actively seeking ways to minimize the negative effects of the damage. One approach to give people some hearing after loss of cochlear nerve function is to stimulate the cochlear nucleus directly. Our findings provide knowledge about the chemical state of the cochlear nucleus with which such stimulation would have to work. The encouraging result is that, other than the loss of cochlear nerve chemicals, there are not large changes in most amino acid levels, so the cochlear nucleus nerve cells that are directly stimulated may be able to send normal signals to the higher hearing centers of the brain.

Donald A. Godfrey
Department of Neurology and Division of Otolaryngology and Dentistry, Department of Surgery
University of Toledo College of Medicine
Toledo, USA

Publication

Cochlear ablation effects on amino acid levels in the chinchilla cochlear nucleus.
Godfrey DA, Chen K, Godfrey MA, Lee AC, Crass SP, Shipp D, Simo H, Robinson KT.
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