Neural plasticity and aural rehabilitation

BY CHUL-HEE CHOI

Neural plasticity refers to an ability of the brain and central nervous system to change their structure and function or their reorganisation in response to environmental cues, experience, learning, behaviour, injury and / or diseases and treatments. Neural plasticity is directly or indirectly related to aural rehabilitation. A better understanding of the mechanisms of neural plasticity and its relationship with aural rehabilitation may contribute significantly to improvement in audiological management of damaged or lost auditory function.

Recently, noninvasive auditory evoked potentials and brain imaging techniques such as magnetoencephalography (MEG), functional magnetic resonance imaging (fMRI), and positron emission tomography (PET) have made the study of neural plasticity in humans possible. Neural plasticity is associated with aural rehabilitation of adults with sensorineural hearing loss in three ways [1]. First, reduced auditory input from cochlear pathology may cause functional changes of the auditory system. Second, aural rehabilitation may result in secondary plasticity due to the altered input to the auditory system. Finally, aural rehabilitation can lead to functional changes in the auditory system. How aural rehabilitation can cause neural plasticity will be reviewed in terms of cochlear implantation and hearing amplification, and pharmacological intervention.

In cochlear implantation, neural plasticity is associated with deprivation of auditory input, adaptation to the absence of stimuli, and neural reafferentation provided by cochlear implants [2]. The recovery of hearing after cochlear implantation (CI) was mainly found in the bilateral middle and superior temporal gyri, corresponding to Brodmann areas 21 and 22. The neural activity of these areas was significantly stronger when CI users listened to running speech than when they listened to multi-talker babble. A significant difference between CI users with postlingual hearing loss and CI users with prelingual hearing loss was observed. While the CI users with postlingual hearing loss showed greater activation in the left superior temporal gyrus, the CI users with prelingual hearing loss displayed undifferentiated activity which indicates insufficient development of neuronal networks due to their prolonged deafness. Generally, the best performance provided by cochlear implantation for the CI users with postlingual hearing loss was observed during the first year after cochlear implantation. Although recent studies have established a direct correlation between a level of brain activity and the auditory performance after cochlear implantation, the success of cochlear implantation can be affected by multiple variables such as severity, duration and the age of onset of the hearing loss.

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The perceptual recovery provided by hearing aids may also induce neural changes in the auditory cortex in terms of the studies of late-onset auditory deprivation and hearing aid acclimatisation [1]. Late-onset auditory deprivation represents a significant auditory deprivation in speech recognition performance of the unaided ear during monaural hearing aid use of people with bilateral, symmetrical sensorienural hearing loss. Auditory acclimatisation refers to a systematic improvement in auditory performance over time that is linked to a change in the acoustic information available to the listener but is not caused by task or training effects. These effects come from changes in cortical plasticity after use of the hearing aid for some period of time. Many studies provide direct evidence of a strong correlation between neural plasticity in the central nervous system and changes in auditory performance from amplification. However, as we know, the auditory benefits provided by hearing aids can be affected by many factors including device centred variables such as directional microphone, signal processing and gain settings and people centred variables such as age, attention, motivation and biology [3]. We are not yet aware of a clear relationship between each factor affecting the hearing aid performance and the corresponding

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cortical plasticity.

Exposure to strong noise induces oxidative stress exceeding the capacity of the antioxidant defence mechanisms in the cochlea through excessive formation of free radicals including reactive oxygen and nitrogen species, which leads to hair cell damage and sensorineural hearing loss. Pharmacological interventions for the prevention or treatment of noise induced hearing loss have been developed by antioxidant drugs increasing the antioxidant defense system destroyed in the cochlea. These antioxidant drugs include N-acetyl-L-cysteine (NAC), acetyl-L-carnitine (ALCAR), and phenyl-N-tert-butylnitrone (PBN), disufenton sodium (NXY-059), and 4-hydroxy PBN (4-OHPBN), which help a recovery of the degenerated synapses in the dorsal cochlear nucleus and brain after noise induced hearing loss [4,5].

The length of recovery in aural rehabilitation, in terms of neural plasticity is directly associated with the age of the subject. The effect of neural plasticity on aural rehabilitation is stronger in subjects than in older ones [6]. However, it has been reported that neural plasticity is not restricted to early periods of development but occurs throughout the life span. Although neural plasticity in adults was observed in changes in cortical and subcortical responses, there is a stability / plasticity dilemma in terms of how the auditory system in adults can be modified by experience without compromising the stability necessary to execute its previously established functions [7].

Conclusion

Although we do not know whether the exact locations of neural plasticity induced by cochlear implantation are different from those of hearing aids, it is clear that the mechanisms of brain reorganisation after aural rehabilitation using cochlear implantation and hearing aids play an important role in auditory improvement.

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