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Why Do We Have “Central Gain”? Why Is The Neural Signal Amplified (And Not Attenuated) As It Progresses Up The Auditory System?

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Hearing loss can diminish the strength of auditory signals transduced in the cochlea. However, as these weak signals travel along the central auditory pathway, downstream neurons, such as those in the inferior colliculus and auditory cortex can respond by amplifying or boosting them. This process is known as central gain. While it seems like a paradox that hearing loss results in increased auditory neural activity, central gain in the auditory system is evidence that the nervous system actively adapts to the loss of sensory input.[1]

Enhanced central gain can happen in a few ways. There is evidence that sensory neurons are tuned to a homeostatic “set point” of activity – neurons prefer to maintain a desired balance of input and output. When this input–output balance is disrupted by sensory loss, neurons can change their properties and restore their sensitivity back to the set point.[2] Neurons accomplish this by increasing excitatory synapses and decreasing inhibitory synapses, or by changing the electrical properties of the cellular membrane.[3] Central gain could also occur from losing inhibitory connections from neighbouring neurons (i.e., lateral inhibition).[1] Central gain is evident in humans with peripheral hearing loss when you record the ABR. ABR Wave I, the synchronous firing of auditory nerve fibers in response to sound, may be smaller due to hearing loss. But Wave V, which is thought to originate in inferior colliculus, shows a larger response.[4] In other words, the Wave V / Wave I ratio increases with more central gain. Increased central gain can also happen in the auditory cortex.

Dysfunctional central gain could produce hyperacusis or tinnitus if the “set point” increases to a higher level than normal for sensory loss. In this circumstance, not just weak signals from the periphery, but other neural noise could be amplified through the auditory system.[1] This could lead to abnormally loud sound perception or the experience of tinnitus. While this suggests central gain mechanisms should be targeted for intervention, more scientific evidence is needed to substantiate this model of hyperacusis and tinnitus.

References

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