

Research has established that electrical stimulation of several parts of our body – such as skin, muscle, cochlea (part of the inner ear) or certain parts of our brain – can alleviate tinnitus. However, not all tinnitus patients benefit from these types of electrical stimulation. Our goal is to understand why some patients can, while some patients cannot, experience this benefit.

Progress Report: Electrical Suppression of Tinnitus

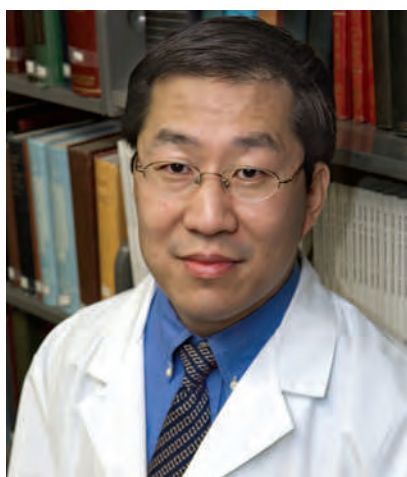
By JINSHENG ZHANG, PH.D.

To reach this goal, we asked a fundamental question: Which particular structures need to be stimulated, and which stimulation parameters are effective, in suppressing tinnitus? To approach this question, we used an animal model to test whether transcutaneous (through the skin) electrical stimulation (TES) of the *pinna* (external part of the ear) suppresses tinnitus-related activity in the brain.

The *dorsal cochlear nucleus* (DCN) is a structure in the brain's auditory system. Several lines of evidence show that sound exposure, the most common tinnitus inducer, causes hyperactive neural activity in the DCN. Our goal was to find out if TES would suppress DCN hyperactivity.

We tested TES on spontaneous activity of the DCN in both control and sound-exposed animals. We found that stimulating the pinna caused both suppression *and* excitation in the DCN of both control and sound-exposed animals. We learned four other notable things:

1. There was a higher incidence of suppression (up to 70 percent) than excitation.
2. At higher levels of electrical current, there was significantly



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more suppression *after* stimulation than during it.

3. Residual inhibition (when tinnitus stays quiet after stimulation is terminated) lasted from three to 10 minutes.
4. Sound-exposed animals had more suppression than control animals.

These results are consistent with the fact that some tinnitus patients benefit from electrical stimulation of their skin and muscles, while others do not. Additionally, we observed that TES did not dramatically decrease the level of DCN hyperactivity. This finding may explain the

clinical observation that the matching level of tinnitus is usually less than 10 dB (decibels) above hearing levels; yet, tinnitus is often described as being very loud (Dobie, 2004). In other words, it may not be necessary to dramatically change the level of neural activity in the DCN in order to significantly change the loudness of tinnitus.

Although our electrophysiological data were useful in understanding the effects of TES, it was still unclear which particular structures needed stimulation and which neural pathways needed activation to produce the desired suppression of DCN hyperactivity and especially to induce long-lasting residual inhibition.

To obtain this information, we conducted several experiments that tracked the pathways involved in the TES. In the first experiment, we injected a chemical tracer that tracks nerve pathways from the stimulation sites of the pinna. We found that the tracer traveled from the stimulation sites of the pinna to the *trigeminal nuclei*, a group of brain structures (parts) that are responsible for somatic sensation. Somatic or somatosensory means “pertaining to the body or senses.”

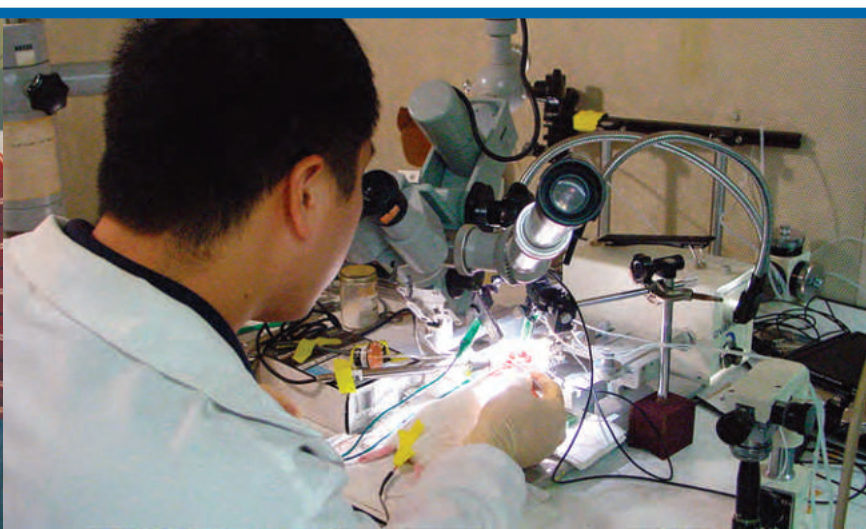
We later injected two other tracers in order to find the pathways between the trigeminal nuclei and the DCN. The results indicated that the electrical stimulation of the pinna activated the pathways that traveled to the DCN, and that there is an intimate interaction between the somatosensory and auditory systems. In addition, we observed a significant increase in activity in several non-auditory structures, including structures that mediate arousal, emotion and pain processes. The fact that these structures were activated suggests that electrical stimulation of the skin and muscles of the pinna activates several

(one type of nerve fibers that are possibly involved in TES) while we electrically stimulated the pinna. We found that electrical stimulation activity still affected DCN. This led us to think that there may be neural components other than parallel fibers that affect DCN activity following TES. Further experiments will help determine whether additional mechanisms are involved in the TES-induced effects.

In the second experiment, we used laser scanning confocal microscopy to find out if there are direct connections between nerve fibers from the trigeminal nuclei

that hyperactivity in central auditory structures – which underlies tinnitus – could be suppressed by transforming it into a type of activity that resembles normal spontaneous activity. That is, could sound-induced hyperactivity in the DCN be altered by high-rate electrical stimulation of the cochlea? We expect to complete this work toward the end of this year.

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Above right: Surgical and recording preparation.

Lower right: Stimulation and recording setup.

other pathways in addition to the somatosensory pathway.

These findings prompted us to investigate whether the effects of somatosensory electrical stimulation involved direct pathways from the trigeminal nuclei to the DCN, indirect pathways via certain non-auditory structures, or both.

To determine this, we conducted two experiments. In the first, we surgically removed parallel fibers

and nerve cells that are involved in suppression in the DCN. So far our data have not revealed any direct links. Through ongoing experiments, we continue to search for the mechanisms through which TES exerts suppressive effects on DCN activity.

Toward the end of year two, we initiated experiments to test the effects of high-rate electrical stimulation of the cochlea on DCN activity. We hypothesized

