Tinnitus and Auditory Cortex

LETTER TO THE EDITOR: I would like to congratulate De Ridder and colleagues on their paper describing a case in which magnetic and electrical brain stimulation were used to suppress a patient’s tinnitus (De Ridder D, De Mulder G, Walsh V, et al: Magnetic and electrical stimulation of the auditory cortex for intractable tinnitus. Case report. J Neurosurg 100:560–564, March, 2004).

Tinnitus is a distressing symptom that affects up to 15% of the population for whom no satisfactory treatment exists. The authors present a novel surgical approach for the treatment of intractable tinnitus, based on cortical stimulation of the auditory cortex.

Tinnitus can be considered an auditory phantom phenomenon similar to deafferentation pain, which is observed in the somatosensory system. Tinnitus is accompanied by a change in the tonotopic map of the auditory cortex. Furthermore, there is a highly positive association between the subjective intensity of the tinnitus and the amount of shift in tinnitus frequency in the auditory cortex, that is, the amount of cortical reorganization. This cortical reorganization can be demonstrated by functional magnetic resonance (fMR) imaging.

Transcranial magnetic stimulation (TMS) is a noninvasive method of activating or deactivating focal areas of the human brain. Linked to a navigation system that is guided by fMR images of the auditory system, TMS can suppress areas of cortical plasticity. If it is successful in suppressing a patient’s tinnitus, this focal and temporary effect can be perpetuated by implanting a cortical electrode.

A neuronavigation-based auditory fMR imaging–guided TMS session was performed in a patient who suffered from tinnitus due to a cochlear nerve lesion. Complete suppression of the tinnitus was obtained. At a later time an extradural electrode was implanted with the guidance of auditory fMR imaging navigation. Postoperatively, the patient’s tinnitus disappeared and remains absent 10 months later.

Focal extradural electrical stimulation of the primary auditory cortex at the area of cortical plasticity is capable of suppressing contralateral tinnitus completely. Transcranial magnetic stimulation may be an ideal method for noninvasive studies of surgical candidates in whom stimulating electrodes might be implanted for tinnitus suppression.

I have one comment and one question regarding this truly innovative and commendable work. At the University of Iowa we have been studying the normal functional organization of human auditory cortex in neurosurgery patients for many years. Our experimental findings indicate that the primary auditory cortex is located on the mesial portion of the Heschl gyrus and that an associated auditory cortex field is located on the posterior lateral superior temporal gyrus (field PLST). From the description provided in the authors’ report, it seems more likely that the brain site being electrically stimulated is auditory association cortex (field PLST), rather than primary auditory cortex as stated in the article’s conclusion.

We have observed that field PLST and primary auditory cortex are functionally connected; however, in our experience the sensory percepts evoked by electrical stimulation of the two fields are different. When field PLST is electrically stimulated some patients describe bilateral suppression of hearing that lasts beyond the period of electrical stimulation. In one of our patients with tinnitus, electrical stimulation of field PLST suppressed the patient’s tinnitus, but also suppressed his normal hearing bilaterally. Is this patient’s hearing affected during electrical stimulation?

The idea of electrically stimulating auditory cortex to treat intractable tinnitus is very attractive theoretically and is the subject of a number of US patents. The results presented by De Ridder and colleagues provide objective evidence to support the concept. They are to be congratulated for their pioneering work.

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Fig. 1. Prestimulation and poststimulation fMR images demonstrating a decrease in activity in the Heschl gyrus (arrows), indicating that the extradural lead interferes with activity in the primary auditory cortex.

References

Response: We appreciate the comments made by Dr. Howard. He points to some important problems regarding the physiological basis of our approach to the treatment of tinnitus.

Regarding the exact location of the stimulation, we do not know whether we only stimulate the primary auditory cortex, the PLST, or both. Anatomically, the stimulating electrode is positioned extradurally over the PLST and, as confirmed by Dr. Howard, the PLST is anatomically and functionally connected to the primary auditory cortex. Functional imaging studies have revealed increased activity both in the primary and secondary auditory cortex in patients with tinnitus.1–4

Recently we were able to obtain an fMR image in a patient whose tinnitus disappeared (placebo controlled) immediately after the beginning of electrical stimulation of the auditory cortex. The tinnitus recurred after 4 days of auditory cortex stimulation with approximately the same frequency and intensity as before the temporary suppression. The fMR image demonstrated a suppression of the hotspot in the Heschl gyrus but not in the superior temporal gyrus (Fig. 1), indicating that the stimulation had suppressed the primary auditory cortex. After implantation of a second electrode placed on a new hotspot located on the an-terior superior temporal gyrus the patient's tinnitus was suppressed again, indicating that the superior temporal gyrus might be a neural generator of tinnitus as well.

We believe that it is impossible in this setting to apply the electrical stimulation specifically to either primary or secondary auditory cortex, and it seems that stimulating primary and secondary cortex may be effective in suppressing tinnitus. The stimulation of the auditory cortex that we have performed in patients with intact hearing has not affected their hearing, either when stimulating the dominant or the nondominant side. We have so far not implanted electrodes bilaterally.

The patient described in the paper was deaf at the side of the tinnitus, so hearing was consequently not affected.

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References