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A stroke of silence: tinnitus suppression following placement of a deep brain stimulation electrode with infarction in area LC

Case report

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The authors report on a case of tinnitus suppression following deep brain stimulation (DBS) for Parkinson disease. A perioperative focal vascular injury to area LC, a locus of the caudate at the junction of the head and body of the caudate nucleus, is believed to be the neuroanatomical correlate.

A 56-year-old woman underwent surgery for implantation of a DBS lead in the subthalamic nucleus to treat medically refractory motor symptoms. She had comorbid tinnitus localized to both ears. The lead trajectory was adjacent to area LC. Shortly after surgery, she reported tinnitus suppression in both ears. Postoperative MRI showed focal hyperintensity of area LC on T2-weighted images. At 18 months, tinnitus localized to the ipsilateral ear remained completely silenced, and tinnitus localized to the contralateral ear was substantially suppressed due to left area LC injury.

To the authors' knowledge, this is the first report of a discrete injury to area LC that resulted in bilateral tinnitus suppression. Clinicians treating patients with DBS may wish to include auditory phantom assessment as part of the neurological evaluation.

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Key Words • **tinnitus** • **vascular injury** • **area LC** • **caudate nucleus** • **dorsal striatum** • **functional neurosurgery**

Tinnitus percepts are auditory phantoms without physical correlates. The condition is common, affecting 10%–15% of the adult population. Recently, a locus of the basal ganglia, area LC, located at the junction of the head and body of the caudate nucleus, was identified as a neuromodulation target for suppressing tinnitus.[2,3] Modulation of tinnitus by DBS in area LC has been attributed to neuromodulation and microlesion effects.

Depending on the specific combination of stimulation parameters, acute neuromodulation of area LC alters tinnitus perception by temporarily decreasing or increasing loudness and by controllably triggering wholly new phantoms. The microlesion of area LC, though not visible on routine postoperative MRI, is surmised to be responsible for tinnitus loudness suppression without electrical stimulation and for suppressing auditory phantom loudness beyond the duration of the acute experiment. Area LC of the dorsal striatum appears to be an important locus of the basal ganglia where sensory information is gated for further processing by the ventral striatum and related corticobasal circuits.[3]

We describe the case of a patient with a movement disorder and concomitant tinnitus who experienced enduring auditory phantom suppression in connection with surgery-related vascular injury to area LC. The injury occurred during a DBS procedure for PD, and its extent and location was documented by immediate and delayed postoperative MRI. Long-term clinical follow-up occurred 18 months after surgery. As area LC is often traversed during DBS surgery for movement disorders, we urge clinicians in the neurosciences to consider including auditory phantom assessment as part of the comprehensive neurological evaluation.

Abbreviations used in this paper: DBS = deep brain stimulation; LC = locus of caudate; PD = Parkinson disease.

Case Report

History and Examination. This 56-year-old right-

handed woman reported a decade-long history of progressive PD and a several-year history of tinnitus. Her PD-related complaints were motor fluctuations and dyskinesias that no longer responded to medical therapy. Tinnitus had become more noticeable over the 2 years leading up to surgery. Phantom percept sound quality was described as a hissing noise. Constant tinnitus was localized to both ears but was much louder in the right. Tinnitus loudness level rated on a 0- to 10-point scale (0–none, 5–conversation, 10–jet engine) was Level 8 in the right ear and Level 4 in the left ear.

Operative Course. The patient underwent clinically uneventful stereotactic implantation of a DBS lead (model 3387, Medtronic, Inc.) in the left subthalamic nucleus. One microelectrode recording pass was performed, followed by lead insertion along the same trajectory.

Postoperative Course. On the 1st day after surgery, with the pulse generator in the off mode, the patient reported a dramatic change in tinnitus perception. A routine postoperative 1.5-T MRI study was performed to evaluate the DBS lead position and rule out potential complications. The MRI study on the 1st day after surgery revealed a new abnormality of the left caudate. A T2-weighted sequence demonstrated focal signal hyperintensity (Fig. 1A) at the junction between the head and body of the nucleus. The DBS lead was positioned just lateral to the abnormality. Imaging results were consistent with focal vascular injury of the anterior body of the caudate, a locus of the dorsal striatum within area LC. Tinnitus loudness had declined to Level 2 in both ears (Fig. 1B). Tinnitus sound quality and its constant temporal profile were unchanged.

One month after surgery, motoric function improved substantially and the auditory phantom percept was nearly completely silenced. Tinnitus loudness was rated Level 3 in the right ear and Level 0 in the left ear (Fig. 1B). Tinnitus-related distress was slight, corroborated by a tinnitus handicap inventory score of 10. Eighteen months after surgery, tinnitus loudness reduction was sustained and tinnitus-related distress remained slight. Tinnitus loudness was rated Level 4 in the right ear and Level 0 in the left ear, and the tinnitus handicap inventory score was 2. Hearing acuity during and after the DBS procedure was unperturbed. An audiogram obtained 1 week after surgery showed bilateral mild high-frequency sensorineural hearing loss without significant asymmetry (Fig. 1C). Interaural thresholds at corresponding frequencies were within 5 decibels.

Discussion

Focal vascular insult of the dorsal striatum resulted in tinnitus loudness suppression without altering auditory phantom sound quality or temporal presence or disrupting matched hearing sensitivity between the two ears. In a case report[4] on a much more extensive unilateral stroke that also involved the dorsal striatum, longstanding tinnitus was completely silenced. In that case and the current case, unilateral stroke in the vicinity of the dorsal striatum suppressed tinnitus loudness localized to both ears. The dorsal striatum is the primary input station from secondary auditory and associative cortices to the basal ganglia.[5,6] Vascular injury to the dorsal striatum, a principal site of corticostriatal information conveyance, may have caused inactivation of certain neural circuits that gate auditory phantom signals.[3] The mechanism by which unilateral lesioning of area LC yields bilateral auditory phantom suppression is unknown.

The microlesion effect or peri-electrode edema is not likely to be a contributing factor to the tinnitus change in our patient. Prior studies have shown that leads traversing outside area LC do not produce changes in tinnitus perception.[2] In our patient, the lead trajectory was outside area LC with the infarct involving area LC itself. Only one microelectrode penetration was made, and the lead was placed along the same trajectory, so only one tract was made through the brain. The patient is currently undergoing chronic stimulation of the subthalamic nucleus for her PD, but stimulation of this nucleus has not been

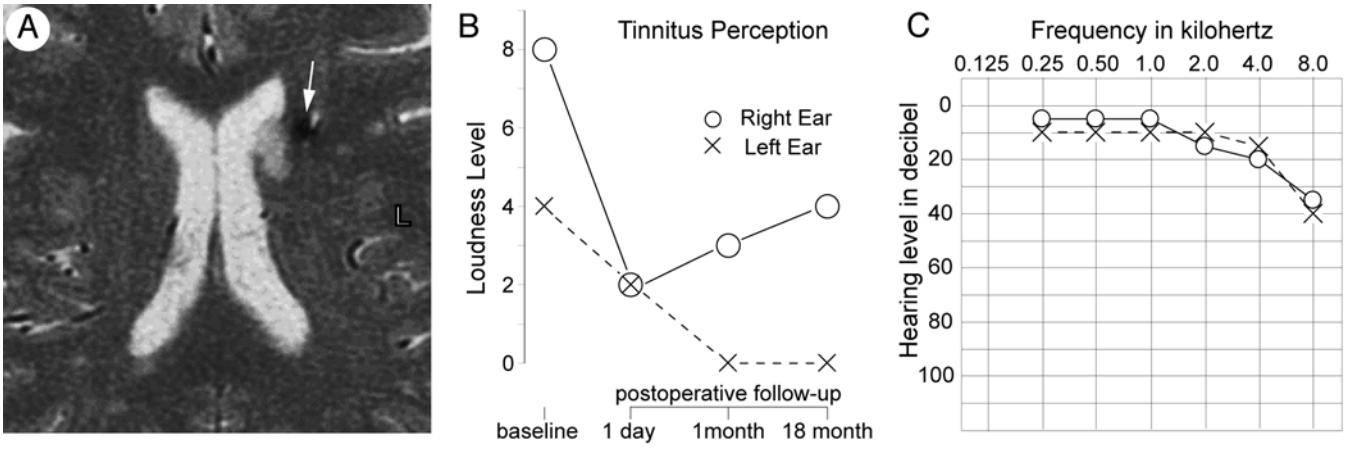

Fig. 1. **A:** Early postoperative MR image showing the DBS lead position *(arrow)* lateral to the caudate nucleus and focal T2 signal hyperintensity involving the anterior body of the caudate. **B:** Graph showing tinnitus perception changes in the right and left ears. **C:** Postoperative audiogram documenting symmetric mild high-frequency sensorineural hearing loss, without asymmetry of hearing thresholds.

shown by our group or others to have any effect on tinnitus perception.

The risk of symptomatic or asymptomatic vascular injury during DBS surgery is generally believed to be in the range of 1%–5%.[1,7] Since many vascular injuries are asymptomatic, routine postoperative imaging with CT or MRI is valuable for detecting the presence of these complications. In our case, the patient's observation of tinnitus loudness suppression would not have been attributed to a CNS event if routine postoperative imaging had not been obtained. The vascular injury was detected on a post–DBS implantation MRI protocol with limited sequences; based on the obtained images, findings were most consistent with a focal venous infarction. No diffusion-weighted sequences or CT scans were obtained to further characterize the findings, as the patient was otherwise asymptomatic. Repeat MRI 16 months after her surgery for a contralateral DBS implant showed cystic encephalomalacia of the caudate, consistent with a prior vascular injury.

This case report provides further evidence that area LC is involved in the perception of auditory phantoms. Our group has previously enrolled patients with movement disorders and comorbid tinnitus in studies that involved transient stimulation in area LC.[2,3] Patients who were stimulated in area LC reported changes in tinnitus loudness; none of those study patients had any evidence of vascular injury or peri-electrode signal change in the caudate on postoperative MRI. The patient described in the present case was not participating in a research protocol and was not stimulated in or around area LC at any time. She was only asked to quantify changes in her auditory phantom after the infarct in area LC was seen on routine postoperative MRI and she noted that her tinnitus had changed on postoperative examination.

This region is frequently traversed during DBS surgery for movement disorders, and yet our clinical assessments during and after surgery focus mainly on motor function. As tinnitus is a relatively common disorder and is not infrequently present in those undergoing DBS surgery, we urge clinicians to consider including assessment of auditory phantoms as part of the comprehensive neurological evaluation.

Conclusions

Focal vascular injury of the dorsal striatum in a patient with PD and tinnitus produced long-term tinnitus suppression. Tinnitus modulation consequences arising from direct electrical stimulation and destructive lesioning of area LC provide evidence that the dorsal striatum plays an important role in gating auditory phantom percepts. Although DBS for medically refractory movement disorders has largely focused on motor outcomes, its potential impact on dorsal striatal function suggests that the comprehensive neurological evaluation should also include phantom percept assessment.

Disclosure

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References

1. Binder DK, Rau GM, Starr PA: Risk factors for hemorrhage during microelectrode-guided deep brain stimulator implantation for movement disorders. **Neurosurgery 56:**722–732, 2005
2. Cheung SW, Larson PS: Tinnitus modulation by deep brain stimulation in locus of caudate neurons (area LC). **Neuroscience 169:**1768–1778, 2010
3. Larson PS, Cheung SW: Deep brain stimulation in area LC controllably triggers auditory phantom percepts. **Neurosurgery 70:**398–406, 2012
4. Lowry LD, Eisenman LM, Saunders JC: An absence of tinnitus. **Otol Neurotol 25:**474–478, 2004
5. Selemon LD, Goldman-Rakic PS: Longitudinal topography and interdigitation of corticostriatal projections in the rhesus monkey. **J Neurosci 5:**776–794, 1985
6. Yeterian EH, Pandya DN: Corticostriatal connections of the superior temporal region in rhesus monkeys. **J Comp Neurol 399:**384–402, 1998
7. Zrinzo L, Foltynie T, Limousin P, Hariz MI: Reducing hemorrhagic complications in functional neurosurgery: a large case series and systematic literature review. Clinical article. **J Neurosurg 116:**84–94, 2012

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