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Hearing thresholds and fMRI of auditory cortex following eighth cranial nerve surgery

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Abstract

Objective—Determine whether auditory cortex (AC) organization changed following eighth cranial nerve surgery in adults with vestibular-cochlear nerve pathologies. We examined whether hearing thresholds pre- and post-op correlated with increased ipsilateral activation of AC from the intact ear.

Study Design—During magnetic resonance imaging sessions pre- and 3 and 6 months postoperation, subjects listened with the intact ear to noise-like random spectrogram sounds.

Setting—Departments of Radiology and Otolaryngology of Washington University School of Medicine.

Subjects and Methods—Three with acoustic neuromas received Gamma Knife radiosurgery (GK); one with Meniere's disease and five with acoustic neuromas had surgical resections (SR); two of the latter also had GK. Hearing thresholds in each ear were for pure tone stimuli from 250 to 8000 Hz before and after surgery (3 and 6 months). At the same intervals, we imaged blood oxygen level-dependent responses to auditory stimulation of the intact ear using an interrupted single-event design.

Results—Hearing thresholds in two of three individuals treated with GK did not change. Five of 6 individuals became unilaterally deaf after SRs. Ipsilateral AC activity was present pre-op in 6/9 individuals with ipsilateral spatial extents greater than contralateral in 3 of 9. Greater contralateral predominance was significant especially in left compared to right ear affected individuals, including those treated by GK.

Conclusion—Lateralization of auditory evoked responses in AC did not change significantly post-op possibly due to pre-existing sensory loss before surgery, indicating that less than profound loss may prompt cortical reorganization.

Keywords

Auditory cortex; Acoustic Neuroma; Unilateral Deafness; Neuroplasticity; Gamma Knife Radiosurgery; functional imaging

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INTRODUCTION

Monaural acoustic stimulation evokes larger responses contralateral to the stimulated ear with normal hearing. This results in hemispheric asymmetry in auditory cortex (AC), characterized by greater contralateral compared to ipsilateral response magnitudes and spatial extents to the stimulated ear. According to one study, contralateral dominant responses only occur with left ear stimulation,1 but others reported contralateral asymmetry with auditory inputs to either ear.2–4 In contrast, with unilateral hearing loss, stimulation of the intact ear evokes greater ipsilateral than contralateral activity, especially notable in core and adjacent belt AC fields.3–10 Neural changes occur immediately, from periphery to cortex, following deafferentation in many sensory systems.11–17 For example, in adult guinea pigs enhanced ipsilateral auditory evoked responses occurred in AC within 2–3 weeks after unilateral cochlea hair cell damage.18 Sudden unilateral hearing loss in humans similarly reduced asymmetrical AC activity within days.19–22 After surgical resection (SR) of a left acoustic neuroma, one study reported symmetrical bilateral responses to 1 kHz tone bursts at 5 weeks and expanded ipsilateral activation at 1 year post SR.9 Comparably, auditory evoked field potentials (AEF) were larger and had shorter latencies for ipsilateral compared to contralateral ear inputs at 1 month after SRs.10,23 However, one study reported near normal contralateral asymmetrical AEF response magnitudes and shorter latencies to hearing speech and non-speech sounds with an intact left ear and right ear deafness. For those with an intact right ear and left ear deafness, significant ipsilateral activation occurred.8 These differences among studies warrant additional investigation of altered asymmetrical activation to monaural stimulation in auditory cortex.

We evoked responses in AC with random spectrogram sounds (RSS) before and at 3 and months after surgical treatment for unilateral eighth cranial nerve pathology. Only one of 6 individuals treated with a SR, had preserved hearing on the affected side. In three individuals treated with Gamma Knife radiosurgery (GK), pre-op hearing persisted in 2; in 1, hearing decreased but did not reach a profound level. These results are consistent with individuals receiving GK treatments in multi-institution meta-analyses24,25 where >60% had hearing preservation. Given hearing differences with treatment modalities, we also determined whether individuals treated with GK showed less lateralization reorganization in AC.

METHODS

We measured blood oxygen level-dependent (BOLD) responses using echo-planar imaging (EPI) sequences in 9 individuals before and after surgery. The study was reviewed and approved by the Human Studies Institutional Review Board of Washington University and was in compliance with the Code of Ethics of the World Medical Association (Declaration of Helsinki). All enrolled individuals gave informed consent.

Eight individuals had unilateral acoustic neuromas and one had Ménière's disease (Table 1A). The acoustic neuromas mostly were at the cerebellopontine angle but with varied sizes including several extending intracanalicular (Table 1B). Cases 1, 3, 5, and 6 had surgical resections (SR); cases 2 and 4 had SR followed by GK; and cases 7, 8, 9 had GK exclusively (Table 1B).

We measured monaural audiometric hearing thresholds for each pre- and post-op session with insert phones and pure tone stimuli from 250 to 8000 Hz, presented in a double walled sound booth while using a standard Hughson-Westlake procedure.26

During imaging, individuals heard noise-like random spectrogram sounds (RSS) presented monaurally to the intact ear through magnetic resonance compatible circumaural, cushion

sealed headphones.3 These previously described stimuli3,27,28 result from manipulation and combination of temporal and spectral parameters for 1638 pure tones spanning a 6-octave bandwidth (250–16000 Hz). RSS stimuli have matching average intensities across spectral regions and temporal ranges, thereby avoiding confounds of differing bandwidth, intensity, or duration as specifying variables common to speech. Additionally, there is independent control of spectral and temporal sound complexity. The complexity of RSS was low or high, based on temporal rates (8 for low and 30 Hz for high) or number of spectral bands (3 for low and 16 for high). Participants pressed an optical key to signal detection of an oddball trial in which the complexity of the RSS differed from that during most other trials in an imaging run.3 Stimulus intensity in the intact ear was 70 dB SPL. The stimuli were predictably below audibility for the opposite ear, which was plugged and muffed with an expected mean interaural bone conduction attenuation of 64 dB for the RSS bandwidth. 29 The sound system bandwidth was approximately 160 to 5 kHz with a 10dB/Octave falloff at >5 kHz.

RSS presentations of 2 second durations occurred during 9 s silent intervals in 11 s volume acquisitions (TRs) of an interrupted single event design.30 EPI at the beginning of an immediately following TR had delays of 2–9 s from the onset of a RSS during silence in the preceding TR, which allowed reconstruction of a hemodynamic response.30 Image acquisition, preprocessing procedures, and analyses of BOLD responses were as previously described.3 Briefly, following a GLM analysis, an F-test per voxel assessed whether the BOLD response variance associated with presentation of a RSS stimulus was greater than that due to baseline noise. This test of significance involved no assumptions regarding the hemodynamic response function. Additionally, we transformed F-statistics to equally probable z-scores (F-Zstats) that were multiple comparisons corrected based on Monte Carlo simulations.31 The correction threshold for p=0.05 was z-scores of z=4.0 over 12 face-connected voxels.

Each individual's brain was rendered into the PALS-B12 CARET surface-based atlas32,33 by using Surefit software. The vertex mesh approximated the mid-cortical thickness of each hemisphere in the native brain. We registered volume-based data (VBD) of corrected F-Zstats to vertices based on nearest coordinate neighbors. Next, deformation of each native brain surface to the vertices for the left and right hemispheres of the PALS-B12 atlas normalized the brains.32 These procedures also registered VBD to the atlas coordinate surface space. The deformation maps created for each brain when applied to the native anatomy retained original brain structure but in the atlas coordinate space of vertices. Thus, surface maps for registered F-Zstats were viewable with respect to participant brain anatomy, but with all distribution distinctions between individuals based on a standard number of vertices. The analyses focused on AC areas Te1, Te2, and Te3 as previously described.3 The combined Te areas occupied the posterior superior temporal plane (Figure 1). Te1 encompasses Heschl's gyrus and adjoining caudal rostral areas as part of a core primary auditory cortical field; Te2 is caudal to Te1 within planum temporale and is within the caudal belt cortical field; and Te3 is lateral to Te1 along the superior temporal gyral crown within planum polare and is a component of the lateral belt cortical field.34–38

Spatial extents reflected area measurement within the combined surface of the three Te areas whose uncorrected F-Zstats had a threshold value of > 2.57 (i.e., $p < 0.005$). The boundaries of these areas reflected brain anatomy in each individual brain. We computed a lateralization index (LI) across the Te combined surface area for pre- and each post-op imaging session and for the measurements of surface area that was ipsilateral and contralateral to stimulation of the intact ear.2,39,40 The LI was in percent: LI = 100 × [measure (contralateral) − measure (ipsilateral)] / [measure (contralateral) + measure (ipsilateral)]. Positive LIs indicated greater spatial extents contralateral to the monaurally stimulated ear.

RESULTS

Hearing Loss

The audiograms in 5 individuals showed profound hearing loss across all frequencies at 3 months post-op (Figure 2, #1–4, 6). These losses at 120 dB hearing level were in the right ear for #1–4 and left ear in #6. Four other individuals had a moderate to severe sloping high frequency loss. Three had losses pre-op and thresholds were unchanged at 3 months postop (Figure 2, #5, 7, and 8). One individual showed a hearing loss at 3 months post op (Figure 2, #9). Three of the four with minimal or no change in hearing threshold following surgery had GK treatments (Table 1B, Figure 2, #7–9). The group pre-op pure tone average (PTA) hearing threshold was ~40 dB hearing level for 3 frequencies (0.5, 1, and 2 kHz) and ~49 dB hearing level for the full frequency range (Table 1A). Hearing levels in the intact ear were mostly normal or showed a mild sloping high frequency hearing loss that did not change after treatment. Laterality of Activity in AC: Significant pre-op activation in the combined Te areas was ipsilateral to the intact ear in 7 of 9 individuals (Figure 3A1 and B1) and was contralateral to stimulation in all individuals (Figure 3A2 and B2), indicating bilateral activation of AC to monaural stimulation in most individuals. Patches of contralateral activity with p values of <0.0001 occurred in ipsilateral AC in 4 (Figure 3A1, B1, Pre-op: red painted patches) and in contralateral AC in 5 individuals (Figure 3A2 and B2). Bilateral activity was present in all but individual #7 at 3 months post-op. All 7 available individuals at 6 months post-op showed some bilateral activity (Figure 3).

Surface Areas

The lateralization index (LI) used surface areas to quantify the balance between ipsilateral compared to contralateral activation distributions (Figure 4). Prior to surgery, 3 individuals with an intact left ear (#1, 2, and 4) showed a higher ipsilateral LI percent. Two others with intact left ears and all with good right ears had a higher contralateral LI percent (Figure 4). At 3 months post-op, 4 individuals with intact left ears had higher ipsilateral LIs (Figure 4, #1, 3–5). Two of these 4 had LIs that reversed from a pre-op contralateral LI (Figure 4, #3 and 5). At 6 months post-op, individuals with intact left or right ears showed reversals from contralateral LIs at 3 months post-op to ipsilateral LIs.

The right ear affected individuals, who had increased ipsilateral activated surface areas prior to surgery (Figure 4, #1, 2, and 4), possibly showed this lateralization due to prior small elevations in hearing thresholds (Figure 2). Of the 4 individuals with affected right ears and total hearing loss after surgery, two had persistent ipsilateral predominance (#1 and 4), one gained ipsilateral activation (#3), and one switched to contralateral (#2). The right ear affected individual #5 with no post-op change in the hearing threshold showed a switch from contralateral to nearly symmetrical lateralization.

All left ear affected individuals (#6–9) showed LIs favoring contralateral AC extents through all imaging sessions even though some ipsilateral activity was present in all but individual #7. Only individual #6 sustained total hearing loss whereas the other three received GK treatment and showed no alteration in pre-op hearing thresholds.

In summary, prior to any treatments, 8 individuals showed some ipsilateral activation but only three with intact left ears showed a higher ipsilateral than contralateral lateralization index. LIs indicating a contralateral bias occurred in 6 individuals and 3 of them had GK treatments. Lateralization distinctions for significant activity in AC did not vary with the stimulated ear.

The regression of hearing thresholds with surface areas ipsilateral to the intact ear was not significant prior to surgery but reached significance at the 3 months post-op (Figure 5 A1

and A2). The lowest spatial extents ipsilateral to stimulating the intact ear occurred in individuals treated with GK and whose hearing thresholds in the affected ear changed the least. There was no significant regression for contralateral surface areas (Figure 5 B1 and B2).

DISCUSSION

Most individuals showed bilateral activation of auditory cortex before and after eighth cranial nerve surgery, indicating little evidence for treatment induced reorganization of lateralization in auditory cortex. These results are at variance with a study of one individual with an affected right ear who had predominant contralateral activation before and a shift towards more symmetrical activation following a surgical resection of an acoustic neuroma.9 This individual had matching normal binaural hearing thresholds before and total right hearing loss after surgical resection of the neuroma. In contrast, hearing thresholds before surgery of 9 individuals in the current study were above the 15–25 dB hearing level of age-matched normal hearing individuals tested by us3 and the pre-op hearing threshold reported previously.9 Elevated hearing thresholds in the affected ear before surgery were prevalent in those with acoustic neuromas even without a hearing loss perceived by the individual.25,41 Consequently, the single individual described by Bilecen and colleagues might have been exceptional rather than representative.

Auditory cortex lateralization varies in different auditory cortical fields and also differs with unilateral deafness in left and right ears. In chronic left ear deafness, right ear stimulation evoked greater left hemisphere (contralateral) response magnitudes only in primary auditory cortex, greater right hemisphere (ipsilateral) responses in belt area Te3, and equal magnitudes in bilateral parabelt areas in comparison to activation from monaural right ear stimulation in normal hearing individuals.3 In the current study, a contralateral spatial activation asymmetry occurred in the left hemisphere (contralateral) of the three left ear affected cases receiving GK treatments. However, this asymmetry was present even before surgery. Auditory cortex lateralization in right ear affected individuals did not confirm prior findings that right ear deafness leads to fewer examples of changes in contralateral (right hemisphere) asymmetry according to AEF measures.6,8 As noted previously in comparing individuals with chronic right ear deafness to those with normal hearing, left ear stimulation evoked larger left hemisphere (ipsilateral) response magnitudes in primary-core auditory cortex, larger responses in right hemisphere (contralateral) belt auditory fields (Te2 and Te3), and equivalent response magnitudes in bilateral parabelt areas.3 These prior findings were consistent with the current finding of larger left hemisphere (ipsilateral) spatial activation extents even prior to surgery in 3 of 5 right ear affected patients. Thus, contrary to prior speculations, there was no evidence that functional plastic changes were more prevalent in the right than in the left auditory cortex. More important, the presence of auditory cortex lateralization changes in the studied cases prior to surgery and the larger sample data set of previously studied chronic cases did not support the speculation that the right temporal lobe has a greater potential for structural re-organization possibly involving re-myelination.6,8 However, our data relied on activation evoked by RSS stimulation and might not reflect auditory cortex lateralization evoked by speech inputs in different intact ears of individuals with unilateral deafness.

A clinically important and unexpected finding was minimal, non-significant changes in auditory cortex lateralization from pre- to post-op imaging sessions with monaural stimulation. A practical implication of this finding is that the studied individuals already sustained some deafferentation prior to surgery. Others have previously found audiograms with elevated thresholds in some patients who were unaware of hearing loss.24,25 Despite preservation of preop hearing levels after gamma knife surgeries, the current findings

realistically indicate little likelihood of reversing altered auditory cortex lateralization changes that resulted from pre-op hearing loses. These observations suggest that neuroplasticity in pre-op auditory cortex reflected the effects of possibly less than complete unilateral hearing loss. Reversible lateralization shifts can also occur without material deafferentation as shown by such changes following sudden short-term functional yet reversible deafness.19,21,22 Several studies in animals have shown that partial damage to isolated portions of the cochlea can provoke auditory cortex reorganization of tonotopic maps.13,15,42 Similarly, cortical reorganization with minimal sensory deficits is not an exclusive property of the auditory cortex as shown by changes in the somatosensory system of adult animals and humans experiencing sensory deafferentation.11,43,44 Another clinically significant finding was confirmation of prior reports that gamma knife surgeries better maintained pre-op hearing levels.24,25 Additionally, however, GK, in better preserving the eighth nerve, functionally supported the lateralization pattern in auditory cortex, especially a more normal contralateral asymmetry despite enhanced ipsilateral activity not normally present in most normal hearing individuals. Thus, lateralization patterns found before surgery persisted in post-op imaging sessions because GK possibly did no or minimal further nerve damage. The observed lateralization reflected what pre-existing nerve injury had already instigated. An important notion, however, was that even optimal tumor excision did not reestablish a normal auditory cortex organization because the pre-existing tumor already induced nerve pathology.

Alterations in crossed inhibitory connections normally present with ipsilateral inputs possibly provide the underlying mechanism responsible for the effects of partial unilateral deafferentation before surgery. Altered inhibition probably arises from changes in the auditory brainstem45 and also interhemispheric cortical connections that influence local inhibitory synapses.14,16 The observed increase in ipsilateral spatial extents to auditory stimulation of an intact ear might have indicated prior deafferentation and reduced inhibition of crossed inhibition even without severely affecting hearing levels. A relevant future clinical objective might involve direct attempts to effect crossed inhibition through micro-stimulation of interhemispheric auditory connections or the auditory brainstem, above the damaged eighth nerve.

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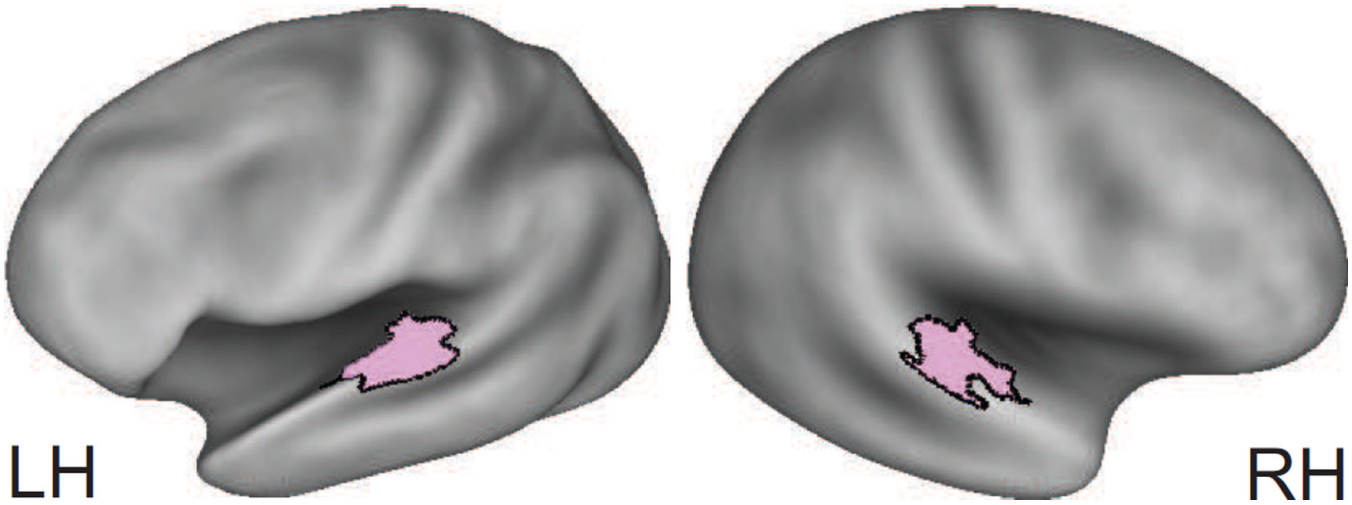

Figure 1.
Combined surface areas for Te1, Te2, and Te33 (painted pink and within black borders) shown on very inflated PALS-B12 Caret Atlas. The view into the sulcus is tilted and rotated.

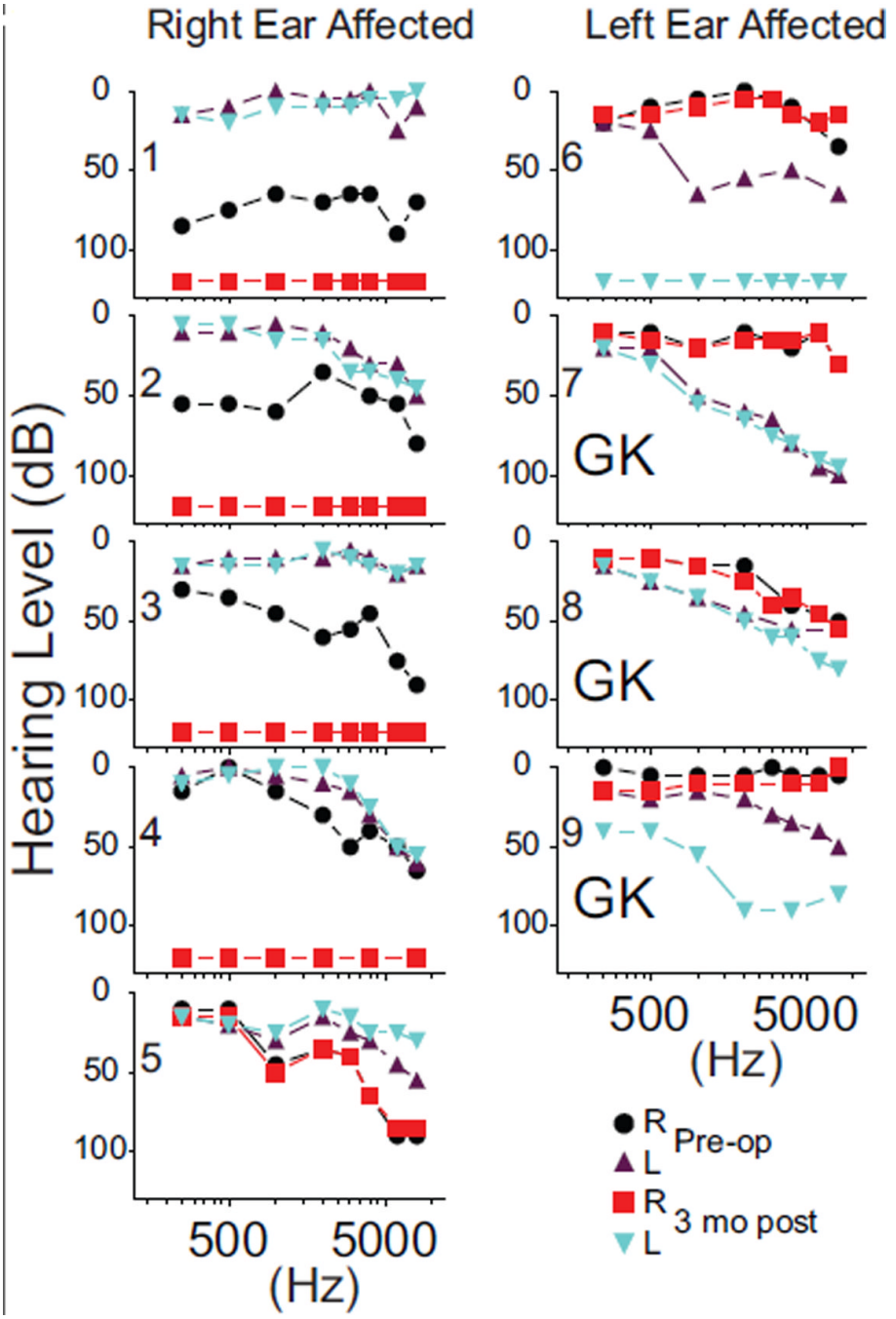

Figure 2.
Pure-tone semilog audiometry plots prior to (preoperative) and 3 months after (3 months postoperative) eighth nerve resections in cases with right or left affected ears. GK indicates, Gamma Knife radiosurgery.

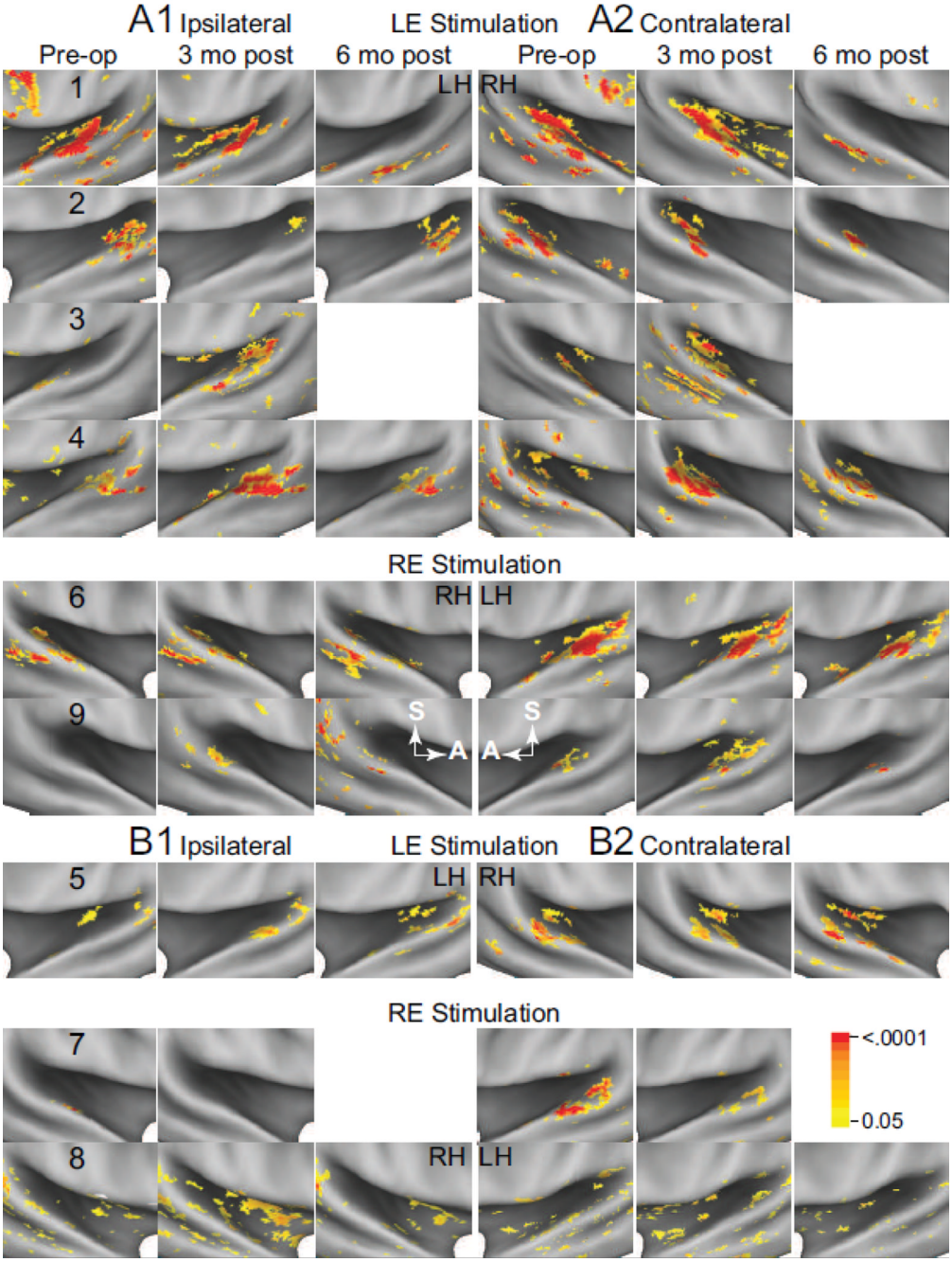

Figure 3.

Z score maps for individuals with (A) and without (B) hearing loss. View as shown in Figure 1. White arrows A and S indicate anterior and superior directions in each hemisphere. LH indicates left hemisphere; RH, right hemisphere.

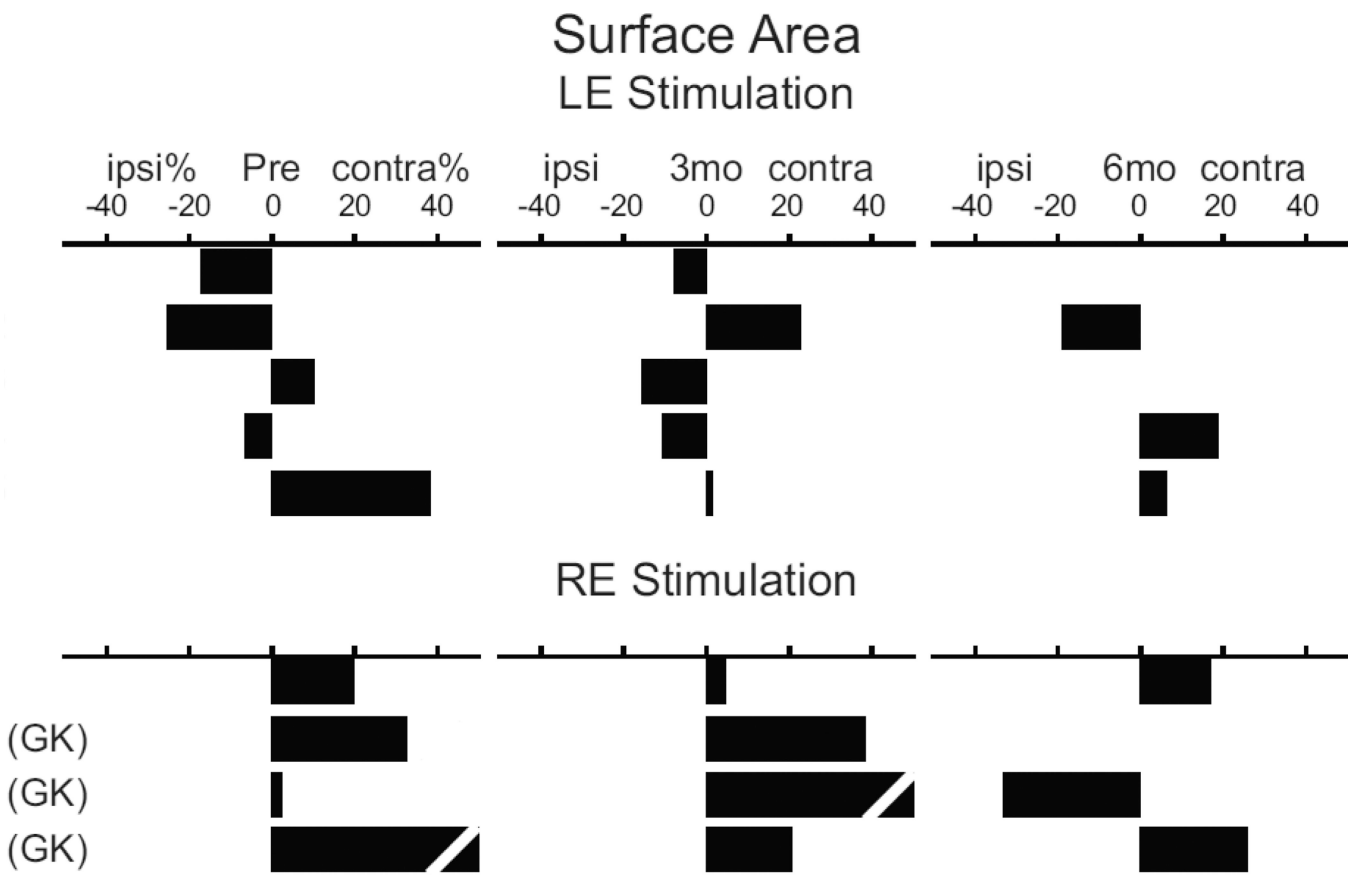

Figure 4.
Laterality index for surface areas within auditory cortex for preoperative and postoperative imaging sessions. Diagonal gaps in bars for 8 and 9 indicate LI% that exceeded scale maximum. LE indicates left ear; RE, right ear.

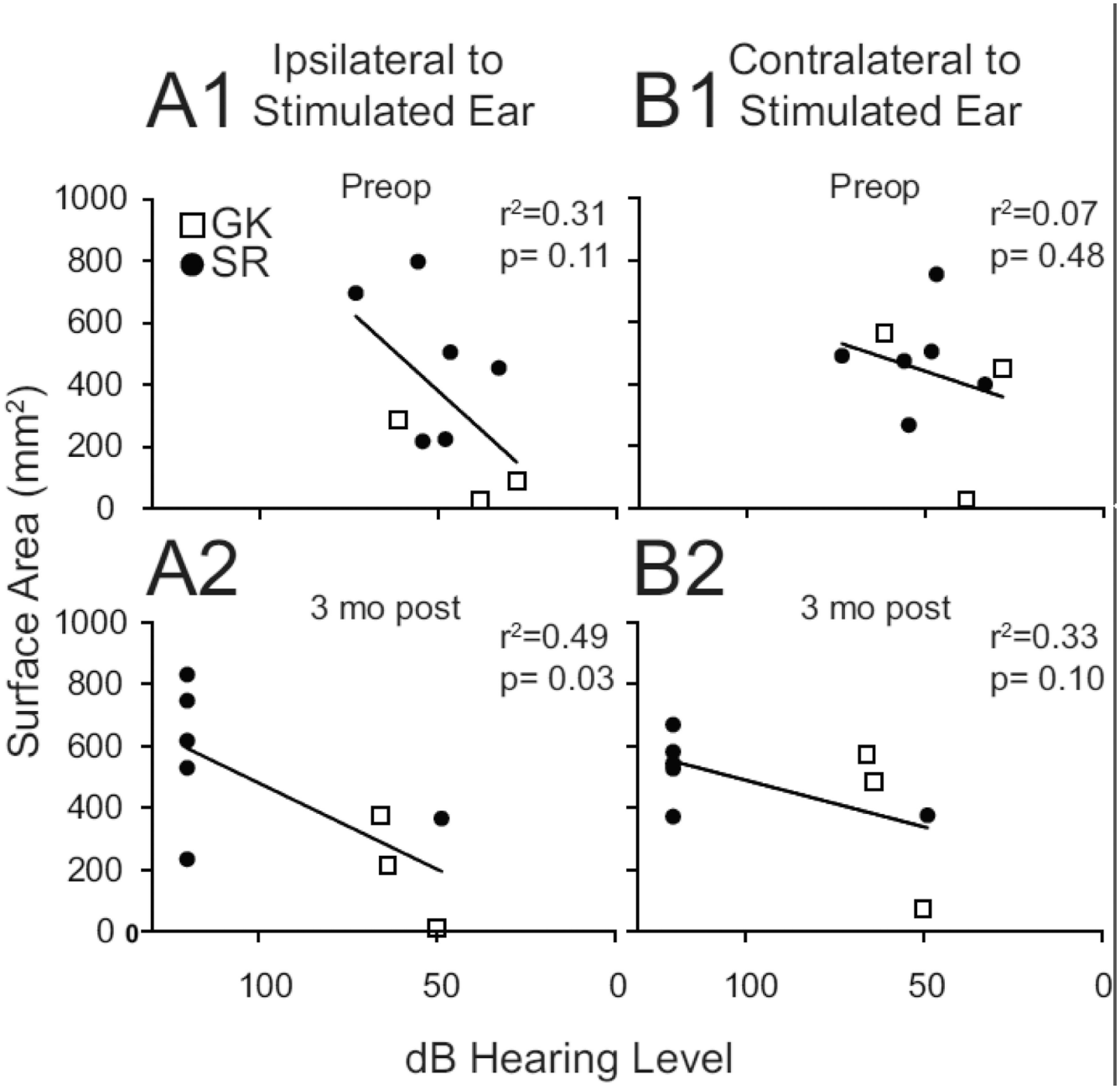

Figure 5.
Regression analyses of surface area relative to hearing levels in auditory cortex ipsilateral (A1, 2) and contralateral (B1, 2) to the stimulated intact ear. GK indicates Gamma Knife radiosurgery; SR, surgical resection.

Table 1

Patient demographics and hearing thresholds in the affected ear.

				Affected Ear Thresholds					
Case	**Affected Ear**	**Gender**	**Age[a]**	**3fPTA Pre**	**FFPTA Pre**	**3fPTA 3 mo[b]**	**FFPTA 3 mo**	**3fPTA 6 mo**	**FFPTA 6 mo**
1[c]	R	F	28	70.0	73.1	120.0	120.0	120.0	120.0
2	R	M	50	50.0	55.7	120.0	120.0	120.0	120.0
3	R	F	43	46.7	54.4	120.0	120.0	NA[d]	NA
4	R	M	45	15.0	33.1	120.0	120.0	120.0	120.0
5	R	F	47	30.0	48.1	33.3	48.8	35.0	49.4
6	L	M	39	48.3	46.7	120.0	120.0	120.0	120.0
7	L	M	52	43.3	61.3	50.0	63.8	NA	NA
8	L	F	56	35.0	38.3	36.7	50.0	38.3	40.8
9	L	F	53	18.3	28.1	61.7	65.8	61.7	61.1
		Mean	45.9	39.6	48.8	86.9	92.0	87.9	90.2
		SEM	2.9	5.7	4.7	13.4	11.2	15.5	14.2

Tumor size and location per case.

Case	**Size (cm)**	**Volume**	**Location**	**Treatment[e]**
1	n/a[f]	n/a	n/a	SR vestibular nerve
2	3.4 × 3.2		CPA, residual tumor	Radical subtotal SR followed with GK
3	2		CPA, extending in	SR
4	2.7 × 2.2 ×		CPA, extending in	SR followed with GK
5	0.5 × 1		IAC	Middle fossa craniotomy
6	1.6 × 2.1 ×		CPA	Sub-occipital craniotomy with SR
7	1.2 × 1	683.4	CPA, extending in	GK
8	0.6	217	IAC mid canal	GK
9		1700	CPA, extending in	GK

[a] Age at time of study enrollment.

[b] One hundred twenty assigned to all thresholds with no response to test equipment limits.

[c] Case 1 had Meniere's syndrome.

[d] Patient not available for threshold testing or imaging scans, NA.

[a] Cerebellopontine angle (CPA); intracanalicular (IAC).

[b] Treatments included Gamma Knife radiosurgery, GK and surgical resection, SR.

[c] Not applicable (n/a); other dimensions as noted by surgeons in the patient records.