The Comparative Cortical Activation Study on Conductive and Sensorineural Hearing Loss

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Introduction

Hearing loss is classified into conductive, sensorineural, and mixed types. Disorders of the auditory pathways and inner ear cause sensorineural hearing loss (SNHL) whereas conductive hearing loss is resulting from disorders of outer or middle ear. For two types of hearing loss, the comparative cortical activation study has not been performed using fMRI. In this study, we evaluate and compare the brain functions of conductive and sensorineural hearing loss patients on auditory stimulation using BOLD fMRI techniques.

Material and Methods

Subjects: Five conductive hearing loss patients (3 males, 2 females; age: 1-56 years; mean age: 35 years) and five profound sensorineural hearing loss patients (2 males, 3 females; age: 2-7 years; mean age: 4 years) were included in this study. In addition, three normal controls (2 males, 1 females; age: 24-26 years; mean age: 25 years) with no history of auditory abnormalities were included. All subjects provided written informed consent in accordance with protocols approved by the institutional review board.

Functional MR Imaging: fMRI images were acquired on a 1.5T Vision Plus scanner (Siemens, Erlangen, Germany) with gradient strength of 24 mT/m using standard head coil. The BOLD T2*-weighted images were acquired with echo planar imaging sequence (TR = 6.0 sec, TE = 60 msec, and flip angle = 90°). The other sequence parameters are: FOV = 210 mm, matrix = 64 × 128 or 64 × 64, slice number = 15 and slice thickness = 5 mm. The anatomic images were obtained with spin-echo T1-weighted images. Auditory fMRI acquisition was performed using block paradigm of 500 Hz and/or 2KHz pure tone stimulation. To avoid possible interference with acoustic scanner noises, the ear muff and ear plug were used. In addition, since the Blood Oxygen Level Dependent (BOLD) fMRI has physiological delay between onset of stimulation and cortical activation, we used long repetition time of 6 sec and pure tone stimulation was applied without any gradient switching noise. The patients were asked to passively listen to the tone during fMRI session.

Statistical Analysis: the raw images from fMRI measurement were transfered to independent workstation and post-processed to generate functional map using .Statistical Parameter Map (SPM) 99. Statistical significance level was adjusted for each patients and typically is less than $p<0.05$.

Results

For normal subjects, the typical fMRI finding was the primary auditory cortex (Brodmann area 41,42) activation to sound stimulation (Fig. 1). Cortical responses of conductive hearing loss patients were that the auditory cortex (primary and secondary), which is contralateral to normal ear, shows strong activation but the auditory cortex, which is contralateral to affected ear, is weakly activated. Fig. 2 shows the auditory activation of a patient with right ear otosclerosis. This conductive hearing loss patient showed the diffuse and weak activations on the left primary and secondary auditory cortex, which is contralateral to otosclerositic ear. For profound SNHL patients, the typical cortical activation patterns were that primary auditory cortex did not response to sound stimulation. Instead, our preliminary results indicated the broad activation of secondary auditory cortex, which is adjacent to primary cortex (Fig. 3).

Discussion

The structure of central auditory pathway in mammals has traditionally been considered to be relatively rigid; however, recent magnetoencephalography (MEG) studies suggest that reorganization may also happen in the human auditory pathway after sensorineural hearing loss as a result of deficit on the auditory pathway [1]. On the other hand, patients with conductive hearing loss show rather normal but weak auditory evoked magnetic fields (AEFs) to auditory stimulation [2]. Our fMRI results suggest the similar findings. That is, our cortical activation study using fMRI showed the difference in response to auditory stimulation between conductive and profound SNHL patients. For conductive hearing loss cases, the major fMRI findings was the activation of primary cortex although the activation was weak in magnitude. Therefore, the auditory pathway seems to be conserved. However, in case of profound SNHL patients, the broad activation of auditory association cortex, rather than primary cortex, was observed. Our results therefore seem to suggest that the neural deficits, which found on most of SNHL patients, make reorganization in auditory pathway and resulted in the diffuse activations on auditory association area, which is adjacent to primary cortex.

References

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