Visual stimuli activate auditory cortex in deaf subjects: evidence from MEG

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Studies using fMRI have demonstrated that visual stimuli activate auditory cortex in deaf subjects. Given the low temporal resolution of fMRI, it is uncertain whether this activation is associated with initial stimulus processing. Here, we used MEG in deaf and hearing subjects to evaluate whether auditory cortex, devoid of its normal input, comes to serve the visual modality early in the course of stimulus processing. In line with previous findings, visual activity was observed in the auditory cortex of deaf, but not hearing, subjects. This activity occurred within 100–400 ms of stimulus presentation and was primarily over the right hemisphere. These results add to the mounting evidence that removal of one sensory modality in humans leads to neural reorganization of the remaining modalities. *NeuroReport* 14:1425–1427 © 2003 Lippincott Williams & Wilkins.

Key words: Auditory cortex; Deafness; MEG; Plasticity; Vision

INTRODUCTION

The ability of the brain to reorganize in response to the removal of input from one sensory modality early in development, referred to as cross-modal plasticity, has been extensively studied in animals (for reviews see [1,2]). In humans, the study of blind and deaf individuals provides an opportunity to examine the perceptual and neural consequences of modality-specific sensory deprivation. For instance, blind subjects possess superior tactile discrimination [3] and auditory localization [4–6] abilities. A neural basis for these sensory enhancements is suggested by brain imaging studies showing activation of primary visual cortex in response to tactile or auditory stimuli of blind subjects [7–11].

Deaf subjects appear to have enhanced tactile abilities [12] and altered visual perception (particularly for motion processing) [13–15]. Few studies, however, have assessed cross-modal plasticity in the auditory cortex of deaf subjects. Levanen *et al.* [16] reported tactile responses in auditory cortex of a deaf subject tested with MEG, but Hickok *et al.* [17] found neither tactile nor visual activation in auditory cortex of another deaf subject studied with fMRI and MEG. Finney *et al.* [18], however, found clear evidence for visual responses to motion stimuli in deaf subjects' auditory cortex using fMRI.

Given the low temporal resolution of fMRI, it is unclear whether visual activity in auditory cortex of deaf subjects reflects initial stimulus processing in auditory cortex or delayed feedback from higher brain areas. To test these competing possibilities, we investigated cross-modal plasticity in a different group of deaf and hearing subjects using MEG and distributed source reconstruction methods. This approach allowed us to evaluate strengths of multiple cortical activities simultaneously during the first few hundred milliseconds of stimulus processing.

MATERIALS AND METHODS

Subjects: Five deaf (three female) and five hearing (three female) right-handed subjects with normal or corrected-to-normal vision provided data for this study. The study was approved by the IRB committee at UCSD, and all subjects gave informed consent. Deaf subjects were born deaf and had learned to sign before age two. Hearing subjects had no exposure to ASL other than finger spelling, and were monolingual English speakers. The mean age of the deaf subjects was 30.2 ± 4.9 years; the mean age of the hearing subjects was 29.8 ± 6.3 years.

Visual stimuli: Stimuli were presented on a ceiling screen above the subjects with a Proxima 4200 LCD projector and mirror system (viewing distance 153 cm). The stimulus consisted of a sinusoidal luminance (black/white) grating (0.7 cycles/deg) subtending the entire field ($34 \times 43^\circ$), with a small fixation cross in the center of the display. A stimulus event consisted of a $5.7 \times 5.7^\circ$ patch of this display temporally modulated at 4.5 Hz. This event occurred once per second for 300 ms. To ensure subjects were attending to the stimulus event, the grating patch either moved (left or right) or counterphase reversed; subjects were required to discriminate (using button presses) between stimulus types. Stimuli were presented at two different contrasts, 10% and 50%, in separate sessions of 1200 stimuli/session. Within a session, stimuli appeared randomly in one of five locations; one in the center and four in the periphery (superior left, superior right, inferior left, and inferior right), centered 15° eccentric to the fixation cross, randomly intermixed. Because no significant differences were found in response to these different stimuli, the results are presented averaging over all conditions.

MEG data acquisition: Neuromagnetic recordings were obtained in a magnetically shielded room using a 148-channel whole-head biomagnetometer (4D Neuroimaging, Inc., San Diego, CA, USA). Eye movements were recorded with electrodes placed above and below the left eye (for monitoring blinks) and at the outer canthi of the two eyes (for monitoring horizontal eye movements). MEG data were recorded for 600 ms beginning 100 ms before stimulus onset at 678 Hz with an analogue filter bandpass of 1–400 Hz. Head shape and fiducial points (nasion, left and right pre-auricular points) were digitized using a Polhemus Fastrak (Polhemus Inc., Colchester, VT, USA) for later co-registration of head position relative to MEG sensor locations prior to analysis.

MEG data analysis: MEG data were screened to remove trials with blinks, horizontal eye movements in response to the peripheral stimuli, and/or other artifacts. Data were averaged and digitally filtered from 1 to 40 Hz with a third-order Butterworth filter. An average of 305 subjects' MR images was used to define brain geometry [19]. A three-compartment realistic (Boundary Element Method) head model was constructed from this averaged MR image using Curry (Version 4.0, Neurosoft, Inc., 1999; see [20]). For all time points we calculated a distributed source reconstruction given the MEG data using the L2 minimum norm constraint as implemented in Curry. Noise estimates were obtained from the 100 ms pre-stimulus baseline. Sources were constrained to reside within the cortex, on which there were 5596 source locations separated by an average of 3.1 mm.

Source results were averaged over the interval from 100 to 400 ms after stimulus presentation and were then smoothed (using routines written in Matlab) by averaging over each source location and its nearest neighbors. To eliminate the possibility that between-subject differences in baseline levels of activity could influence source magnitudes, activity over the whole brain and over time was standardized to mean = 0 and unit variance for each subject. For each subject, the average amount of activity over primary and secondary auditory cortex (Brodmann's areas 41 and 42) was calculated for left and right hemispheres. In addition, activity was measured within the motion area MT complex (at the intersection of Brodmann's area 19, 37, and 39) and the posterior parietal cortex region supporting visual stimulus processing during high attentional demands (at the intersection of Brodmann's areas 7 and 40 [21]).

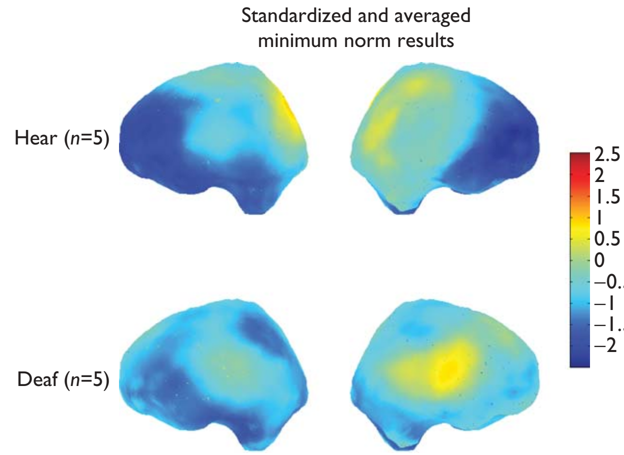

Fig. 1. Standardized distributed source activity for hearing and deaf subjects in response to visual stimuli. Activity was averaged across all stimulus conditions from 100 to 400 ms after stimulus presentation. The figure shows activity over left and right hemispheres projected onto an averaged MR image of the cortical surface. The color scale for the plots is shown at the right.

RESULTS

Figure 1 shows standardized and averaged activity in deaf and hearing subjects in response to our visual stimuli. When averaged over left and right hemispheres, activity in auditory cortex was significantly greater in deaf (0.62 ± 0.31) than in hearing (0.11 ± 0.29) subjects ($t = 2.69$, $p = 0.028$). Importantly, only for deaf subjects was this visual activity significantly above baseline (deaf: $t = 4.49$, $p = 0.011$, hearing: $t = 1.59$, $p = 0.19$). When analyzed separately in the two hemispheres, this effect was stronger in deaf subjects' right hemisphere (mean activity 0.96 ± 0.42) than left hemisphere (mean activity 0.28 ± 0.65), although the difference was only marginally significant ($t = 2.17$, $p = 0.098$). This MEG result corroborates our previous fMRI findings showing significant visual activity in deaf subjects' right auditory cortex [18]. Interestingly, differences between groups were also observed in other brain regions. Specifically, responses in posterior parietal cortex were significantly smaller in deaf (-0.10 ± 0.25) than in hearing (0.42 ± 0.25) subjects ($t = 3.32$, $p = 0.01$), and a similar trend was observed in the area MT complex.

DISCUSSION AND CONCLUSIONS

The results of the present MEG study corroborate our previous fMRI findings demonstrating the recruitment of auditory cortex in deaf subjects for the processing of purely visual stimuli. This MEG result adds significantly to the original finding because the temporal resolution of this technique allows us to demonstrate this effect within the first few hundred milliseconds of stimulus presentation, indicating involvement of auditory cortex in the initial processing of visual stimuli in deaf subjects. This early response suggests that, in deaf individuals, there may be direct projections from the visual thalamus (i.e. the lateral geniculate nucleus) to primary auditory cortex (see [22] for evidence of such projections in early-deafened ferrets). Further mirroring the previous fMRI study, the cross-modal plasticity observed in the present MEG study appeared predominantly within deaf subjects' right auditory cortex. Because both studies employed moving visual stimuli, this hemispheric asymmetry is likely to reflect a predisposition for motion processing in the right auditory cortex. This possibility is supported by the finding that right auditory cortex is specialized for processing auditory motion in hearing subjects [23]. Thus, right auditory cortex in deaf subjects, devoid of its normal auditory input, may come to serve motion processing in the visual modality. Interestingly, the reciprocal result has been reported in blind subjects, among whom responses to moving auditory stimuli are observed predominantly in the right visual cortex [9], suggesting a predisposition towards motion processing in the right hemisphere. In combination with previous findings, the present MEG results attest to the robust ability of the human brain to reorganize in response to the removal of input from one sensory modality early in development.

REFERENCES

1. Rauschecker JP. *Trends Neurosci* **18**, 36–43 (1995).
2. Sur M, Angelucci A and Sharma J. *J Neurobiol* **41**, 33–43 (1999).
3. Blank M and Bridger WH. *Child Dev* **37**, 29–38 (1966).
4. Lessard N, Pare M, Lepore F and Lassonde M. *Nature* **395**, 278–280 (1998).
5. Muchnik C, Efrati M, Nemeth E *et al*. *Scand Audiol* **20**, 19–23 (1991).
6. Roder B, Teder-Salejarvi W, Sterr A *et al*. *Nature* **400**, 162–166 (1999).
7. Cohen LG, Celnik P, Pascual-Leone A *et al*. *Nature* **389**, 180–183 (1997).
8. Sadato N, Pascual-Leone A, Grafman J *et al*. *Nature* **380**, 526–528 (1996).
9. Weeks R, Horwitz B, Aziz-Sultan A *et al*. *J Neurosci* **20**, 2664–2672 (2000).
10. Roder B, Rosler F and Hennighausen E. *Psychophysiology* **34**, 292–307 (1997).
11. Kujala T, Huotilainen M, Sinkkonen J *et al*. *Neurosci Lett* **183**, 143–146 (1995).
12. Levanen S and Hamdorf D. *Neurosci Lett* **301**, 75–77 (2001).
13. Bosworth RG and Dobkins KR. *Psychol Sci* **10**, 256–262 (1999).
14. Bosworth RG and Dobkins KR. *Brain Cogn* **49**, 170–181 (2002).
15. Neville HJ and Lawson D. *Brain Res* **405**, 268–283 (1987).
16. Levanen S, Jousmaki V and Hari R. *Curr Biol* **8**, 869–872 (1998).
17. Hickok G, Poeppel D, Clark K *et al*. *Hum Brain Mapp* **5**, 437–444 (1997).
18. Finney EM, Fine I and Dobkins KR. *Nature Neurosci* **4**, 1171–1173 (2001).
19. Collins DL, Neelin P, Peter TM and Evans AC. *J Comput Assist Tomogr* **18**, 192–205 (1994).
20. Fuchs M, Wagner M, Kohler T and Wischmann HA. *J Clin Neurophysiol* **16**, 267–295 (1999).
21. Merriam EP, Colby CL, Thulborn KR *et al*. *Neuroimage* **13**, 794–800 (2001).
22. Pallas SL, Razak KA and Moore DR. *Soc Neurosci Abstr* **28**, abstr. 220.8 (2002).
23. Baumgart F, Gaschler-Markefski B, Woldorff MG *et al*. *Nature* **400**, 724–726 (1999).

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