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Visual cortex activation decrement following cochlear implantation in prelingual deafened children

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ABSTRACT

Objective: Visual take-over of the auditory cortex in prelingual deaf children has been widely reported. However, there have been few studies on visual cortex plasticity after cochlear implantation (CI). In this study, we investigated the hypothesis that extrinsic auditory stimulation following CI in prelingual deafened children can induce visual cortex plasticity.

Method: Visual evoked potentials (VEPs) were recorded in 37 CI children (4 groups with different use times) and 8 control subjects, in response to sound and nonsound stimuli. Latency and amplitude were analyzed for the P1, N1 and P2 components on the Oz electrode. Comparisons of VEP were conducted between the sound and nonsound stimuli and among different groups in order to view evidence of visual cortex reorganization.

Results: The latency of the P2 component was significantly longer at the occipital site (Oz) in CI 0M than those in the other four groups. After the effect of age was excluded, a significant negative correlation was found between CI usage and P2 latency of nonsound stimuli. Occipital P1N1 latency and P1 amplitude were not affected by group or stimulus category. However, the N1 and P2 amplitudes were significantly larger in response to a sound stimulus than to a nonsound stimulus.

Conclusion: Our findings suggest that P2 latency develops with CI usage and may be a biomarker of visual cortex plasticity.

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1. Introduction

There is increasing evidence for visual take-over of the auditory cortex in prelingual deaf children [1,2]. In other words, if the cortex is deprived of auditory input during development, there exists a distinct possibility that the auditory cortex will take on visual functions [3,4]. Furthermore, prelingual deafened individuals with pronounced cross-modal take-over of auditory regions are less likely to benefit from cochlear implantation (CI) [4,5]. In addition, the literature on adult CI research has reported that cross-modal reorganization from the visual modality is linked to deficits in speech perception performance [6] [7].

Indeed, after CI, the auditory cortex receives auditory stimuli, and the auditory cortex changes after sufficient training. For example, P1 cortical auditory evoked potentials have been reported as a biomarker for central auditory system development and reorganization in congenitally deaf children fitted with CI [8].

In addition to recruitment of the auditory cortex, visual responses in the visual cortex in early deaf individuals have also been found to differ from those of normal-hearing controls, suggesting that the absence of auditory input may additionally induce intramodal reorganization in the visual cortex of early deaf individuals [9]. However, it remains unclear whether visual cortex plasticity occurs in CI children. Thus, the purpose of this study was to

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investigate whether extrinsic auditory stimulation following CI in prelingual deafened children can induce visual cortex plasticity. We recorded visual evoked potentials (VEPs) in control and CI children with different use times.

2. Materials and methods

2.1. Subjects

The subjects included 37 CI children; the mean test age was 5.90 years, ranging from 2.6 to 9.2 years. All CI patients were implanted unilaterally. To ensure that all participants in different group can cooperate the test and to balance implanting age of different group, we choose participants who received cochlear implantation after age 2. The average implant age was 4.53 years (standard deviation = 1.28 years). Table 1 shows the demographic profiles of the CI participants. We divided the 37 children into 4 groups based on the duration of CI usage. These groups consisted of children with CIs that had recently been switched on and those with 9–12 months, 18–24 months and more than 2 years of CI usage. None of these CI patients with prelingual profound hearing loss reported a previous history of special infection, kernicterus or ototoxic drug application. In addition, no inner ear or auditory nerve malformation was found during pre-operative CT and MRI evaluations. All subjects were right-handed and reported no visual problems. None of the subjects hand any record of neurological or psychiatric illness.

Eight children (aged 6.23 years, standard deviation = 1.37 years) with moderate-severe conductive hearing loss (congenital external middle ear malformation) were used as the normal control group. Ethical approval was obtained from the Institutional Review Board at Sun Yat-Sen Memorial Hospital of Sun Yat-Sen University before the study began. Written consent was obtained from the parents of all subjects before any of the study procedures were conducted.

2.2. Methods for VEPs

2.2.1. Visual stimuli

Following the concepts of a 'sound photo' and 'nonsound photo' first reported by Proverbio [10], in our study, one 'sound' photo (a photograph with imaginative sound) and one 'nonsound' photo (a photograph without imaginative sound) were presented as visual stimuli. To make sure that the subjects concentrated on the stimuli, one deviant photo (wolf) was presented. Subjects were comfortably seated in front of a high-resolution 19-inch VGA computer monitor at a viewing distance of approximately 1 m in a soundproof and electromagnetically shielded room. A DELL computer running the E-prime®2.0 program controlled the experiment. Fig. 1 shows the experimental block design, which consisted of an intermittent stimulus mode with a total of 100 'sound photo' trials and 100 'nonsound photo' trials divided into two blocks. The duration of each image presentation was 1 s, which was followed by blank-screen inter-stimulus intervals (ISI) ranging from 1.2 to 1.7 s. Each presented blank stimulus image included a fixation point (a white cross) at the center of the screen. The children were instructed to keep their eyes on the pictures and were requested to respond to only the wolf picture by pressing the blank button. The subjects were allowed to have a rest between blocks.

2.2.2. EEG recording and analysis

A 128-channel electroencephalography (EEG) electrode recording system (Electrical Geodesics, Inc.) was used to record the VEPs. Children with CI removed the external processor during testing. The sampling rate for the EEG recording was 1 kHz, and all electrode impedances remained below 50 kΩ.

The EEG recordings of each child were bandpass filtered offline at 0.3–30 Hz and segmented with 100 ms pre-stimulus and 600 ms post-stimulus time. Artifact rejection set at 200 μV was applied to visual EEG, and epochs were rejected if they contained any eye blinking (eye channel exceeded 140 μV) or eye movement (eye channel exceeded 55 μV). Bad channels were removed from the recording. Data were then re-referenced using a common average reference. The data were baseline corrected to the pre-stimulus period of −100 to 0 ms. The 'sound photo' and 'nonsound photo' stimuli were added together, and individual waveform averages were averaged together for each of the groups to compute a grand average waveform.

Amplitudes and latencies of the P1-N1-P2 complex on the Oz electrode for individual participants were analyzed. The highest positive amplitude between 110 and 180 ms was selected for P1. The N1 component was defined as the highest negative amplitude between 180 and 290 ms. In addition, the P2 component was observed as the second positive-going peak with a latency approximately between 240 and 400 ms. The amplitude of the P170, N1, and P2 peaks was measured from baseline to the peak value. Latencies were chosen at the highest amplitude of the peak. The amplitudes and latencies of the VEP components were analyzed with respect to two independent factors: the stimulus type (sound or nonsound photo) and different group (five groups).

3. Results

The grand average VEP waveforms recorded for sound and nonsound stimuli of the five groups at the occipital (Oz) electrode site are shown in Fig. 2. Three obligatory cortical VEP components elicited in response to the visual stimulus were analyzed: P1 (occurring at approximately 150 ms), N1 (occurring at approximately 220 ms), and P2 (occurring at approximately 310 ms). We calculated the peaks for each of the three components (P1, N1, P2) at the maximum amplitudes recorded at the Oz location (occipital) and compared the amplitudes and latencies of these components between the five groups. The mean and standard deviation (SD) of VEP latencies and amplitudes recorded at the Oz location (occipital) in response to the sound and nonsound stimuli of the five groups are presented in Table 2 and Table 3. The latencies and amplitudes of the P1, N1 and P2 components were analyzed by two-way repeated-measures analysis of variance (rANOVA) with one between-group factor (group) and one within-group factor (type of stimulus).

For P2 latency, significant main effects of group ($F = 3.665$, $p = 0.012$) and stimulus category ($F = 6.097$, $p = 0.018$) were found with no significant group*stimulus category interaction ($F = 1.550$, $p = 0.207$). Table 4 summarizes the results of further comparison among different groups. The latency of the P2 component was significantly longer at the occipital site (Oz) in CI 0M than in the other four groups. Partial correlation analysis was then used to analyze the correlation between CI usage and P2 latency with age as controlling variable. After the effect of age was excluded, a significant negative correlation was found between CI usage and P2 latency of the nonsound stimulus ($r = -0.374$, $p = 0.025$).

Occipital P1 latency and amplitude were not affected by group ($F = 0.233$, $p = 0.918$; $F = 0.440$, $p = 0.779$, respectively) or stimulus category ($F = 0.001$, $p = 0.980$; $F = 2.227$, $p = 0.143$, respectively). N1 latency was also not affected by group ($F = 0.510$, $p = 0.729$) or stimulus category ($F = 0.263$, $p = 0.611$). However, the N1 and P2 amplitudes were significantly larger in response to sound stimuli than to nonsound stimuli ($F = 4.753$, $p = 0.035$; $F = 4.320$, $p = 0.044$, respectively), and no significant group effect ($F = 0.746$, $p = 0.566$; $F = 0.633$, $p = 0.642$, respectively) or significant group*stimulus category interaction ($F = 0.229$, $p = 0.920$;

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