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# Altered visual-evoked potentials in congenitally deaf adults

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Visual-evoked potentials recorded from the scalp of congenitally deaf adults were significantly larger over both auditory and visual cortical areas than in normal hearing adults. Over temporal and frontal areas peripheral stimuli presented at long intervals elicited N150 components which were larger in deaf than in hearing subjects. Over occipital and parietal areas peripheral and foveal stimuli elicited larger P230 components in deaf than in hearing subjects. These results imply that early auditory experience influences the organization of the human brain for visual processing.

The immature nervous system has a remarkable capability for reorganization under the influence of environmental stimulation. Both enriched and reduced sensory experiences during specific developmental periods have been found to alter not only the fine structure of the nervous system but also the physiology and electrophysiology of the cortex[4,5,7,16]. Most of these studies have investigated the effects of early experience on brain areas directly associated with the altered sensory modality, rather than on the organization of areas that subserve other modalities. There is, however, some evidence suggesting that two types of compensatory neural reorganization may occur after unimodal sensory deprivation.

First, neural systems that normally develop to subserve the deprived modality may process information from the remaining modalities instead. This effect may underlie reports of the abnormal extension of auditory-evoked potentials (EPs) to visual cortical areas in early blinded cats and of visual EPs extended to auditory areas of congenitally deaf cats[2,11]. Second, the brain areas subserving the remaining modalities may develop increased capabilities or 'compensatory hypertrophy'. In support of this possibility are the reports of an abnormal increase in axons, dendrites, and dendritic spines in auditory cortical areas in mice visually deprived since birth[8,12] and reports of compensatory hypersensitivity after unimodal deprivation in man[1]. Very little is known of the neurophysiological processes which underlie such changes in humans; however, the evoked potential (EP) technique offers a unique means of addressing these questions in humans.

In particular, several studies have demonstrated that two of the modality-specific EP components elicited by simple sensory stimuli are sensitive to the rate of stimulus presentation. The amplitude of both the N1 (100–150 ms) and the P2 (170–250 ms) components are reduced as the stimulus repetition rate is increased. This variation in responsiveness with interstimulus interval (ISI), known as the relative refractory period or recovery cycle, has been regarded as a measure of the excitability of cortical neurons under stimulation and may reflect the rate of sensory processing in different cortical areas[6].

In the present investigation we compared the refractory periods of visual EPs recorded over visual and auditory brain regions in normal hearing and congenitally deaf adults. We assessed the refractory periods separately for stimuli presented to the periphery and the center of the visual

field to test the hypothesis that the different neural systems postulated to mediate these two types of stimuli[9] might be differentially affected by early auditory experience.

The subjects were 13 normally hearing and 8 deaf adults (mean age: hearing 23 years; deaf 26 years). Each subject had normal or corrected vision. All deaf subjects were profoundly and congenitally deaf, but were free of other neurological disorders. During testing subjects fixated on a small white dot in the center of a darkened video screen 61 cm in front of them. A white rectangle (1.2 degrees of visual angle wide by 0.6 degrees wide; 100 ms duration) appeared at random either at the center or 8.3° to the left or right of fixation. The ISI was either 0.5, 1.0 or 3.0 s, intermixed randomly such that 50% of the stimuli were preceded by 0.5 s (of no stimulation), 33% by 1.0 s and 17% by 3.0 s. Stimulus position and ISI were randomized in 6 blocks of 200 trials each.

Electrical activity was recorded from the scalp using non-polarizable electrodes over the vertex (CZ) and homologous positions on the left and right hemispheres over occipital (O1, O2), parietal (P3, P4), temporal (33% of the interaural distance lateral to CZ), anterior temporal (1/2 the distance between F7(8) and T3(4), and frontal (F7, F8) regions (International 10-20 System). Potentials from these electrodes and the electrooculogram, from beneath the left eye, were referred to linked mastoids. Electrical activity was amplified with a bandpass of 0.01–100 Hz, and was recorded on FM tape for offline analysis. Trials contaminated by excessive eye movement or muscle artifact were rejected during averaging. For each subject and scalp location, average VEPs were computed for each of the 9 combinations of position in the visual field (left, center, right) and preceding ISI (0.5, 1.0, 3.0). The ERP data from the hearing subjects were analyzed by a 3-way analysis of variance with repeated measures on stimulus location, ISI and electrode. Comparisons between deaf and hearing subjects were analyzed by 4 way analysis adding group as a factor.

The VEPs from the hearing subjects were characterized by a negative component around 150 ms (N150; measured as the peak negativity between 100 and 200 ms relative to 100 ms of prestimulus baseline) and by a positive component around 230 ms (P230; maximum positivity between 180 and 300 ms). N150 was significantly larger to foveal than to peripheral stimuli ($P < 0.01$), and the distribution over the scalp of N150 and its refractory period were different for foveal and peripheral stimuli. Peripheral stimuli evoked N150 responses that were larger over the hemisphere contralateral to the visual field of presentation ($P < 0.001$) and N150 amplitude was of equal magnitude for all ISIs. By contrast N150 to the foveal stimuli was bilaterally symmetric and displayed larger amplitudes with larger ISI at the anterior electrode sites and at CZ ($P < 0.001$). Like N150, P230 was also of larger amplitude to foveal than peripheral stimuli for the hearing subjects ($P < 0.001$). However, unlike N150, P230 was bilaterally symmetric for both peripheral and foveal stimuli and displayed a more posterior distribution for foveal than peripheral stimuli ($P < 0.001$).

These data would not be consistent with the view that N150 and P230 arise from a unitary neural generator. Moreover, the data suggest that the generators of N150 and P230 are differentially sensitive to peripheral and foveal stimuli. These normative data are in sharp contrast to the results from the deaf subjects which suggest that auditory deprivation differentially affects N150 and P230 and the systems that subserve processing of peripheral and foveal stimuli.

A comparison of typical VEPs from the deaf and hearing Ss is provided in Fig. 1. While the morphologies of the waveforms for the deaf and hearing subjects were quite similar, the amplitudes of the N150 and the P230 components under certain conditions were strikingly different. Over the anterior scalp regions and at CZ, the N150 component was 1.5–3 times larger in the deaf than in the hearing subjects for stimuli presented to the periphery ($P< 0.001$) but not those presented to the fovea (see Table I). Whereas in hearing subjects N150 amplitude was larger to foveal than to peripheral stimuli; in the deaf subjects peripheral stimuli evoked N150 re-

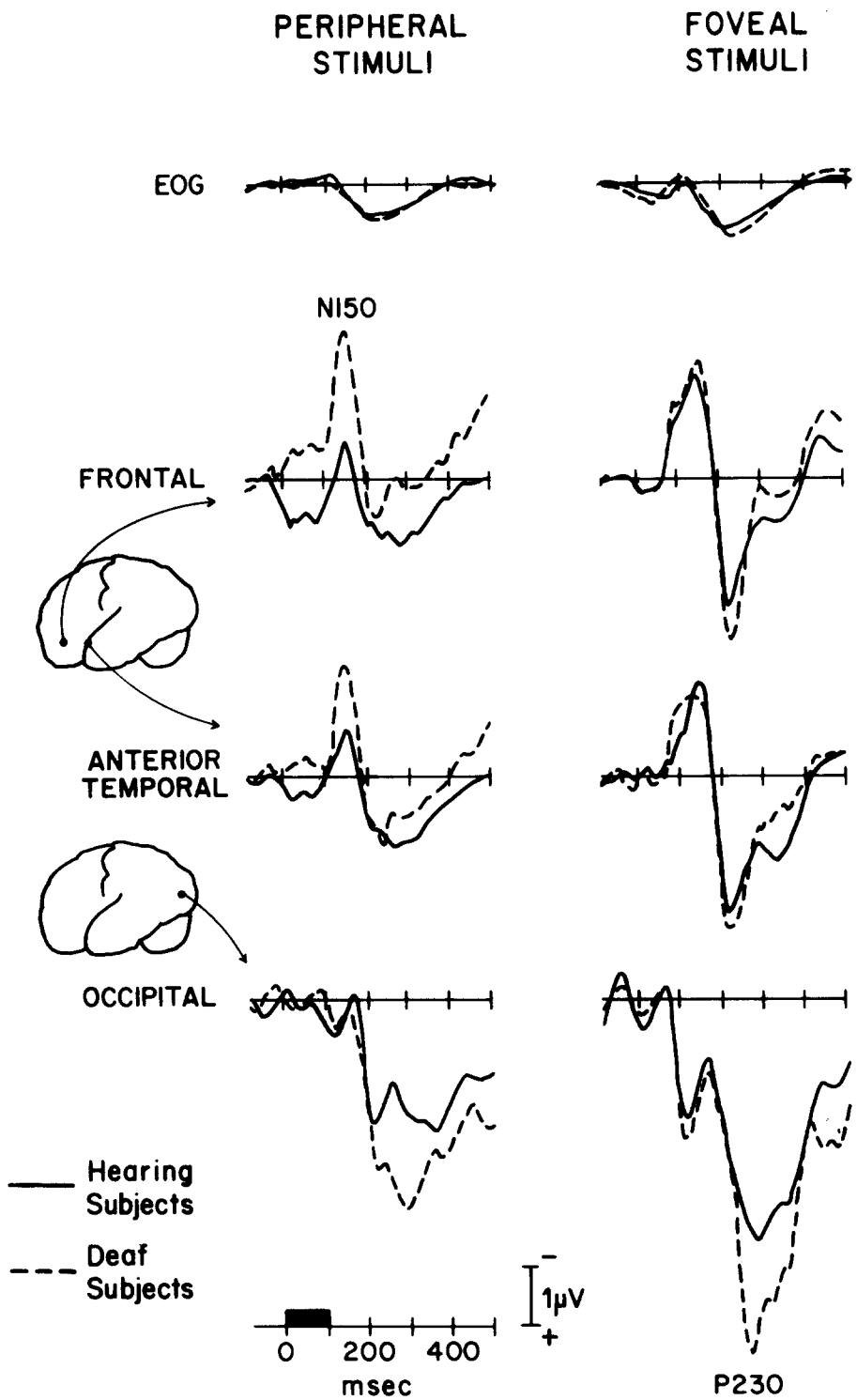


Fig. 1. Visual-evoked potentials (VEPs) from left frontal, anterior temporal and occipital electrodes to peripheral and foveal stimuli after the 3.0 s interstimulus interval. Also shown is the EOG recorded from under the left eye. VEPs and EOG averaged over 13 normal hearing (solid line) and 8 congenitally deaf (dashed line) adults are superimposed. The N150 component elicited by peripheral stimuli was larger from the contralateral than the ipsilateral hemisphere (mean amplitude all electrodes: hearing subjects contralateral −1.7 μV, ipsilateral −0.8 μV; deaf subjects contralateral −2.8 μV, ipsilateral −2.0 μV). However, as the effect of preceding ISI was independent of lateral visual field of stimulus presentation, the peripheral VEPs in Fig. 1 are collapsed across right and left visual field stimuli.

TABLE I

| *Electrode* | *N150 amplitude (μV)* | | *P* |
|---|---|---|---|
| | *Hearing Ss* | *Deaf Ss* | |
| *Peripheral stimuli* | | | |
| Frontal | −1.3 ± 0.4 | −2.9 ± 0.8 | <0.001 |
| Anterior temporal | −1.2 ± 0.4 | −2.6 ± 0.6 | <0.001 |
| Temporal | −1.3 ± 0.4 | −2.5 ± 0.5 | <0.001 |
| Vertex (CZ) | −2.0 ± 0.5 | −3.4 ± 0.6 | <0.001 |
| *Foveal stimuli* | | | |
| Frontal | −2.6 ± 0.5 | −2.5 ± 0.7 | ns |
| Anterior temporal | −2.3 ± 0.3 | −2.1 ± 0.5 | ns |
| Temporal | −2.3 ± 0.4 | −2.5 ± 0.5 | ns |
| Vertex (CZ) | −3.1 ± 0.4 | −3.4 ± 0.6 | ns |

sponses that were as large as those to foveal stimuli (see Fig. 2). Over the posterior scalp (i.e. at parietal and occipital electrodes) the P230 component was much larger in the deaf than in the hearing subjects for both peripheral and foveal stimuli ($P < 0.001$; see Table II). These effects were of equal amplitude bilaterally. As shown in Fig. 3, the major differences between the deaf and hearing subjects were observed in VEPs preceded by the longest interval (i.e. 3 s ISI).

Thus, the electrophysiological scalp responses elicited by simple visual stimuli were altered systematically at electrode sites overlying both auditory and visual areas in these persons who have been deaf from birth. These specific changes are consistent with both types of compensatory cortical reorganization mentioned above.

The relative enlargement of the amplitude of the visual N150 component over temporal and frontal regions in deaf subjects could be due to a reassignment of brain areas normally subserving audition and speech to the processing of visual information. This type of intermodal reorganization might reflect the dominant influence of

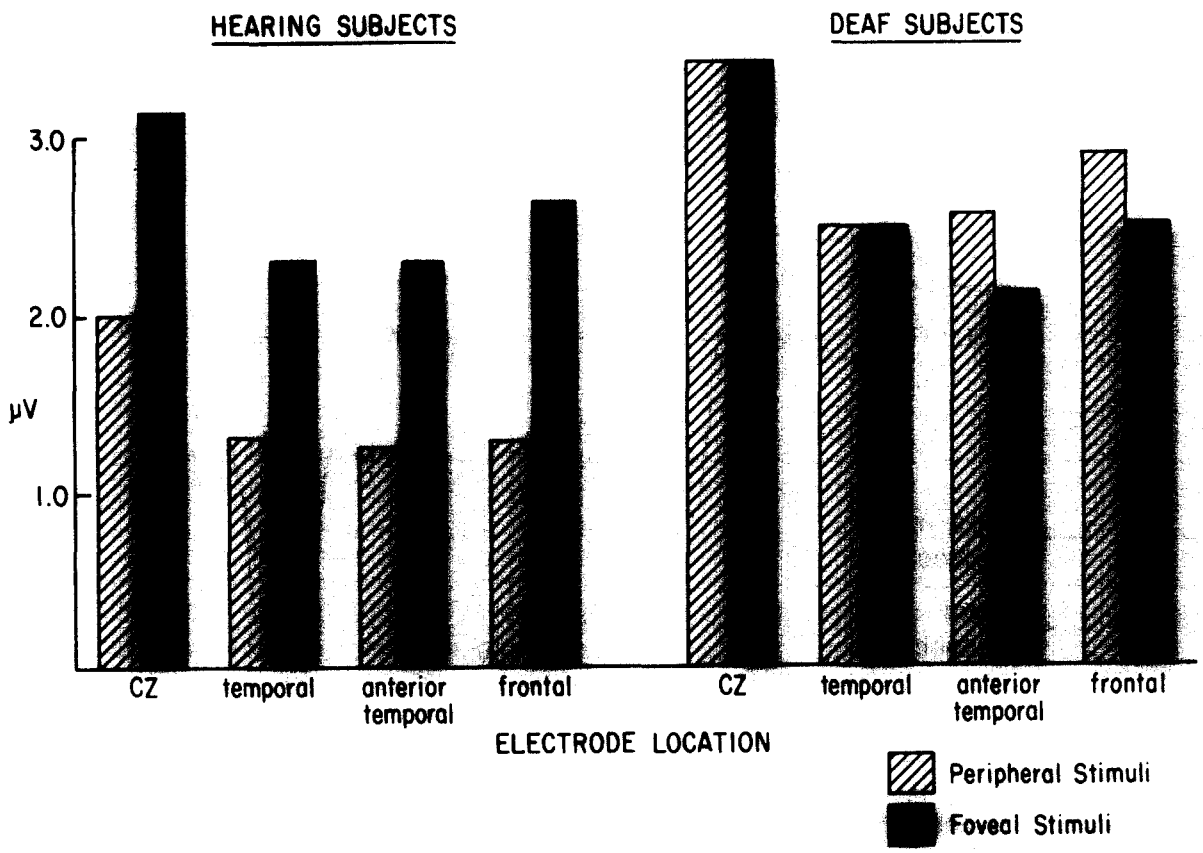


Fig. 2. Amplitude of N150 recorded from the vertex (CZ), temporal, anterior temporal and frontal regions to stimuli preceded by the 3.0 s ISI. Mean values from 13 normal hearing and 8 congenitally deaf adults for peripheral (dashed bar) and foveal (solid bar) stimuli.

TABLE II

| *Electrode* | *P230 amplitude (μV)* | | *P* |
|---|---|---|---|
| | *Hearing* | *Deaf* | |
| *Peripheral stimuli* | | | |
| Parietal | 3.5 ± 0.5 | 4.7 ± 0.8 | <0.001 |
| Occipital | 3.2 ± 0.5 | 4.9 ± 0.8 | <0.001 |
| Vertex (CZ) | 4.7 ± 0.6 | 6.6 ± 0.8 | <0.001 |
| *Foveal stimuli* | | | |
| Parietal | 4.7 ± 0.7 | 6.8 ± 0.7 | <0.001 |
| Occipital | 5.1 ± 0.9 | 7.4 ± 0.8 | <0.001 |
| Vertex (CZ) | 6.5 ± 1.0 | 9.6 ± 1.1 | <0.001 |

visual afferents on multimodal cells when auditory input is absent[10,11]. This account would be in line with the 'cortical competition hypothesis' proposed to account for changes in cortical neurons after monocular deprivation in cats[16] and with the proposal that neuronal activity specifies the selective elimination and stabilization of synapses[3]. On the other hand, cortical neurons committed to auditory processing may be taken over by visual afferents when auditory stimulation is absent. The presence of retinal projections to the medial geniculate body after early destruction of the brachium of the inferior colliculus in hamsters shows that this type of change can occur[13].

The specificity of the enhancement of N150 to peripheral and not foveal stimuli in the deaf subjects might result from a special compensation for peripheral sensory reception in deaf people, who rely primarily on vision for the detection of localization of events in the periphery. A second possibility is that foveal input reflected in these EPs is less modifiable by experience than is that from the periphery. In any case, such a specific pattern of differences is not likely to have arisen from group differences in overall level of arousal or from such differences as might accompany denervation supersensitivity[14], which would have resulted in non-selective amplitude increases in EP components to all stimuli.

The increase in the response amplitude of the P230 potential over parietal and occipital areas in deaf subjects might be related to structural changes like those observed in cortical areas associated with the remaining modalities in unimodally deprived experimental animals[8,12].

Since reliable amplitude differences in the VEPs appeared only following the 3.0 s interval, the results suggest that it is the slowly recovering neural responses which are less refractory in deaf than in hearing subjects. Such changes might be associated with the reported increased perceptual sensitivity in the remaining modalities after unimodal deprivation in man and other animals.

The significance of the particular anterior/posterior distribution of group differences in N150 and P230 remains to be clarified by studies

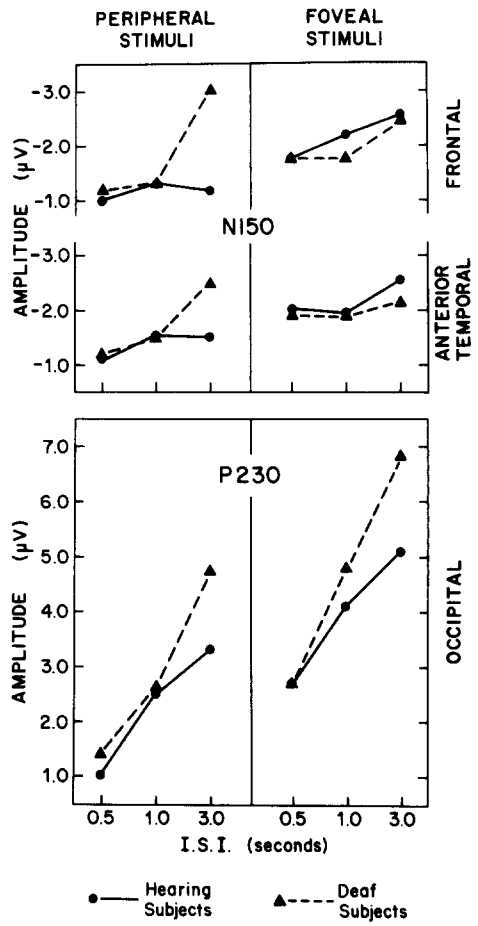


Fig. 3. Amplitude in microvolts (μV) of VEP component N150, recorded from left frontal and anterior temporal regions, and P230 from left occipital electrodes. Mean values from 13 normal hearing (solid line) and 8 congenitally deaf (dashed line) adults when stimuli were presented to the periphery (mean left and right visual fields) or to fovea after 0.5, 1.0 or 3.0 s interstimulus interval (ISI).

of the neural origins and functional relevance of these EP components. Nonetheless, it is of interest that these same components are enhanced during selective visual attention in normal adults[15]. Future research is required to determine the extent to which the present results reflect altered visual sensitivity and/or attentional capacity in deaf and hearing adults. In conclusion, our data suggest that early auditory deprivation alters the intermodal organization of the brain's sensory systems in man.

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