Reorganisations of the visual and auditory Cortex for auditory Processing in congenitally blind individuals

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ABSTRACT
Background and objective: We assessed whether individuals with total blindness are compensated for the loss of their visual neuronal circuitry by augmentation in the auditory and tactile perception by comparing the P300 components of the auditory evoked potentials in the individuals with total blindness and normal vision.
Methods: Twenty individuals each with total blindness or normal vision were recruited for this study. P300 components of the auditory evoked potentials were recorded. Latency and amplitude of the waveforms were measured and analyzed.
Results: The latencies of all the waveforms were significantly reduced in the subjects with total blindness when compared to the subjects with normal vision. In contrast, the amplitude of the waveform P300 at Oz site was significantly increased in the subjects with total blindness.
Conclusion: Our study suggests that individuals with total blindness demonstrate remarkable neuroplasticity with neurophysiological evidence of much better information processing in the auditory system with the visual cortex additionally participating in hearing process.
Keywords: Total blindness, auditory evoked potentials, P300, neuroplastic changes, Plasticity.

Introduction
Individuals with total blindness are often compensated for their visual handicap by developing supranormal abilities in their other sensory systems such as the auditory and the somatosensory systems. Previous studies have reported plasticity in the neural tracts and the higher central nervous system in individuals with total blindness as they depend on the non-visual sensory modalities.[1, 2, 3, 4, 5] Neural plasticity is an important and vital adaptation, which is the capacity of the nervous system to modify its organization by the changing neuron types, their networks and their function consequence to new experiences.[6] These changes are beneficial and the brain continually responds to changes in stimuli by reorganizing itself, which is now believed to be much more dynamic. While cross modal plasticity refers to the capacity of the brain to replace the functions of a lost part by another part. Indeed cross modal plasticity has been documented both in animals and humans deprived of a particular sensory modality such as vision or audition. The most commonly used form of sensory substitution (cross modal plasticity) is Braille reading, which enables the blind individual to read by using the somatosensory tactile system.[7, 8, 9]
Auditory evoked potentials(AEPs) are a subclass of Event related potentials (ERPs) wherein the
sound (event) is the sensory input. AEPs and ERPs are very small electrical voltage potentials originating from the brain recorded from the scalp in response to an auditory stimulus. AEPs are classified into early (the first 10 to 15 milliseconds), middle (10 to 80 ms) and late (80 to 750 ms) components. Several studies have reported augmentation of the AEP among individuals with total blindness in comparison to individuals with normal vision.\[2, 3, 4, 5\] Interestingly the components of the middle latency auditory evoked potentials (MLAEP) have shorter latencies individuals with total blindness in comparison to individuals with normal vision.\[10\] However the data on the P300 response of the long latency component of the auditory evoked potential (LLAEP) in individuals with total blindness is not reported. Hence, the present study was designed to advance our understanding of P300 responses among individuals with total blindness vis a vis the plasticity of the auditory system.

**Materials and Methods**

A comparative study involving 20 female subjects with total blindness (recruited from blind schools) and 20 age matched female subjects with normal vision (from the general population) was designed. Subjects were explained about the study protocol and informed written consent was obtained from all the subjects. The study was approved by the institute’s ethical committee for research involving humans.

Female subjects aged 18-40 years, in early phase (first week) of menstrual cycle, who were congenitally blind or with total blindness (category 5) for more than 2 years were included in the study. Subjects with other visual defects and other causes of blindness, history of hearing impairment, neurological disorders, subjects using drugs (narcotics, stimulants and neurotropic drugs), which would affect the outcome of the study, were excluded from the study.

Subjects were assigned to two different groups - Group (A) Subjects with total blindness and Group (B) Subjects with normal vision.

Personal details such menstrual history and last date of previous menstrual cycle was procured through history from all subjects. Subjects were screened for general physical health to rule out any clinical disorder likely to interfere with the study findings. Anthropometrical details such as weight in kilograms, height and head circumference in centimeters were recorded from all the subjects. An ophthalmologist certified the group A subjects for blindness through ophthalmologic and fundoscopy examinations. All the subjects were screened for hearing threshold by audiometry.

AEPs were recorded with the subjects awake, comfortably lying down in the bed in a semi-darkened room and were requested to remain calm keeping their eyes closed to avoid electrooculographic artifacts due to eye movements and improve the concentration and attention to the stimuli presented. Electrode placing, nomenclature and methodology of AEP recordings were according to study published previously.\[11\] Briefly, AEP recordings were performed in an air conditioned, sound-proof room by using Ag/AgCl disc electrodes affixed with Ten 20 conductive paste after cleaning the sites with Nuprep EEG and ECG abrasive skin prepping gel at Fz, vertex (Cz) ,Pz and Oz for the recording of P300 recording, left and right ear lobules (A1, A2). All electrodes were plugged to a junction box and skin to electrode impedance was kept below 5 KOhm.

P300 was measured from the vertex (Fz, Cz, Pz and Oz) in response to random stimuli presented mono-aurally through headphones applied to the subject's ear. The ground electrode was placed at FPz. Standard auditory oddball paradigm was used. Briefly, the subject was presented with 300 stimuli as a sequence of two distinguishable sound stimuli, one of which occurred frequently (frequent stimulus /non-target) for 240 times and the other infrequently (rare stimulus/target) for 60 times. The frequency of the frequent stimulus was 1000Hz and that of the rare stimulus was 2000Hz. The subjects were instructed to press the button as soon as a target or infrequent stimulus was
presented. The stimulus sequence was random and presented at 80 dB SPL. The settings were properly selected and evoked responses to the rare stimuli were filtered with a bandpass 1-30 Hz and averaged. Samples contaminated with artifacts were auto discarded. The latency of N100, P200, N200, P300 and amplitude of waves P200 and P300 for target stimuli (rare) were calculated. The responses to the frequent and rare stimuli were averaged. The waveform pattern was replicated and the different waveform latencies and amplitudes were calculated. Amplitude (µV) was measured from the peak of one polarity to the immediately following peak of the opposite polarity. Contralateral ear was masked with a white noise of –30 dB. Signals picked up by electrodes were filtered (10 Hz and 200 Hz), amplified, averaged and displayed on the screen of GALILEO NT Evoked Potential Recorder.

Statistical analysis
The data were analyzed using descriptive statistics. Results on continuous measurements are presented on Mean ± SD and results on categorical measurements are presented in Number (%). Significance was assessed at 0.5 % level of significance. Student t test (two tailed, independent) was used to find the significance of study parameters on continuous scale between two groups (Inter group analysis). Effect size was also computed. Statistical software (SPSS version 15.0) was used for the analysis of the data and Microsoft Word and Excel was used to generate graphs and tables.

Results
Subjects were matched for the basic characteristics (Table 1). The mean pattern of amplitude of P300 wave ( Fz, Cz, Pz, Oz) was significantly (p > 0.05) different between the two groups only at Oz while the amplitude of P300 waveform was significantly increased in the blind subjects as compared to subjects with normal vision. (Table 2, Graph 1, Graph 2)

Table 1: Age, height and weight and head circumference of subjects studied

<table>
<thead>
<tr>
<th>Basic characteristics</th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>24.33±6.29</td>
<td>26.05±6.59</td>
<td>0.409</td>
</tr>
<tr>
<td>Height in cm</td>
<td>157.80±4.58</td>
<td>156.65±5.79</td>
<td>0.490</td>
</tr>
<tr>
<td>Weight kg</td>
<td>54.55±8.14</td>
<td>56.60±7.94</td>
<td>0.425</td>
</tr>
<tr>
<td>Head circumference cm</td>
<td>30.75±1.11</td>
<td>30.60±1.19</td>
<td>0.683</td>
</tr>
</tbody>
</table>

Table 2: Comparison of P300 (amplitude and latency) between two groups

<table>
<thead>
<tr>
<th>P300</th>
<th>Group A</th>
<th>Group B</th>
<th>P value</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMPLITUDE (microvolts)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fz</td>
<td>6.83±2.09</td>
<td>6.24±3.96</td>
<td>t=0.584;p=0.562</td>
<td>0.18</td>
</tr>
<tr>
<td>Cz</td>
<td>7.82±2.30</td>
<td>7.83±3.51</td>
<td>t=0.008;p=0.994</td>
<td>0.00</td>
</tr>
<tr>
<td>Pz</td>
<td>8.65±2.68</td>
<td>9.60±5.27</td>
<td>t=0.715;p=0.479</td>
<td>0.22</td>
</tr>
<tr>
<td>Oz</td>
<td>8.76±2.55</td>
<td>12.51±6.08</td>
<td>t=2.544;p=0.015*</td>
<td>0.79</td>
</tr>
<tr>
<td>LATENCY(ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fz</td>
<td>334.7±11.53</td>
<td>311.97±5.08</td>
<td>t=8.069;p&lt;0.001**</td>
<td>2.50</td>
</tr>
<tr>
<td>Cz</td>
<td>336.26±13.09</td>
<td>313.37±5.42</td>
<td>t=7.227;p&lt;0.001**</td>
<td>2.24</td>
</tr>
<tr>
<td>Pz</td>
<td>338.67±12.94</td>
<td>311.46±5.27</td>
<td>t=8.712;p&lt;0.001**</td>
<td>2.70</td>
</tr>
<tr>
<td>Oz</td>
<td>340.82±11.9</td>
<td>312.61±4.51</td>
<td>t=9.912;p&lt;0.0001**</td>
<td>3.07</td>
</tr>
</tbody>
</table>

* Significance at 5% ** Significance at 1%
Graph 1 Comparison of P300 amplitude between two groups

Graph 2 Comparison of P300 latency between two groups
P300 RECORDING OF TOTALLY BLIND SUBJECT
Discussion

Individuals with total blindness have to rely on non-visual information to a greater extent to efficiently interact with the external environment and consequently exhibit optimal skills in their routine. Our findings are in agreement with the hypothesis that there is neurophysiological evidence of better information processing in the auditory system with an additional input from the visual cortex in hearing in the individuals with total blindness when compared to the individuals with normal vision.

Base to peak (baseline to peak) and peak to peak (peak of one polarity to the immediately following peak of opposite polarity) of all AEPs- P300 for the amplitude of the waves were measured. Although both these two methods are commonly used, the difficulty in defining the baseline sometimes makes that measurement more subjective than the peak to peak method. The mean pattern of absolute latency of P300 wave (Fz, Cz, Pz, and Oz) was significantly reduced among the blind subjects when compared to the normal subjects. This probably indicates that the rate of automatic processing and information categorization is faster among the blind subjects because of sensory compensation. Nevertheless neural plasticity can also increase the rate of auditory processing and attention in early blind subjects.

The latency of the late cortical potentials(P300) provides information on the whole sensorium and its timing, which is consistent with previous reports. The mean pattern of amplitude of P300 wave (Fz, Cz, Pz, Oz) was significantly different between the two groups at Oz where the amplitude was significantly higher among the blind subjects compared to subjects with normal vision. This posterior shift in the distribution of the evoked potentials indicates that there may be recruitment of posterior temporal, parietal and occipital cortices in auditory processing suggestive of cross modal plasticity. Our findings are again consistent with previous study reporting that the targets
elicited larger and more posteriorly distributed N2 responses in the blind subjects compared to subjects with normal vision.[1] It is interesting to note that the brain reorganizes itself in response to blindness, possibly as a result of the blind individuals’ greater attention to and reliance on non visual sensory avenues to maintain interaction with the surround environment. Hence our findings are probably linked to increased attention leading to quicker processing during the discrimination tasks of the event related potentials. Focused attention on behaviorally relevant stimulation over extended periods are found to produce a substantial enlargement in the representational zones of the involved portions of the body in somatosensory cortex in experimental animals,[16, 17] and humans. [18,19] Similarly, the primary auditory cortical fields can be dramatically refined or profoundly degraded. Interestingly use-dependent reorganization in the frequency receptive fields is previously reported.[20,21,22]

Several studies have looked at cross-modal plasticity in blind humans, wherein auditory [8, 9, 23,24] stimuli are additionally processed in the visual cortex. Indeed plastic reorganization could result from an increase in the effectiveness of pre-existing pathways, suggesting that the representational plasticity is a consequence of the heavy differential sensory input. The elaboration of a use-dependent cortical reorganization involving either an unmasking of previously silent connections and/or sprouting of new neural elements from those that previously existed may be responsible for expansion of auditory cortex in blind subjects in our study. However reduced cell death in the cortical territory of other modalities, including auditory cortex, or to stabilization of transient connections, expanded auditory area might include a larger number of contributing neurons and hence larger dipole moments in addition to higher frequency tuning of neurons activating a smaller set of other neurons; an expansion of the auditory cortex[25] may also be possible factors. Nevertheless the elaboration of the use-dependent cortical reorganization involving either an unmasking of previously silent connections and or sprouting of new neural elements from those that previously existed may also be possible. Indeed several mechanisms, which are not mutually exclusive, are possibly involved, which necessitates further research.

The expansion of the tonotopic map, the reorganization of the auditory cortex in the blind [19,23,26] would appear to be an excellent composite example of the principle formulated by Merzenich et al of the continual competition for cortical space.[28] Indeed functional MRI study supports the notion of altered capabilities for surviving modalities through reorganization of cortical functions.[29] The changes constitute de novo cross-modal plasticity in response to severe unimodal sensory deprivation.

The potential processes responsible for novel effects involve anatomical and physiological changes that are studied extensively using animal models.[30] Alternatively, observed access of surviving modalities to deprived cortex is an expression, although exaggerated, of normal physiology that usually is inhibited or hidden when vision is present. This potential mechanism relies on possible changes in the balance of activity within existing cortical and subcortical networks. However as highlighted above these mechanisms are not mutually exclusive considering the different ages for blindness onset and, therefore, differences in developmental sensitive periods in establishing connections.

Nevertheless our study provides evidence of neuroplasticity in the visually deprived subjects but necessitates future studies to further tease the cellular and molecular mechanisms important in neuroplasticity, which will be vital in designing improved educational and rehabilitative programs for the blind.

**Conclusion**

We conclude that individuals with total blindness have significantly reduced latencies suggestive of much better information processing in the auditory system in addition to larger P300
amplitude more posteriorly, which is suggestive of the visual cortex being activated for auditory stimulation.

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References