MIDDLE LATENCY AUDITORY EVOKED POTENTIALS IN CONGENITALLY BLIND AND NORMAL SIGHTED SUBJECTS

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Middle latency auditory evoked potentials were recorded in two groups of ten subjects each, viz, congenitally blind (CB) and age-matched subjects with normal vision (NV). The age range for both groups was 13 to 16 years. The CB group subjects had peripheral deficits, with absence of visual evoked responses. The peak latency of the Nb wave (the maximum negativity between 38 and 48 ms) was significantly lower in the CB group compared to NV group ($p < .05$, one-tailed, two factor ANOVA, Tukey test). In addition to these recordings from the vertex, recordings were also made from occipital areas, to test whether the visual cortex contributes to information processing at primary auditory cortical levels in the blind, as was reported in earlier studies on the generation of potentials during auditory selective attention. No such effect was observed. Hence, it appears that in blind subjects changes in generators of auditory middle latency evoked potentials are mainly related to latency, rather than to scalp distribution of these components.

Keywords: Middle latency auditory evoked potentials; congenitally blind; normal-sighted; primary cortical areas

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Blindness at an early stage of development has been found to modify nondeprived sensory modalities, as a compensatory measure. A study of the long-latency auditory event related potentials (ERPs) in early blind humans showed that the negative deflection 100 ms from stimulus onset (N1) and subsequent deflections (P2 and P3) show shortened latencies and enhanced amplitudes (Niemeyer & Starlunger, 1981; Woods, Clayworth & Bach-y-Rita, 1985). In a subsequent study auditory ERPs were recorded in early blind subjects and sighted controls when they attended to stimuli delivered to a designated ear under dichotic conditions (Alho, Kujala, Paavilainen, Summala & Näätänen, 1993). The scalp distribution of the processing negativity, i.e., the endogenous negativity elicited by attended stimuli, as well as the mismatch negativity elicited by occasional higher frequency tones, was recorded more posteriorly in the blind compared to the normal sighted. These results suggest that posterior brain areas normally involved in vision participate in auditory selective attention in the blind.

The present study was designed to compare: (1) the peak latencies and amplitudes of middle latency auditory evoked potential (AEP-MLR) components in blind and age-matched normal sighted subjects, also (2) the peak amplitudes of AEP-MLR components recorded from the conventional site (i.e., vertex) with recordings made from posterior brain areas (i.e., occiput). This comparison was made in both blind and normal-sighted subjects.

METHOD

Subjects

There were two groups of ten subjects each. One group consisted of blind subjects (group average age ± SD, 14.3 ± 1.4 years), with a single female subject. All subjects had a diagnosed peripheral deficit with an inability to differentiate between light and dark, from birth. This was confirmed by an absence of visual ERPs to light flashes. Apart from visual impairment these subjects had no other physical or mental deficit. The other group consisted of subjects with normal vision without correction, confirmed by normal ERPs elicited by light flashes, who were selected to match the blind subjects for age (± 1 year). The group average age ± SD of this group was 14.1 ± 1.1 years. For both groups the informed consent of the subjects and their guardians was taken in accordance with the ethical guidelines of the Indian Council of Medical Research (New Delhi, India).

Design of the Study

All subjects were assessed in a single sitting. Recordings were made in the following sequence: two consecutive recordings (R1, R2) of AEP-MLR to assess the immediate reproducibility of the waveforms, followed by a recording of VEP.
Recording of Evoked Potentials

(1) Auditory middle latency evoked potentials (AEP—MLRs) were recorded in the 100 ms poststimulus time period, from Cz, referenced to the right earlobe, with the ground electrode on the forehead. The preamplifier band width was set at 10 to 1500 Hz. 1500 responses were averaged for each period. Sweeps containing artifactually large signals were rejected through software specification. The rejection level was expressed as a percentage of the full scale range of the analog-to-digital convertor. This level was set at 85%. The number of sweeps was displayed on the monitor. Click stimuli of 40μs duration and alternating polarity were delivered at 5 Hz binaurally, through acoustically shielded earphones (Elga DR—531, Japan). The intensity was kept at 80 dB for all assessments. The threshold of hearing was also noted in all subjects.

Visual evoked potentials were recorded in the 200 ms poststimulus time period, from Oz, referenced to the right earlobe, with the ground electrode on the forehead. The preamplifier band width was set at 1 to 100 Hz and 100 responses were averaged for each period. Artifact rejection was similar to the method described above for recording auditory evoked potentials. Light flashes were given using a LED visual stimulator (Nihon Kohden SLS 3500). Stimuli were given to each eye separately and also binocularly.

AEP-MLRs Components

Peak amplitudes of short latency wave V, and middle latency Na, Pa and Nb waves were measured from the baseline existing at the beginning of the sweep. Peak latency was measured from the time of click delivery.

The middle latency auditory evoked response components (AEP—MLRs components) were described as follows: wave V was the maximum positive peak between 5 and 8 ms, the Na wave was the maximum negative peak between 14 and 18 ms, the maximum positive peak between the Na wave and 35 ms was described as the Pa wave, and the maximum negative peak between 38 and 48 ms was described as the Nb wave. These descriptions are similar to those in other studies on AEP—MLRs (Erwin & Buchwald, 1986; McPherson, Tures & Starr, 1989).

Data Analysis

[1] Comparison of peak latencies between congenitally blind subjects and those with normal vision. The data were analyzed using a two factor analysis of variance (ANOVA), with factor A = groups, i.e., congenitally blind versus normal vision, and factor B = repeat recordings (R1, R2). The Tukey multiple comparison test was used to detect significant differences between group mean values.

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[2] Comparison of peak amplitudes of potentials in congenitally blind subjects and those with normal vision using a two factor analysis of variance (ANOVA), with factor A = groups, i.e., congenitally blind versus normal vision, and factor B = repeat recordings (R1, R2). Separate ANOVAs were performed for peak amplitudes of potentials recorded from the two sites, viz Cz and Oz. This analysis was restricted to the Pa and Nb waves, which are known to have cortical generators. The Tukey multiple comparison test was used to detect significant differences between group mean values.

RESULTS

[1] Comparison of peak latencies between congenitally blind subjects and those with normal vision showed a significant difference (two factor ANOVA) between the Nb wave peak latency (38 ms–48 ms range) of the two groups, viz congenitally blind and normal sighted [F for Factor A = 5.35, since F .05 (1) 1, 36 = 4.11, hence p < .05]. However, there was no significant difference between the repeat assessments, i.e., R1 and R2, Factor B (p > .50), and the interaction between factors (A × B) was also not significant (p > .10). The F value for df = 1, 36 has been derived by linear interpolation from the df = 1, 30 and df = 1, 40 from the standard table as has been described (Zar, 1984).

Also, the Tukey test for multiple comparisons between mean values showed a marginal significant difference between the mean values (second assessments) of the congenitally blind and normal sighted subjects [q = 3.65, since q at probability level .07 for df = 36, 4 = 3.62, hence p < .07]. The significantly lower Nb wave peak latency in the congenitally blind group was observed as: (1) a difference of more than 3.0 ms in 4 subjects, (2) more than 1.0 ms in 2 subjects, (3) in 3 subjects this difference was less marked (0.56 to 0.83 ms) and (4) in a single CB subject the Nb wave peak latency was higher (by 1.0 ms). Two examples of (1) and a single example of (3) are illustrated in Figure 1. The two factor ANOVA did not show significant differences between either factors or interactions for the peak latencies of the other AEP—MLRs components (p < .20, in all cases).

[2] Comparison of the peak amplitudes of the Pa and Nb waves recorded from the occipital area (Oz) showed that the peak amplitude of the Pa wave at Oz was significantly lower in the congenitally blind compared to the normal vision group [F for Factor A = 5.07, since F .05 (1) 1, 36 = 4.11, hence p < .05]. There was no significant difference between repeat assessments or interaction between factors (Factors B, A × B, p > .20) for Pa wave. For the Nb wave there were also no significant differences (Factor A, Factor B, A × B, p > .50).

When recordings were made at Cz, there were no significant differences for all 4 auditory evoked potential components studied (p > .20, for all comparisons).
FIGURE 1 Three examples of AEP-MLRs recorded in three pairs of congenitally blind (CB) and normal vision (NV) subjects. The first two pairs (I, II) show examples of noticeably lower Nb wave peak latency in CB group subjects compared to NV group subjects. The third pair (III) shows a less obvious difference in Nb wave peak latency between the two groups.
The group average values of peak amplitudes and latencies of the four components studied, for the congenitally blind and normal sighted subjects are given in Table I.

**DISCUSSION**

The present results show that the Nb wave peak latency was significantly lower in congenitally blind (CB) subjects compared to age matched subjects with normal vision (NV). The peak amplitude of the Pa wave was significantly lower for the CB group compared to the NV group when the recording was made from Oz. There was no obvious difference in the threshold of hearing of CB and NV groups.

The lower peak latency of the Nb wave in the CB group is in keeping with previous reports of lower latency of the N1 wave in CB subjects (Niemeyer & Starlinger, 1981; Woods et al., 1985). However these reports also described an enhanced amplitude in the blind. In the present study there was no significant difference in peak amplitudes of the two groups when recordings were made from the vertex (Cz). Another study (Alho et al., 1993), had described a larger processing negativity (PN) to attended tones at occipital scalp sites in CB subjects compared to subjects with normal vision. These results suggested that in blind subjects in addition to the participation of conventional neural areas (auditory cortex, frontal cortex) the parietal and even occipital cortices may contribute to the PN.

In the present results no such effects were observed. In fact the peak amplitude of the Pa wave recorded from the occipital area of the congenitally blind subjects was significantly less than that of the normal sighted subjects, recorded from the same site. The Pa wave is understood to be related to simultaneous activation of both supratemporal auditory cortices (Deiber, Ibáñez, Fischer, Perrin & Mauguière, 1988). Intracerebral recording in man has shown that the generator of the Nb wave

| TABLE I | Peak latencies and peak amplitudes of AEP-MLRs recorded in congenitally blind (CB) and normal vision (NV) subjects, (N = 10) |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                | Wave V          | Na wave         | Pa wave         | Nb wave         |
|                | CB   NV         | CB   NV         | CB   NV         | CB   NV         |
| Peak Latency   | m     ms         | m     ms         | m     ms         | m     ms         |
| (ms)           | 5.4 ±1.0        | 14.6 ±1.8       | 28.3 ±5.6       | 44.3* ±3.6      |
|                | 5.4 ±1.0        | 14.5 ±1.8       | 28.9 ±6.2       | 47.7 ±4.2       |
| Peak Amplitude | (μV) at Cz      |                |                |                |
| (μV) at Cz     | 1.2 ±0.5        | 0.7 ±0.8        | 1.1 ±0.9        | 0.7* ±0.8       |
|                | 1.2 ±0.6        | 1.9 ±1.3        | 1.2 ±1.0        | 1.0 ±0.5        |
|                | 1.3 ±0.8        | 1.1 ±0.5        | 1.3 ±0.5        | 0.5 ±0.5        |
| Peak Amplitude | (μV) at Oz      |                |                |                |
| (μV) at Oz     | — —            | — —            | — —            | — —            |
|                |                 |                 |                 | 0.5* ±0.3       |
|                |                 |                 |                 | ±0.5 ±0.5       |

*p <.05, two factor ANOVA, one-tailed, CB versus NV
is localized with relatively focal topology, in the dorsoposteromedial part of Heschl’s gyrus, or primary auditory cortex (Liègeois-Chauvel, Musolino, Badier, Marquis & Chauvel, 1994). In the present study the lower Nb wave peak latency in the blind implies that facilitation of processing of auditory information occurs at the level of the primary auditory cortex. The absence of change in the wave V and Na wave may mean that brainstem and diencephalic areas, which are known generators of these waves (Deiber et al., 1988), appear unchanged.

The results of the present study also suggest that the occipital areas do not participate in the information processing at primary cortical areas. This result is in contrast to the previous study (Alho et al., 1993) which demonstrated participation of more posterior areas in generation of potentials during auditory selective attention.

The lower peak amplitude of the Pa wave recorded from Oz in the blind compared to the normal sighted is contrary to the above cited study (Alho et al., 1993). None of the blind subjects had central (cortical) blindness, making it unlikely that underlying neuropathology could explain the lower amplitude recorded at Oz in the CB group. Hence, it appears as though the occipital area does not play a role in auditory information processing at primary cortical areas, in the congenitally blind. Compensatory changes at this level occur as shortened latency of the corresponding components.

References