Central Auditory Maturation and Language Development in Children with Hearing Loss: A Preliminary Study

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Abstract

The present study was aimed to know and compare the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children. Twelve children with normal hearing sensitivity (control group) and 13 children with bilateral severe to profound hearing loss (clinical group) were taken. Participants were divided into 4 sub groups based on their language age and participants in both the groups had language age ranging from 1 to 5 years. Subjects in both the control and clinical group were tested on a test battery including auditory long latency response (ALLR). P1 latency was considered for further analysis. The mean P1 latency decreased as the language age increased from 1 to 5 years in both the control and clinical group, but this was not statistically significant. The ALLR waveform morphology improved and the presence of P1, N1, P2 and N2 components was increased as the language age increased. Within a given language age group, children with a higher language age had earlier P1 latency than the children with a lower language age. P1 latency were found to be longer in clinical group compared to control group in all the language age groups, but this again was not statistically significant.

Keywords: *Auditory long latency response (ALLR), language age.*

Introduction

Central auditory maturation is a constant process from pre-natal period to puberty. Development of the peripheral auditory system (ear & auditory brainstem) is complete in early childhood (Eggermont, 1989). In contrast, central auditory pathways of the human brain exhibit progressive anatomical and physiologic changes through early adulthood (Kraus, Smith, Reed, Stein & Cartee, 1985; Courchesne, 1990; Huttenlocher, 1979). This maturation is likely to have an impact on speech and oral language skills, speech production and perception which are primarily acquired through the auditory modality.

Language development occurs from phonetic perception to building up of the vocabulary. Children first develop differential cry, then babbling and then to the one word utterances and progressively to sentences by one and a half years of age. By the age of 5 to 7 years of age children sound like as if they have mastered the phonology of their language. Children become more fluent in producing complex sentences of sounds and multisyllabic words (Vihman, 1988b).

The correlation between central auditory maturation and language development has been studied behaviorally (Tallal, Stark & Mellits, 1985). Tallal, Stark, and Mellits (1985) reported that the variables assessing temporal perceptual and production abilities, which taken in combination correctly classified 98% of participating subjects as language-impaired or normal. However, for children as young as 5 years and below 5 years these correlations between central auditory maturation and language development can be studied well accurately using the auditory evoked potentials (AEPs). Also the neurophysiologic responses to the consonant-vowel syllables indicate the representation of such sounds is undoubtedly important for speech and language development. Auditory evoked potentials (AEPs) reflect maturation of the human brain through changes in their latency, amplitude and morphology (Eggermont, 1989; Courchesne, 1990). The cortical auditory evoked potential (CAEP) reflects the cerebral maturation through the change in latency and the shape of the waveform. P1 shows robust positivity in the CAEP and the P1 latency ranges from 50 ms to around 300 ms. It is known that the latency of the P1 continuously changes as age increases and mainly from infancy to adolescence with a range of 50-150 ms latency (Kraus et al., 1985; Sharma, Kraus, McGee & Nicol, 1997). There have been several studies that focused on the developmental status in the auditory pathway using this characteristic of P1 (Ponton et al., 1996; Ponton, Don & Masuda, 1996). Because the P1 latency reflects the developmental status of the central auditory pathway (Ceponiene, Cheour & Naatanen, 1998; Cunningham, Nicol, Zecker & Kraus, 2000; Ponton, Eggermont, Kwong & Don, 2000), it has been used to evaluate the change of maturation in the auditory pathway for congenitally deafened children after they have been

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fitted with hearing devices such as hearing aid or a cochlear implant (Sharma, Dorman, Spahr & Todd, 2002; Sharma, Dorman & Spahr, 2002).

Children with congenital hearing impairment, when provided with early intervention have beneficial effects on early language (Vohr et al., 2008). Children who were identified as having hearing loss by 6 months of age demonstrated significantly better language scores than children identified after 6 months of age **(**Yoshinaga-Itano, Sedey**,** Coulter & Mehl, 1998). Also, it is reported that as the age of identification and intervention increases 'language gap' (the gap between chronological and language age) increases. But this language gap seems to decrease over time with stimulation **(**Rhoades & Chisolm, 2001).

Studies on the effects of sensory deprivation on central auditory pathways in humans indicate that, children who were deprived of sound for greater than 7 years, show delayed P1 latencies. Whereas, children who experienced fewer years of deprivation, between 3.5 and 7 years, had normal P1 latencies and children who experienced fewer than 3.5 years of deprivation showed normal P1 latencies. Thus, if stimulation is delivered within that period, then P1 latency and morphology reach age-normal values within 3 to 6 months following the onset of stimulation. However, if stimulation is withheld for more than 7 years, then most children exhibit a delayed P1 latency and abnormal P1 morphology, even after years of implant use (Dorman, Sharma, Martin, Roland & Gilley, 2007).

Series of investigations by Sharma and Dorman has demonstrated that, cortical maturation reaches normal in 3 to 6 months of stimulation, if children with hearing loss identified and rehabilitated below 3.5 years of age. Delayed maturation is noticed if age of identification is more than 7 years of age and also that the P1 latency can be used as a biomarker to assess the status of central auditory maturation. However, it is not clear, whether there is a relation between language age and the development of P1 latency. There is a dearth of information on comparison of P1 latency and language age in a group of children with hearing impairment fitted with hearing aid and normal hearing children.

Hence, aim of the present study is to know the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children. Present study also aims to compare the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children.

Method

Participants

A total of 25 participants (12 males and 13 females) were considered for the study. These participants were divided into two groups, control group and clinical group. Both the groups were matched in terms of their language age. Control group comprised of 12 children (6 males & 6 females) with normal hearing sensitivity in the age range of 1 to 5 years with a mean age of 3.08 years. Clinical group comprised of 13 children (6 males & 7 females) with bilateral severe to profound sensori-neural hearing loss in the age range of 2.5 to 6 years with a mean age of 4.93 years. The participants in both control and clinical group were further divided into 4 sub groups based on their language age, that is, 1 - 2 years, 2 - 3 years, 3 - 4 years and 4 - 5 years. Three children were considered in each sub-group in the control group. Four children in the first sub-group and three children were considered in each of the next three sub-groups in the clinical group. None of the participants had any history and complaint of middle ear pathology and any complaint and history of observable medical or neurological impairment. The Chronological and language age of the participants for each sub group in both the control and clinical group are given in Table 1 and 2 respectively.

The subjects in the control group had normal hearing sensitivity in both the ears, with air conduction (AC) and bone conduction (BC) thresholds within 15 dB HL at all the octave frequencies from 250 Hz to 8000 Hz and 250 Hz to 4 KHz respectively. Their speech detection threshold or speech recognition threshold and speech identification scores were correlating with the pure tone thresholds. All the subjects had 'A' type tympanogram with normal ipsilateral and contralateral acoustic reflexes present in both the ears. They had Transient Evoked Oto-acoustic Emissions (TEOAEs) with a SNR of +6 dB and the response reproducibility and stimulus stability of greater than 80%. They had normal Auditory Brainstem Response (ABR) with a wave V at 30 dBnHL. They had age appropriate speech and language development with language age ranging from 1 to 5 years.

The subjects in the clinical group had bilateral severe to profound sensori-neural hearing loss with the puretone threshold varying from 71 dBHL to 100 dBHL at octave frequencies from 250 Hz to 8000 Hz for air conduction. The air bone gap was within 10 dBHL with their speech detection threshold or speech recognition threshold and speech identification scores

Table 1: Chronological and language age of the participants for each sub group in the control group.

Sub groups	Participants	Chronological Age (in years)	Language age (in years)
1-2 years	1	2	1.6 - 2
	2	1.5	1 - 1.6
	3	1	1 - 1.6
2-3 years	1	3	2.6 - 3
	2	2.2	2 - 2.6
	3	2.9	2.6 - 3
3-4 years	1	4	3.6 - 4
	2	3.1	3 - 3.6
	3	3.8	3.6 - 4
4-5 years	1	4.2	4. - 4.6
	2	5	4.6 - 5
	3	4.3	4 - 4.6

Table 2: Chronological and language age of the participants for each sub-group in the clinical group.

Sub groups	Participants	Chronological Age (in years)	Language age (in years)
1-2 years	1	2.5	1.6 - 2
	2	5.5	1 - 1.6
	3	5.8	1 - 1.6
	4	6	1 - 1.6
2-3 years	1	4	2 - 2.6
	2	4.2	2.6 - 3
	3	3.8	2.6 - 3
3-4 years	1	5	3 - 3.6
	2	5.4	3 - 3.6
	3	5.2	3.6 - 4
4-5 years	1	4.9	4.6 - 5
	2	5.8	4 - 4.6
	3	6	4 - 4.6

correlating with the pure tone thresholds. They had 'A' type tympanogram with absence of ipsilateral and contralateral acoustic reflexes, in both the ears indicating normal middle ear function. TEOAEs were absent in both the ears indicating outer hair cell dysfunction. The ABR indicated severe hearing loss with the absence of wave V at 90 dBnHL. Their language age was ranging from 1 to 5 years. In the clinical group, ten out of thirteen children were fitted with the hearing aid before three years of age while three of thirteen children were fitted with the hearing aid beyond three years of age. The aided audiogram of all the participants was within the speech spectrum with the most appropriate hearing aid fitting.

Test Materials

Receptive Expressive Emergent Language Scales (REELS) developed by Bzoch and League (1971) and Standard language assessment tool, Language test in Kannada (KLT), developed as a part of UNICEF project at the Department of Speech Pathology, AIISH were used to assess the language age of the participants in both the control and clinical groups.

Test Environment

All the audiological tests were carried out in a sound treated room. The noise levels in the testing room were within the permissible limits as per ANSI S3.1 (1991).

Procedure

Stimulus generation: Stimulus /ba/ spoken by adult male Kannada speaker, into an unidirectional microphone was recorded using a PC with 16 bits processor with Adobe Audition 1.5 software, at a sampling rate of 48,000 Hz. The duration of the stimulus was 248.85 ms. Acoustic characteristics of stimulus /ba/ as obtained from reading the sound file using PRAAT software are given in Table 3.

Table 3: Acoustic characteristics of stimulus /ba/

Stimulus duration	248.85 ms	Mean pitch	136.81 Hz
Burst duration	26.33 ms	Mean intensity	74.98 dB
Transition duration	20.64 ms	Mean first formant frequency (f1)	389.06 Hz
Steady state (vowel) duration	201.87 ms	Mean second formant frequency (f2)	1185.18 Hz
Minimum pitch	120.86 Hz	Mean third formant frequency (f3)	2181.71 Hz
Maximum pitch	162.93 Hz	Mean fourth formant frequency (f4)	3739.63 Hz

A calibrated double channel Madsen Orbiter 922 (Version-2) diagnostic audiometer with TDH-39 headphones with impedance matched loudspeakers was used to present stimuli for behavioural observation audiometry (BOA). BOA was used to assess the child's responsiveness to sounds, for children in the age range of 1 to 1.6 years. TDH-39 headphones with MX 41AR cushion and B-71 bone vibrator were used to measure the hearing thresholds through air conduction and bone conduction respectively. VRA given by Liden and Kankkunen (1969) was used to assess pure tone threshold for children in the age range of 1.6 to 2 years. Thresholds were obtained for the warble tones at

octave frequencies from 250 Hz to 8000 Hz and bisyllabic word /papa/ was used to obtain speech detection threshold. Conditioned play audiometry was carried out to assess pure tone threshold for children in the age range of 2 to 5 years. Thresholds were obtained for the warble tones at octave frequencies from 250 Hz to 8000 Hz and bisyllabic word /papa/ was used to obtain speech detection threshold. Speech recognition threshold and closed set Speech Identification Scores were obtained for children above 4 years of age.

A calibrated Grason Stadler Inc. Tympstar was used to record tympanometry using a probe tone frequency of 226 Hz. The ipsilateral and contralateral acoustic reflex thresholds were measured at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz tones. TEOAE was measured using a calibrated Oto-acoustic Emission system ILO - V6 with the default setting mode. TEOAE was measured using non-linear clicks trains presented at 84±3 dB pe SPL of 260 sweeps. The overall TEOAE amplitude of 6 dBSPL above the noise floor, with the reproducibility of greater than 80% was considered as presence of TEOAE (Dijk & Wit, 1987). ABR testing was carried out using calibrated Biologic hearing system (Version 7.0) with ER - 3A insert ear phone, to estimate the threshold for those children in both the control and clinical group in whom reliable pure tone threshold could not be obtained. Subjects with presence of wave V at 30 dBnHL were considered as having normal hearing sensitivity and were recruited in the control group. Subjects with absence of wave V at 90 dBnHL were considered as having severe hearing loss and were recruited in the clinical group.

Aided audiogram: Aided audiogram was obtained for children in the clinical group, with the most appropriate hearing aid fitting, separately for the two ears and also binaurally. Child's aided responsiveness to sounds (for warble tones from 500 Hz to 4000 Hz separated in octaves and speech stimuli) was obtained using BOA for children in the age range of 1 to 1.6 years.

Aided thresholds were obtained using warble tones at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz using either VRA for children in the age range of 1.6 to 2 years or conditioned play audiometry for children in the age range of 2 to 5 years. Aided speech detection threshold was obtained for children below 4 years of age and aided closed set speech identification score was obtained for children above 4 years of age.

Language age assessment: REELS was used to assess receptive language age and expressive language age of all the participants in both the control and clinical group in the age range 0 to 3 years. KLT was administered and based on the child's pointing behavior or verbal response; child's language age was assessed. Language reception and expression is assessed under Part I: Semantics and Part-II: Syntax.

Recording of cortical auditory evoked potential (CAEP): ALLR was recorded using calibrated Biologic hearing system (Version 7.0) with Fostex PM 0.5 MKII loudspeaker for all the participants in both the control and the clinical group. ALLR was recorded while the child was made to sit comfortably in a reclining chair, watching a silent video. Older children were advised not to sleep and move during the test. A regular single channel recording was done. Electrode sites were cleaned using abrasive gel; Silver chloride electrodes were placed using Cz as the active electrode. Cz (non-inverting) refers to the vertex midline placement. The inverting electrode was placed on the nape of the neck and a ground electrode on the forehead. Natural stimuli /ba/ of duration 248.85 ms was presented at 1.1 Hz rate, in alternating polarity at 65 dBSPL. The stimuli was presented through a loud speaker at 0° azimuth and at 1m distance from the subject and at the subject's ear level. Number of sweeps was set to 300, filter settings to 1 to 30 Hz, analysis time window to 500 ms with 100 ms baseline and amplification to 25,000.

For the clinical group, binaural unaided and aided ALLR was recorded and for the control group, binaural unaided ALLR was recorded. ALLR was recorded twice in the same session to verify the reproducibility and averaged as the final response.

Analysis

The stored waveforms were recalled and analyzed later. P1, N1, P2, and N2 components were identified and marked visually by three experienced audiologists. Blind folded analysis was carried out and two out of three interpretations that correlated, was considered. Descriptive statistics was used to calculate the mean and standard deviation of P1 latency of each of the participants in both the control and clinical group. Though all the observed ALLR components were marked, only P1 was considered for statistical analysis, since, Sharma, et al., (2005) have reported that the P1 latency has been established as a biomarker for assessing the maturation of the central auditory system in children and that there are inconsistencies observed for the N1, P2, and N2 components of ALLR responses. The data were subjected to statistical analysis using SPSS (version 17). Kruskal-Wallis Test was carried out to study the effect of language age on P1 latency in the control and clinical group. Mann-Whitney Test was carried to study the effect of group for P1 latency.

Results and Discussion

ALLR results for the control group

ALLR was recorded for 12 children with normal hearing sensitivity. ALLR was present for all the children. Out of 12 children, 7 children had only P1-N1 components, while the remaining 5 children had all the ALLR (P1-N1-P2-N2) components. The mean and standard deviation of P1 latency for the control group across all the language age groups was calculated. The results are outlined in Table 4.

It is evident from the Table 4 that the mean P1 latency decreased as the language age increased from 1 to 5 years in the control group. That is, the P1 latencies were shorter for higher language age groups.

Comparison of P1 latency across language age in the control group

Kruskal-Wallis test was done to evaluate the effect of language age on P1 latency in the control group. The results revealed that there was statistically no significant effect of language age on P1 latency in the control group [Chi-square=7.61 with 3 df, p>0.05, where p=0.055]. It can be observed from table 4 that there was a negative correlation between P1 latency and language age in the control group, but there was statistically no significant difference across age groups in the present study. The possible reason could be the smaller sample size (n=12) considered in the control group. However, the negative correlation observed between P1 latency and language age in the present study, is in consonance with previous studies (Jang et al., 2010; Pang & Taylor, 2000; Sharma, et al., 1997; Sharma et al., 2002; Sharma et al., 2005). However, all these studies were carried out on a larger sample.

Table 4: Depicts the mean and standard deviation of P1 latency for the Control group.

Language age (in years)	Control group	
	Mean (in ms)	SD
1-2	315.92	35.77
2-3	214.95	54.22
3-4	174.26	46.83
4-5	121.48	22.88

Note: n=3 in each language age group

Jang et al., (2010) reported that there was statistically significant negative correlation between the P1 latency and age in Korean children with normal hearing aged 1.7 to 17.5 years (n=53) using synthetic /ba/ stimulus of 90 ms duration. Sharma et al. (1997), Sharma et al. (2002b) reported a strong negative correlation between age and latency of P1 in 136 normal hearing subjects ranging in age from 0.1 years to 20 years. They concluded that the decrease in P1 latency with increasing age suggests more efficient synaptic transmission over time and may reflect a more refined or pruned auditory pathway. Similar results were reported by Pang and Taylor (2000) in 69 normal hearing children aged from 3-16 years using natural stimulus /da/ of 212 ms duration and Sharma et al. (2005) in 50 normal hearing children aged 3-12 years, using natural speech syllable /uh/ of 23 ms duration by varying inter stimulus interval.

ALLR response patterns for different language age groups in the control group

The absolute latency of various ALLR components for each of the subject across various language ages in the control group was calculated and it is tabulated in Table 5.

The ALLR waveform morphology improved and the presence of P1, N1, P2 and N2 components increased

Table 5: Depicts the absolute latency of various ALLR components in each of the subject across various language ages in the control group.

Language age group (in years)	Participants	Language age (in years)	ALLR components			
			P1 (ms)	N1 (ms)	P2 (ms)	N2 (ms)
1 – 2 years	1	1.6 - 2	274.71	299.69	--	--
	2	1 - 1.6	336.13	410.77	--	--
	3	1 - 1.6	336.94	367.27	--	--
2 – 3 years	1	2.6 - 3	176.82	219.50	--	--
	2	2 - 2.6	274.76	296.62	--	--
	3	2.6 - 3	193.29	269.29	--	--
3 – 4 years	1	3.6 - 4	120.46	281.96	351.63	405.46
	2	3 - 3.6	205.90	348.40	--	--
	3	3.6 - 4	196.44	275.61	300.94	326.27
4 – 5 years	1	4 - 4.6	133.06	176.78	218.42	257.98
	2	4.6 - 5	95.13	161.63	237.10	257
	3	4 - 4.6	136.27	202.77	228.11	266.11

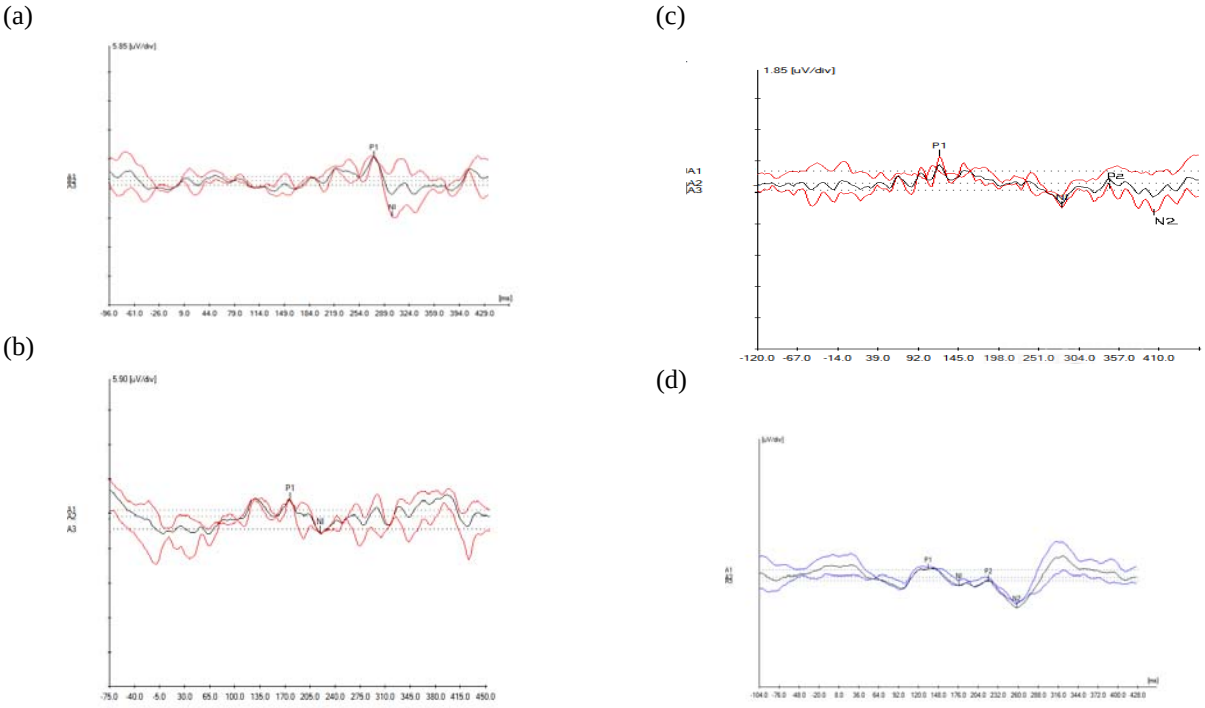

Figure 1: Depicts the ALLR waveforms of a subject in each of the subgroups (a - 1 to 2 years, b- 2 to 3 years, c - 3 to 4 years, d - 4 to 5 years) in the control group.

as the language age increased. Within a given language age group, children with a higher language age had earlier P1 latency than the children with a lower language age. Children with same language age within each language age group had similar P1 latencies. These findings are consistent with the previous study by Sharma et al., (2002b). They reported a strong negative correlation between age and latency of P1 in 190 normal hearing subjects ranging in age from 0.1 years to 20 years. The ALLR waveforms of one of the subjects from each of the language age group of the control group are given in Figure 1 [a–d].

ALLR results for the clinical group

ALLR was recorded in 13 children with bilateral severe to profound hearing loss. Both unaided and aided ALLR recordings were obtained. In the unaided

ALLR recording condition, ALLR was absent for all the 13 children. While, in the aided ALLR recording condition, out of 13 children ALLR was present for 10 children. Out of 10 children, 7 children had only P1-N1 components, while the remaining 3 children had all the ALLR P1, N1, P2, and N2 components. The possible reason for the absence of the ALLR response in unaided ALLR recording condition could be that the sound intensity (65 dBSPL) that was given was not sufficient enough to stimulate the regions responsible for the generation of ALLR response. The mean and standard deviation of P1 latency from the aided ALLR recording condition for the clinical group across all the language age groups were calculated. The results are outlined in Table 6. In the Table 6, the standard deviation is not provided for the first sub-group with the language age of 1–2 years because out of four children tested in this language age group, only one child had ALLR. It is evident from the Table 6, that the mean latency decreased as the language age increased from 1 to 5 years in the clinical group.

Comparison of P1 latency across language age in the clinical group

Kruskal-Wallis test was done to evaluate the effect of language age on P1 latency in the clinical group. The results revealed that there was statistically no significant effect of language age on P1 latency in the clinical group [Chi-square=5.95 with 2 df, $p>0.05$, where $p=0.051$]. It can be observed from Table 6 that there was a negative correlation between P1 latency and language age in the clinical group. But there was statistically no significant difference across age groups in the present study. The possible reason could be the smaller sample size (n=13) considered in the clinical group. The wave form morphology appeared more noisy and saw tooth like similar to what was reported by Jang et al. (2010) in a group of 10 cochlear implanted children in the age range of 3.3 –15.5 years.

Table 6: *Depicts the Mean and Standard Deviation of P1 latency for all the subjects across language ages for the clinical group.*

Language age (in years)	Clinical group	
	Mean (in ms)	SD
1-2 (n=4)	334.13	--
2-3 (n=3)	278.40	19.02
3-4 (n=3)	238.48	39.81
4-5 (n=3)	145.64	34.86

The negative correlation observed between P1 latency and language age in the present study, is in agreement with the previous studies (Sharma et al., 2004; Dorman et al., 2007). However, all these studies were carried out on a large sample. Sharma et al., (2004) reported that the decrease in P1 latencies and changes in response morphology are not unique to children who are cochlear implanted but rather reflect the response of a deprived sensory system to auditory stimulation through hearing aid. Dorman et al., (2007) reported that the P1 latency decreased by 200 ms over a duration of 4 months post stimulation period and also there was a progress in acquisition of speech and language as well in 245 congenitally deaf children who were fitted with cochlear implants. They concluded that the access to audition maintains neural plasticity and allows for the development of the central auditory pathways. It is likely that the development of early communication behaviors following early intervention may be promoted by normal development of the central auditory pathways.

ALLR response patterns for different age groups in the clinical group

The absolute latency of various ALLR components for each of the subject across various language ages in the clinical group is tabulated in Table 7.

The ALLR waveform morphology improved and the presence of P1, N1, P2 and N2 components was increased as the language age increased. Within a given language age group, children with a higher language age had earlier P1 latency than the children with a lower language age. Children with same language age within each language age group had similar P1 latencies. One of the ALLR waveforms from each of the language group of the clinical group is given in Figure 2 [a–d].

Comparison of P1 latency and language age across the control and clinical group

The mean and standard deviation of P1 latency for both the control and clinical group across language ages are given in Table 8.

As it is evident from Table 8, the mean P1 latency of both the control and clinical group decreased with increase in language age. It is also evident that the mean P1 latency of clinical group is longer compared to the mean P1 latency of control group across all the language ages.

For comparison of P1 latency obtained between the two groups, Mann-Whitney U-test was carried out. This in turn helped in knowing the relationship between the P1 maturation and language development in the control and clinical group. The results revealed

that there was no significant difference between the control and clinical group in terms of the P1 latency [z=-0.627, p>0.05, where p (2–tailed)=0.53]. Thus, the two groups were not significantly different from each other in terms of mean P1 latency across the language age. Hence, the present study reveals that with the auditory stimulation the central auditory pathways in children with hearing impairment develop in a similar fashion as seen in normal hearing children. Though, the latencies were prolonged in the clinical group compared to the control, it was not statistically significant due the small sample size that was considered in the present study (n=10). Also the number of subjects in the clinical group with all the P1, N1, P2, and N2 components (3 subjects) was less compared to the control group (5 subjects). These findings are consistent with previous studies (Ponton et al., 1996; Sharma et al., 2002a; Sharma et al., 2002b).

Table 7: Depicts the absolute latency of various ALLR components in each of the subject across various language ages in the clinical group.

Language age group (in years)	Participants	Language age (in years)	ALLR components			
			P1 (ms)	N1 (ms)	P2 (ms)	N2 (ms)
1 – 2 years	1	1.6 - 2	334.13	413.77	--	--
	2	1 - 1.6	--	--	--	--
	3	1 - 1.6	--	--	--	--
	4	1 - 1.6	--	--	--	--
2 – 3 years	1	2 - 2.6	299.65	339.20	--	--
	2	2.6 - 3	272.60	299.66	--	--
	3	2.6 - 3	262.96	405.46	--	--
3 – 4 years	1	3 - 3.6	275.61	319.94	--	--
	2	3 - 3.6	242.40	277.79	297.53	319.39
	3	3.6 - 4	197.44	276.61	--	--
4 – 5 years	1	4.6 - 5	107.99	191.27	--	--
	2	4 - 4.6	152.11	171.11	297.77	319.94
	3	4 - 4.6	176.82	219.50	254.90	275.75

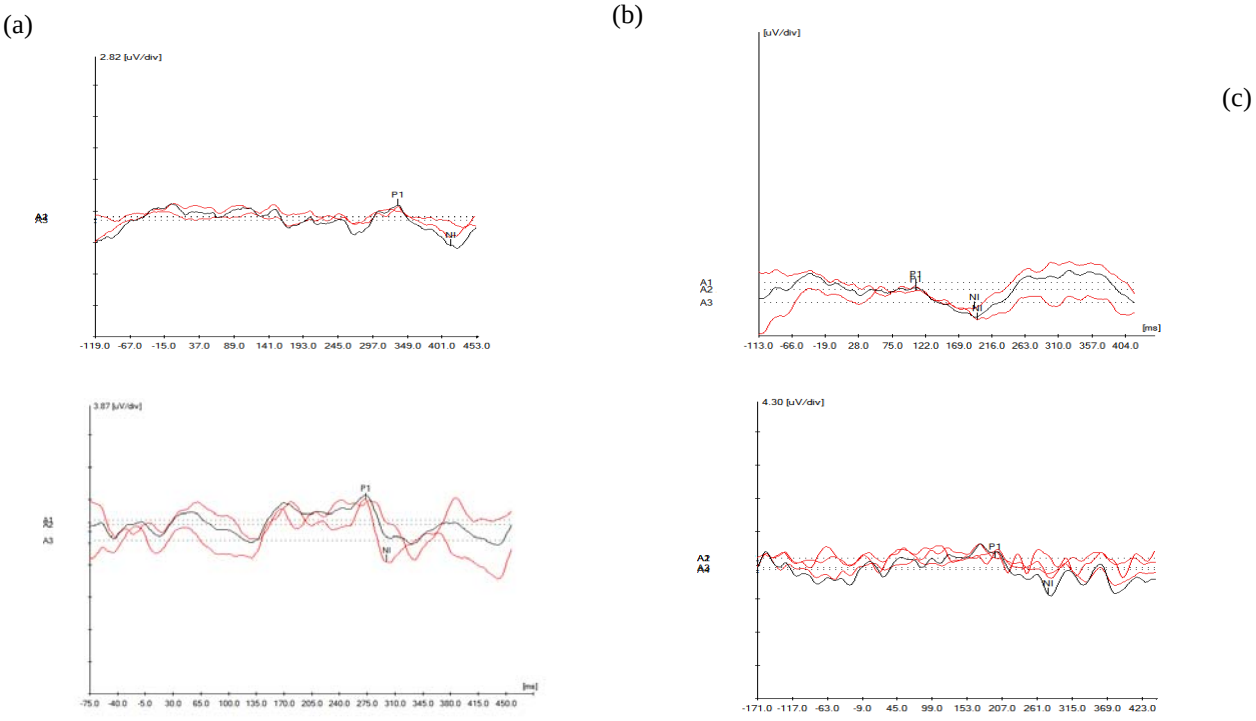

Figure 2: Depicts the ALLR waveforms of a subject in each of the subgroups (a – 1 to 2 years, b – 2 to 3 years, c – 3 to 4 years, d – 4 to 5 years) in the clinical group.

Table 8: Depicts the mean and SD of P1 latency for both the control and clinical group across language ages

Language age (in years)	Control group		Clinical group	
	Mean (in ms)	SD	Mean (in ms)	SD
1 - 2	315.92	35.77	334.13	--
2 - 3	214.95	54.22	278.40	19.02
3 - 4	174.26	46.83	238.48	39.81
4 - 5	121.48	22.88	145.64	34.86

Ponton et al., (1996) reported that the latency changes for P1 occur at the same rate as that of the normal hearing children. Other ALLR components namely N1 and P2 are either delayed in developing or absent in the implanted children. Sharma et al., (2002a; and b) reported prolonged P1 latencies in children with cochlear implants compared to normal hearing children. Further analysis revealed that P1 latency appears to continue a developmental progression after implantation.

In the present study, within a given language age group, children with higher language age had earlier P1 latency than the children with lower language age in both the control and clinical group. This indicates that there occurs a central auditory maturation in children with hearing impairment parallel to that seen in normal hearing children. In the present study, 10 participants who were considered in the clinical group were fitted with their most appropriate hearing aid and started receiving speech, language and listening therapy before 3 years of age, which is considered as the sensitive period. For all the 10 participants who were fitted with their most appropriate hearing aid before 3 years of age, the change in P1 latency across language age groups was similar to subjects in the control group across language age groups. Whereas, ALLR was absent in three children who were identified and fitted with hearing aid beyond 3 years of age. This finding is in consonance with previous studies (Sharma & Dorman, 2006; Sharma, Nash & Dorman, 2009; Gilley, Sharma & Dorman, 2008; Sharma et al., 2002a; Kral et al., 2002).

Sharma and Dorman (2006) reported that in the early implanted children, waveform morphology was normal and characterized by a broad positivity within a week following the onset of stimulation. Sharma et al., (2009) reported that the latency of P1 has been used to examine central auditory system maturation in children with cochlear implants and also they have reported smaller changes in children who were fitted with a cochlear implant early in childhood and larger changes in children fitted later in childhood with respect to normal hearing children.

Gilley, Sharma, and Dorman (2008) analyzed ALLR for speech sound to document the areas of activation in the cortices of normal hearing children and age-matched children who received cochlear implants before and after the sensitive period age cut-offs described by Sharma et al., (2002a). Normal hearing children showed bilateral activation of the auditory cortical areas (superior temporal sulcus and inferior temporal gyrus). Children who received cochlear implants at an early age (<3.5 years of age at fit) showed activation of the auditory cortical areas contralateral to their cochlear implant which resembled that of normal hearing subjects. It also initiated a more widespread (typical) sequence of activation within and between cortical layers resulting in robust cortical responses and shorter response latencies over time. However, late-implanted children (>7 years fit age) showed activation outside the auditory cortical areas and abnormal or absence of auditory cortical activity in the late implanted children. The study suggests absent or weak connections between primary and association areas, and subsequently, weak feedback activity to thalamic areas.

These results are consistent with Kral's decoupling hypothesis (Kral et al., 2002) which suggests that a functional disconnection between the primary and higher order cortex underlies the end of the sensitive period in congenitally deaf cats, and presumably, in congenitally deaf, late-implanted children. Similar findings were also documented, that congenitally deaf children fit with cochlear implants can achieve high levels of oral speech and language skills (Pisoni, Cleary, Geers & Tobey, 1999; Svirsky, Teoh & Neuburger, 2004). However, success depends very critically on the age at which a child receives an implant (Connor et al., 2006; Lee et al., 2004). Lee et al. (2004), reported scores on the Korean version of the CID sentences as a function of a child's age at the time of implantation. Children implanted before the age of 4, generally achieve high scores on the task of sentence recognition. Children implanted after the age of 7 generally achieve poor scores. Children implanted between age 4 and 7 showed a complete range of scores. Sharma et al., (2005) reported that both the latency and morphology of the P1 wave can serve as the biomarkers for the developmental status of the

central auditory pathways. Thus, it can be concluded that the P1 latency can be used as biomarker to know the developmental status of the central auditory maturation in both normal hearing children and children with hearing impairment. Also that the P1 latency can be used as an objective measure to assess the language development in normal hearing children and in children who are fitted with hearing aid.

Summary and Conclusions

Results obtained from the present study revealed that the P1 latency decreases with increase in age and this negative correlation between P1 latency and language age in the present study was similar in both the control and the clinical group. Three children who were identified and rehabilitated beyond 3.5 years of age showed absence of aided ALLR while 10 children who were identified and rehabilitated before 3 years of age had P1 latencies similar to that seen in normal hearing children though the latencies were slightly prolonged. Thus, it can be concluded that P1 latency can be an effective objective tool to know the central auditory maturation and language development in children who are fitted with hearing aid.

Implications

Present study would give an idea about the relation between language development and P1 maturation in hearing impaired children. Present study would give an idea about the trend of changes in P1 latency across language age group in normal hearing and in children who are fitted with hearing aid. Study also suggests the use of P1 latency as a measure of central auditory maturation and language development. Further research can be carried out on a larger sample to know and establish the effect of P1 latency on various language age groups. Further research can be carried out on a larger sample to know the efficacy of P1 latency in predicting the language age in hearing aid users.

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