CENTRAL AUDITORY MATURATION AND LANGUAGE DEVELOPMENT IN CHILDREN WITH HEARING LOSS: A PRELIMINARY STUDY

Register No: 09AUD013

A dissertation submitted in Part of fulfilment for the degree of

Master of Science (Audiology)

University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING,

MANASAGANGOTHRI, MYSORE - 570006

June - 2011

Dedicated

To

Dad,

Mom, Dikku, Aunty,

Mama & Sushruth

CERTIFICATE

This is to certify that this dissertation entitled "***Central auditory maturation and language development in children with hearing loss: a preliminary study"*** is a bonafide work submitted in part of fulfilment for the degree of Master of Science (Audiology) of the student Registration No.: 09AUD013. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any diploma or degree.

Mysore

June, 2011

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CERTIFICATE

This is to certify that this dissertation entitled "***Central auditory maturation and language development in children with hearing loss: a preliminary study"*** has been prepared under my supervision and guidance. It is also certified that this dissertation has not been submitted earlier to any other university for the award of any diploma or degree.

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DECLARATION

This is to certify that this master's dissertation entitled "***Central auditory maturation and language development in children with hearing loss: a preliminary study"*** is the result of my own study under the guidance of **Ms. Mamatha. N.M**, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier to any other university for the award of any diploma or degree.

Mysore **Register No. 09AUD013**

June, 2011

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Chapter 1

INTRODUCTION

Central auditory maturation is a constant process from pre natal period to puberty. The human auditory system starts functioning by the 6th month of gestation when the auditory mechanisms are ready to respond to a sound. During the pre natal period, the middle ear, cochlear, auditory nerve, and neural pathways of the brainstem are mature enough to function (Tucci, 1996).

Development of the peripheral auditory system (ear and auditory brain-stem) 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). The changes in brain organization continue into adolescence, though, the major changes occur during the first year of life and most of them are accomplished by the end of the second year. The anatomical developments parallel the functional maturation: the auditory function is one of the earliest to emerge. Thus, the time required for the stimulus to cross the synaptic junctions continues to shorten until about 3 years of age. 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 5-7 years 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 (cited in Vihman, 1988). With respect to comprehension, children continue to improve in their ability to understand speech under noisy circumstances up to the age of 15 years. Peter Jusczyk's (1997) model of the development of speech perception and word learning suggests that the infant or toddler learns to attend automatically to the relevant information in the speech signal, which will then allow for efficient and rapid identification of native-language speech categories, and thus, better word learning and speech segmentation.

These correlations between Central auditory maturation and language development have been studied behaviorally. 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. Cohen, Gelinas, Lassonde, and Geoffroy (1991) reported that language-impaired children had more difficulty than controls in discriminating place of articulation contrasts only when they were presented to the left ear, as well as a difficulty in discriminating voice contrasts selective to the right ear.

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), that provide a complex but rich source of information about the central nervous system pathways and structures activated by auditory stimulation. Auditory evoked potentials (AEPs) reflect maturation of the

human brain through changes in their latency, amplitude and morphology (Eggermont, 1989; Courchesne, 1990).

AEPs evoked by consonant-vowel syllables provide an opportunity to assess auditory pathways engaged in the acoustic analysis of speech. Also the neurophysiologic responses to these speech sounds because the representation of such sounds is undoubtedly important for speech and language development.

Need for the study:

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. And 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 3–6 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 (Sharma, Dorman, Martin, Roland & Gilley, 2007).

Series of investigations by Sharma and Dorman (2006), has demonstrated that, cortical maturation reaches normal in 3-6 months of stimulation, if children with hearing loss identified and rehabilitated below 3.5 years of age and delayed maturation is noticed if age of identification is more than 7 years of age. And also 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 fitted with hearing aid and normal hearing children. Thus, the current study was taken with the following aims.

Aim of the study:

The aim of the present study was

- To know the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children.
- To compare the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children.

Chapter 2

REVIEW OF LITERATURE

Central auditory maturation is a constant process from pre natal period to puberty, while, the majority of development occurs in the first 3 years of life. During the pre natal period, the inner ear has developed already and function adult like by birth. Lasky and Williams (2005) reported that for the first half of pregnancy, the fetus is unresponsive to sound and by term, basic auditory capabilities relatively mature. The axonal conduction time is adult-like by 40 weeks conceptional age, which is in good agreement with the fact that the auditory nerve and brainstem auditory pathways are well myelinated by the time of term birth (Perazzo, Moore & Braun, 1992; Tucci, 1996; Volpe, 1995; Yakovlev & Lecours, 1967). This is evident from the normal auditory brainstem responses.

Major developmental processes that occur after birth are mostly related to the maturation of the cerebral cortex. Central auditory pathways of the human brain exhibit progressive anatomical and physiologic changes through early adulthood (Kraus et al., 1985; Courchesne, 1990; Huttenlocher, 1979). Cortical development during the first year of life is characterized by an increase in synaptic density, number of synapses per neuron, and dendritic growth (Huttenlocher, 1979). The sequence of cortical synaptogenesis appears to parallel the maturation of cortical functions: in the human primary auditory cortex (Heschl's gyrus) and these structural developments are paralleled by the auditory functional maturation. The time required for the impulse to cross the synaptic junctions continues to shorten until about 3 years of age.

Exposure to a specific language in the first 6 months of life alters infant's phonetic perception (Kuhl, Williams, Lacerda, Stevens & Lindblom, 1992). Eilers, and Oller (1994), Eilers, Cobo-Lewis, Vergara, Oller, and Friedman (1996), Eilers, Gavin, and Oller (1982) reported that normal hearing infants undergo several stages of speech like development. During the first 2 months of life, newborns produce comfort sounds. These sounds appear to be the precursors of vowel production. Between 2 and 3 months, infants enter a gooing stage. During this stage, they learn to articulate in the back of the mouth and, therefore, acquire a repertoire of vowel-like and g-like sounds.

Between 4 and 6 months, infants expand their vocal repertoire to include growls, yells, whispers, squeals, and isolated vowel-like sounds. Well formed syllables appear between age 7 and 10 months during the canonical babbling stage. During this stage, the use of reduplicated sequences such as [mamamama] or [dadadada] begins. Reduplicated babbling is particularly important since it signals the first use of adult like syllables and paves the way for a child to develop his or her first words

Jusckzyk and Houston (1998), Jusckzyk and Luce (2002) reported that by 9 months of age children can recognize their own names, utter their first words, respond appropriately to mommy and daddy. They even begin to segment words, retain information about frequently occurring words and show language specific preferences for prosodic cues.

By 10-12 months of age, sensitivity to non-native language decline and infants appear to integrate various types of word segmentation cues. They even begin to utter using 2- 3 word short sentences or phrases. By 17 months of age, infants show lexical

competition effects which affect word learning. By 2 years of age they talk in sentences, while the adult-like language usage appears by 5-7 years of age.

Peter Jusczyk's (1997) model of the development of speech perception and word learning suggests that the infant or toddler learns to attend automatically to the relevant information in the speech signal, which will then allow for efficient and rapid identification of native-language speech categories, and thus, better word learning and speech segmentation.

The normal language learning and development occur only with early exposure to language. Conversely if the language exposure begins later in life, asymptotic performance in the language declines (Newport, 1990). This phenomenon is termed "Sensitivity period" for language development.

The key factor critical to language development is the normal auditory functioning in the early years of life within the Sensitivity period for language development. Auditory functioning can be assessed behaviorally as well as electrophysiologically.

The correlations between Central auditory maturation and language development have been studied behaviorally. 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. Cohen, Gelinas, Lassonde, and Geoffroy (1991) reported that language-impaired children had more difficulty than controls in discriminating place of articulation contrasts only when they were presented to the left ear, as well as a difficulty in discriminating voice contrasts selective to the right ear.

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), that provide a complex but rich source of information about the central nervous system pathways and structures activated by auditory stimulation.

Auditory Evoked Potentials (AEPs):

By thoroughly characterizing AEP maturation, it may be possible to determine whether a relationship exists between age-related physiological changes reflected in the AEPs and the development of both normal and abnormal auditory behavioral skills. For example, many auditory perceptual skills, such as the ability to recognize degraded speech (Palva & Jokinen, 1975; Marshall, Brandt, Marstonm & Ruder, 1979) or to accurately understand speech presented in background noise (Elliott, 1979) do not become adult-like until adolescence.

Maturational changes in other basic auditory processing skills including gap detection (Trehub, Schneider & Henderson, 1995), masked thresholds (Schneider & Trehub, 1992), and minimum audible angle (Litovsky, 1997) also have extended maturational time courses. The prolonged maturational time courses of these perceptual skills cannot be attributed to immaturities in either cochlear function, which is essentially adult-like at or before the age of normal term birth (Eggermont, Brown, Ponton & Kimberley, 1996; Abdala & Sininger, 1996), or the auditory brainstem pathway, which generates adult-like electrophysiological responses by 2 years of age (Ponton,

Eggermont, Coupland & Winkelaar, 1992). Thus, AEPs can be used to assess the developmental status of the auditory system.

The maturational changes in synaptic density and efficacy might be the major neural substrates underlying the maturation of cortical ERPs. It therefore seems more likely that the prolonged development of at least some auditory perceptual skills is related to ongoing maturational changes in the thalamic-cortical portions of the central auditory system. Maturation of the thalamic-cortical portions of the central auditory system can be assessed by recording age-related changes in the neurophysiologic responses evoked by auditory stimulation.

Electrophysiological responses are non-invasive, objective, and it is precisely measured using auditory evoked potentials (AEPs). AEPs such as, auditory brainstem response (ABR), are widely used to assess neonatal auditory sensitivity and to detect abnormalities of peripheral and subcortical portions of the auditory pathways. However, ABR does not provide information on cortical auditory processing.

The clinical usefulness of middle latency response (MLR), following ABR at latencies of 10 to 50 ms, has been suggested to be limited, since the reliability of MLR is low in the first 5 years of life (Kraus et al., 1985). Stapells and Kurtzberg (1991) found that auditory evoked potentials in a child with higher cortical dysfunction showed normal ABR and MLR results, whereas long-latency ERPs were absent. Thus, testing by the ABR or MLR alone would have missed this child's cortical dysfunction.

Auditory Long latency Response (ALLR), following MLR, is thought to reflect responses central to the brainstem. Long latency ERPs are more variable and are elicited

less reliably near threshold than ABR. However, they offer a unique opportunity to evaluate higher-order cortical auditory processes (Stapells & Kurtzberg, 1991).

Normal maturation of the central auditory system affects the development of speech recognition and the ability to speak. Thus, central auditory pathway shows a progressive change in anatomy and physiology as person's age increases (Kraus et al., 1985). The cortical auditory evoked potential (CAEP) reflects the cerebral maturation through the change in latency and the shape of the waveform.

Auditory Long latency Responses (ALLR):

Cortical auditory evoked potentials (P1-N1-P2) are slow "obligatory" potentials that occur within about 300ms after the stimulus onset in adults (Burkard, Eggermont & Don, 2007). Components of these potentials (P1, N1 & P2) primarily reflect sensory encoding of auditory stimulus attributes. Cortical potentials are affected by both arousal level and stages of attention and are typically recorded when the subjects is awake and alert or in a light sleep stage (Amadeo & Shagass, 1973).The obligatory P1, N1, P2, and N2 components of cortical auditory evoked potentials (CAEPs) provide an important index of auditory system function and plasticity (Ponton & Eggermont, 2001).

The auditory input first reaches the auditory cortex as early as 20 – 30 ms after stimulation as reflected in the auditory middle latency response (Kraus & McGee, 1993; McGee, Kraus, Comperatore & Nicol, 1991). The early components of the CAEP such as the P1 and N1, which have longer latencies in childhood, reflect second order processing in the auditory cortex, including input from feedback and recurrent loops between primary auditory and association areas (Sharma et al., 2007). Evidence from intracranial

recordings in humans, as well from animal models, suggests that the neural generators of the P1 CAEP originate from the thalamo-cortical projections to the auditory cortex and may represent the first recurrent activity in the auditory cortex (Kral & Eggermont, 2007; Liegeois-Chauvel, Musolino, Badier, Marquis & Chauvel, 1994; Ponton & Eggermont, 2001).

In adults, the most conspicuous complex of the long-latency AEPs is formed by a positive - negative - positive - negative sequence, typically labeled P1, N1, P2, and N2. In adults, the latency range of these AEPs starts roughly at 40 ms - 50 ms and continues for another 150 ms - 250 ms.

ALLR components and their Anatomical basis:

Intracerebral recordings in humans indicate that a major source of neural activity contributing to the P1 peak originates from the lateral portion of Heschl's gyrus, i.e. the secondary auditory cortex (Liegeois-Chauvel et al., 1994).

The P1 is followed by a usually larger N1 response, peaking at about 100 ms. This peak is a sum of at least three sub-components (Näätänen & Picton, 1987): 1) The supratemporal N1 (N1b), largest fronto-centrally, originating bilaterally in the superior temporal cortex, including primary auditory cortex; 2) The non-specific N1, maximal over vertex, generated in the modality non-specific brain areas (Näätänen & Picton, 1987), partly in frontal lobe (Alcaini, Theyenet & Pernier, 1994); and 3) The T–complex (Wolpaw & Penry, 1975), largest at temporal electrodes, consisting of a smaller positivity at about 100 ms and a larger negativity at about 150 ms. In adults, the elicitation of the N1 response by threshold level auditory stimuli correlates with behavioral sound

detection (Parasuraman, Richer & Batty, 1982; Squires, Squires & Hillyard, 1975), and its amplitude increases with the increase of sound intensity / loudness (Picton, Hillyard, Krausz & Galambos, 1974; Picton, Woods, Baribeau- Braun & Healey, 1977).

It is reported that the N1 can be elicited from the age of 3 years, but only with a slow stimulation rate (Paetau, Ahonen, Salonen & Sams, 1995; Sharma, Kraus, McGee & Nicol, 1997), this suggests, longer refractory periods of N1 generators in children, which could also be attributed to immaturity of cortico-cortical connections (Webb, Monk & Nelson, 2001).

The N1 is followed by a positive wave P2 peaking at approximately 180-200 ms from stimulus onset (Näätänen, 1992; Ponton, Eggermont, Kwong & Don, 2000). The source of the P2 has been located by magnetoencephalography (MEG) to the superior temporal gyri anterior to the source of the supratemporal N1 (Hari et al., 1987). In addition, some results indicate that the P2 at least partially reflects auditory driven output of the mesencephalic reticular activating system (Knight, Hillyard, Woods & Neyille, 1980; Rif, Hari, Hamalainen & Sams, 1991).

The P2 peak is often followed by a negativity, labeled N2 (Picton et al., 1974). This peak has an adult latency of 220-270 ms (Ponton et al., 2000) and was suggested to be generated in the vicinity of the supratemporal planes, possibly including frontal activity (Ceponiene, Rinne & Näätänen, 2002, Gomot, Giard, Roux, Barthelemy & Bruneau, 2000). The N2 elicited by frequent repetitive stimuli (Näätänen & Picton, 1986) was reported mostly in children (Ceponiene, Cheour & Näätänen, 1998; Enoki, Sanada, Yoshinada, Oka & Ohtahara, 1993; Karhu et al., 1997; Korpilahti & Lang, 1994), but it was also shown in adults (Ceponiene, Shestakova, Balan, Alku, Yaguchi & Näätänen,

2001; Karhu et al., 1997; Picton et al., 1974; Ponton et al., 2000), but with a smaller amplitude (Ponton et al., 2000; Ceponiene et al., 2001), and, in some reports, longer latency (Ponton et al., 2000). In children, the N2 amplitude was larger in response to complex rather than simple tones (Ceponiene et al., 2001) and to low rather than high-pitched tones (Korpilahti, Ceponien, Alpo-Laurinsalo, Laurinsalo & Näätänen, in prep). Unlike the N1, children's N2 is largely insensitive to stimulus rate (Ceponiene et al., 1998, 2001).

Thus, the existing evidence suggests that maturation of the AEPs and their underlying generators may have distinct maturational time courses (Kraus et al., 1993).

Development of ALLR components:

In newborns and infants, auditory ERPs have no resemblance to adult ERP waveform. The adult P50-N100-P200 (P1-N1-P2) complex is not readily identifiable in infants and children before about 10 years of age (Ponton et al., 2000; Courchesne, 1990). Most of the ERP studies in infants have reported a large positive deflection at midline electrodes, with a maximum at about 300 ms, followed by negativity at about 600 ms (Barnet, Ohlrich, Weiss & Shanks, 1975; Graziani, Katz, Cracco, Cracco & Weitzman, 1974; Ohlrich, Barnet, Weiss & Shanks, 1978; Pasman, Rotteveel, deGraaf, Stegeman & Visco, 1992; Rotteveel, Colon, Stegeman & Visco, 1987; Shucard, Shucard & Thomas, 1987).

In infants with normal hearing, the average latency of the P1 waveform is about 300 ms. A rapid decrease in latency occurs during the first few years of life; a normal P1 latency for a 3 year old is about 125 ms (Sharma et al., 1997; Sharma, Dorman & Spahr,

2002). A smaller decrease in P1 latency is expected from there onwards and by the age of 15 years the average P1 latency decreases to approximately 95 ms. The mean P1 latency in middle aged adults (22 years to 25 years of age) is approximately 60 ms (Sharma et al., 2002; Lee et al., 2001).

P1 latency and amplitude demonstrated significant differences between school age (5 to 7, 8 to 10 yr) and older years (13 to 15, 19 to 27, 55 to 78 yr). Beyond young adulthood, there were no significant developmental changes. Accordingly, it may be concluded that P1 is mature by around 20 years of age (Cunningham, Nicol, Zecker & Kraus, 2000). Ponton, Don, Eggermont, Waring & Masuda, (1996) reported that P1 decreases in latency and reaches adult values at 19 years of age.

Sharma et al., (2007) reported that the latency of P1 reflects the accumulated sum of delays in the synaptic propagation through the peripheral and central auditory pathways. Therefore, the gradual decrease in latencies probably results from a gradual increase in neural transmission speed, which is related with changes of myelination as well as an increase in synaptic synchronization. Also, the latency of the P1 wave is thought to reflect the sum of synaptic transmission delays throughout the central auditory pathways. Latency changes in the P1, as a function of increasing age, reflect the maturation of the central auditory pathways occurring in response to auditory stimulation.

While the late components of the AEP have been extensively studied in adults, little is known about the emergence and development of P1/N1 in childhood. The P1/N1 complex is a robust and ubiquitous component of the adult response. In contrast, its characteristics are ambiguous in children. Some studies have reported that the latency of N1 decreases as age increases up to 16 years (Tonnquist-Uhlen et al., 1995) and up to 20

years (Johnson, 1989), and N1 amplitude increases up to 15 years of age (Martin, Barajas, Femandez & Torres, 1988). Conversely, other studies have reported little or no age related changes in N1 latency (Ohlrich et al., 1978; Martin et al., 1988) and amplitude (Fuchigami et al., 1993). Recent studies have also suggested that the component analogous to the adult N1 may not emerge until 8 or 10 years of age (Csepe, 1995; Ponton, Don & Masuda, 1996).

These inconsistent findings may be in part due to the differences in age ranges, stimuli and number of subjects used in the above studies. It is clear that large population studies across wide age ranges are required to describe maturation of P1 and N1 responses.

Ponton et al., (2000) reported that the largest changes between the youngest and oldest subjects in the age range of 5 - 20 years occurred for the two earliest peaks considered in their study, the P1 and N1b peaks of the late AEP. Less consistent and smaller age related changes were noted for P2 peak latency. In contrast to the latency decreases observed for the P1 and N1b peaks, the N2 peak increased in latency as a function of age.

Whereas virtually all of the subjects had a measurable P1, the presence of N1b across subjects was inconsistent until 9 years of age. Moreover, none of the 5 - 6 year olds had a measurable N1b at electrode C3, ipsilateral to the stimulated ear. The latency of N1b, similar to P1, decreases with age from approximately 120 ms - 150 ms in the 5 - 6 year olds (as measured at C4 and the central electrodes Fz and Cz) to 80 ms - 100 ms in the 18 - 20 year olds and N1b latency and amplitude is essentially adult like by age 15 - 16 year.

The maturational time constants for P1, N1b and N2 latencies are all around 6 years. During this period, the latencies for P1 and N1b decrease and that of N2 increases by approximately one third. In contrast, P2 latency does not change over the 5-20 year age span covered in this study, indicating that the generators of the P2 peak may be adult like by age 5 or earlier.

Latency and amplitude of the N1-P2 complex have been studied in association with auditory system maturation and deprivation, rather than perceptual learning. Studies report significant changes in response latency and amplitude well into adolescence (Ponton et al., 1996; Ponton et al., 2000; Sharma et al., 1997).

The AEP data reported by Barnet et al., (1975) indicate that the latency of the P2 peak reaches adult values at the age of 2 - 3 years, closely following the maturational time course of the auditory brainstem response wave I-V interpeak latency. Assuming the proposed generators of the P2 peak are correct, these findings suggest a fast maturation of the reticular activating system pathway, perhaps only limited by brainstem maturation. In contrast, the AEPs putatively generated by the lemniscal pathway, such as N1b, mature more slowly and do not reach adult latency values until adolescence (Courchesne, 1978, 1990; Sharma et al., 1997).

Studies focusing on development of ALLR components using various stimuli

Clicks as stimuli:

Arehole, (1995) recorded long latency responses (LLR) using clicks, from children with learning disabilities (LD) and to compare the findings with a normal group

of children. The results revealed that the inter peak latency (P2 – P1) was found to be significantly different between two test groups.

Tone burst as stimuli:

Tonnquist-Uhlén et al., (1995) analyzed late auditory evoked potentials in 34 healthy, normal hearing children aged 8-16 years, using a 100 msec, 500 Hz tone burst. The resulting auditory evoked potentials showed a prominent N1, after about 100 msec also, there was a significant decrease in the latency of N1 with increasing age.

Speech as stimuli:

AEPs evoked by consonant-vowel syllables provide an opportunity to assess auditory pathways engaged in the acoustic analysis of speech. Also the neurophysiologic responses to these speech sounds because the representation of such sounds is undoubtedly important for speech and language development.

Novak, Kurtzberg, Kreuzer & Vaughan, (1989) studied the maturation of the auditory ERPs to speech stimuli (/da/ & /ta/ syllables) from birth to 6 months. The P2-N2 complex recorded at birth changed in morphology by the age of 3 months. The authors discerned two positive peaks in latency range of the infantile P2 (P1m & P2m) with different scalp predominance: the P1m was larger frontally than centrally, whereas the P2m was largest centrally. A discontinuity (negative trough) between these two positive peaks, at about 160-200 ms, was termed N1m by the authors. The N1m became prominent by the age of 6 months. During the first 6 months of life, the P1m and P2m increased in amplitude and gradually decreased in latency. Further, Kurtzberg, Vaughan & Novak, (1986) reported that between 6 and 9 months, the amplitude of the second

major positive peak (P2m) markedly decreased, and that between 9 and 12 months, the amplitude of the preceding negativity (N1m) increased

ALLR in normal hearing children:

Kushnerenko (2003) reported that during the second half of the first year of life to one year of age, ALLR was characterized by the strong growth of the negative amplitudes (N250 & N450 peaks). Sharma et al., (1997), Sharma, Dorman, Todd, and Spahr (2002) assessed 136 normal hearing subjects ranging in age from 0.1 years to 20 years and they found a strong negative correlation between age and latency of P1. 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.

Jang et al., (2010) assessed the P1 latency in Koreans with normal hearing according to the age during development and reported that there was a statistically significant negative correlation between the P1 latency and age and the findings support that the maturation of the central auditory pathways could be measured objectively using the P1 latency.

Courchesne (1990) reported for children as young as 2 – 4 years of age the waveform is dominated by a large positivity (P1) followed by a broad negativity (N1b). Pasman, Rotteveel, deGraaf, Maassen, and Visco (1999) recorded CAEPs in young children aged 0 to 4 years and obtained latencies in the approximate latency ranges of 150 - 200 ms for P1 and 300 - 350 ms for the late negativity after P1.

Pang and Taylor (2000) recorded ALLR in 69 normal hearing children aged from 3-16 years. They used natural stimulus /da/ of 212 ms duration and they found a strong negative correlation between age and latency of P1. Gilley Sharma, Dorman, and Martin (2005) recorded ALLR in 50 normal hearing children aged 3 - 12 years. They used natural speech syllable /uh/ of 23 ms duration and they also varied the inter stimulus interval (offset-to-onset) of 2000, 1000, 560, and 360 ms preceding each presentation within the stimulus train and they found a strong negative correlation between age and latency of P1 at all stimulation rates.

Ponton et al., (2000) recorded ALLR 118 normal hearing subjects between 5 and 20 years of age and reported that the P1 and N1 latency decreases with age, while P2 and N2 latency show small change in latency with age. They also reported that the peak latency is around 80 ms -110 ms in 5-6 year old normal hearing children.

ALLR in hearing aid users:

Sharma et al., (2004) examined the development of P1 latencies in a child with congenital moderate-to severe sensorineural hearing loss who was fitted with hearing aids at the age of 11 months. P1 latencies were outside normal limits at the time of initial fitting with the hearing aid and decreased to within normal limits 5 months after hearing aid use. The P1 latencies continued to develop normally with 12 months of hearing aid use. This change in response morphology was similar to that found for children who were fitted with cochlear implants.

Sharma, Dorman, and Kral (2005) reported that the auditory stimulation does not necessarily need to come in the form of a cochlear implant in order for the central

auditory pathways to develop normally. In general, children who receive early access to sound have better scores in open set speech discrimination measures than those who are affected by auditory deprivation for longer periods.

ALLR in cochlear implant users:

Sharma et al., (2007) reported that the P1 latency decreased by 200 ms over duration of 4 month 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. They also reported that it is likely that the development of early communication behaviors following early intervention may be promoted by normal development of the central auditory pathways.

Ponton et al., (1996) assessed ALLR in normal hearing children and in children who were fitted with cochlear implant. They found that the latency changes for P_1 occur at the same rate for implanted children, but the overall maturation sequence is delayed. Other typical features of the ALLR, namely N_1 and P_2, are either delayed in developing or absent in the implanted children. By extrapolation of their data, they reported that the age at which P_1 latency becomes adult-like is delayed by approximately 5 yr for the implanted population. It was found that for implanted children, the 5 year delay for maturation of P_1 latency roughly corresponded to the average 4.5 year interval between the onset of deafness and the time of implantation.They concluded that there are both similarities and differences in cortical auditory maturation for normal-hearing and implanted children. These findings suggest that during the period of deafness, maturation

of cortical auditory function does not progress. However, some, if not all, maturational processes resume after stimulation is reintroduced.

Auditory stimulation within the sensitivity period and ALLR development:

Sharma, Dorman, and Spahr (2002) examined the development of P1 response latencies in 104 congenitally deaf children who had been fit with cochlear implants at ages ranging from 1.3 yr to 17.5 yr and three congenitally deaf adults. The results revealed that implanted children, with the longest period of auditory deprivation before implantation by about 7 or more years had abnormal cortical response latencies to speech. Children implanted within 3.5 years evidenced age-appropriate latency responses within 6 months after the onset of electrical stimulation. The ALLR waveform obtained from cochlear implant users, implanted before 3.5 years of age is depicted as a grand average waveform in Figure 2.1.

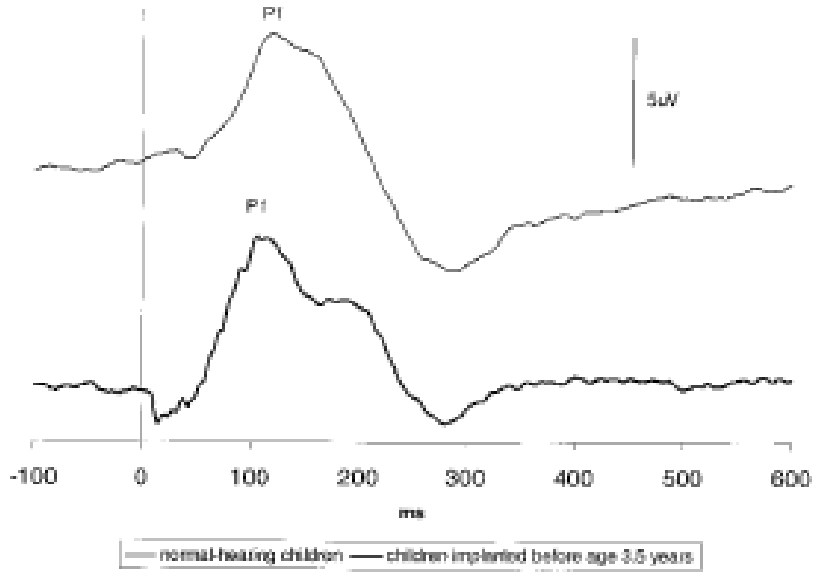

Figure. 2.1. Grand average ALLR waveform obtained from cochlear implant users, implanted before 3.5 years of age (bottom waveform). Grand average ALLR waveform of age – matched normal hearing children (top waveform).

Sharma et al., (2005) examined the longitudinal development of the P1 cortical response in groups of early and late implanted children (The age cut-offs for the two groups <3.5 years for early-implanted and >7 years for late implanted) and reported that for early-implanted children, at implant activation the waveform was dominated by a large negativity preceding the P1 response. This negativity was consistently seen in congenitally deaf children at the time of implantation and in profoundly hearing-impaired children at the time of initial fitting with a hearing aid (Sharma et al., 2004). This early negativity is strikingly similar to the 'long-latency negative potential' reported in studies on preterm infants before 25 weeks postconception (Salamy, Eggermont & Eldredge,

1984; Weitzman, Graziani & Duhamel, 1967). The similarity suggests that CAEP morphology and latency at the time of implantation can be interpreted as a sign of a naive (i.e., unstimulated) auditory system. Figure 2.2 depicts the ALLR waveforms obtained from normal hearing children and children with hearing impairment.

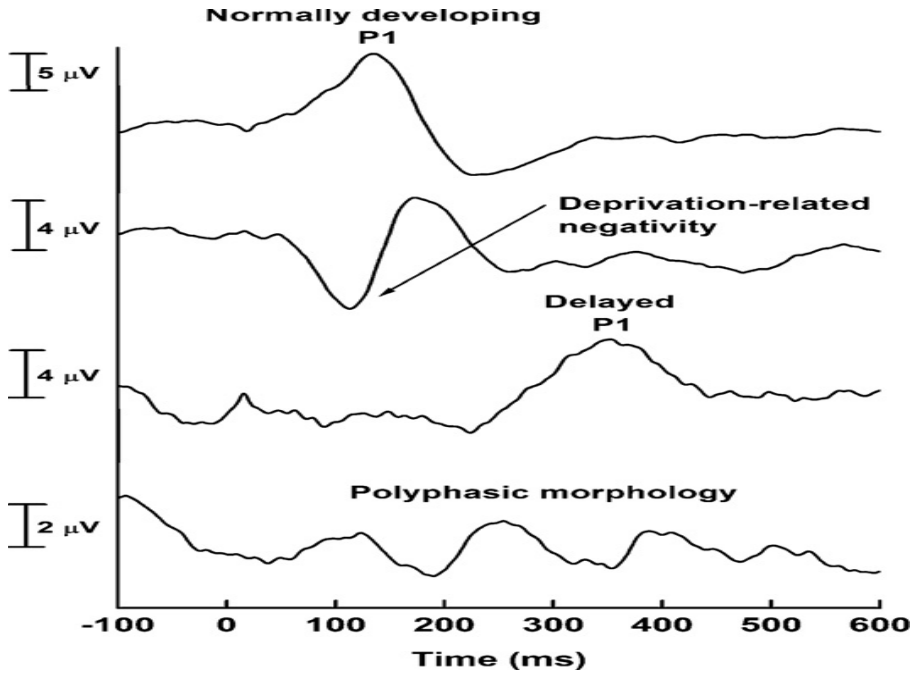

Figure 2.2. Examples of P1 waveforms for a normally developing central auditory pathway (top), an unstimulated central auditory system, (second from top), a partially stimulated central auditory system loss (third from top), a re-organized auditory cortex (bottom) reported by Sharma, Nash, and Dorman (2009).

In case of early-implanted children, there was a large and rapid decrease in P1 latency (approximately 100 ms) within a week of cochlear implant usage. One week after implant use P1 latencies were similar to those of normal hearing newborns. The early negativity preceding P1 seen in children with little auditory experience diminished in amplitude and latency with more auditory experience. Within 6–8 months of age P1

latencies reached normal limits. This outcome is consistent with the previous findings (Sharma et al., 2002).

Late-implanted children show a different pattern of central auditory development than that shown by early implanted children. The early negativity linked with auditory deprivation dominates the waveform at the time of implant activation. In late-implanted children the initial negativity occurs at a shorter latency than the initial negativity in early-implanted children. This outcome suggests that there is some degree of intrinsic development of the central auditory pathways in the absence of stimulation. This outcome is consistent with the findings of (Kral, Hartmann, Tillein, Heid & Klinke, 2002) who showed that middle and long-latency responses appeared early in development in naive congenitally deaf cats even in absence of any auditory inputs, although the further development differed significantly from development in hearing controls.

Correspondingly, in late-implanted children, the polyphasic morphology of the cortical evoked waveform response is atypical and remains atypical for several months following the onset of stimulation. Gradually between the 12^{th} and 18^{th} months after implantation, the morphology of the waveform becomes more typical for late-implanted children. Several investigators have reported that children implanted under ages 3 – 4 years show significantly higher speech perception scores and better language skills than children implanted after 6 - 7 years of age.

P1 maturation and language development:

Early communicative efforts of profoundly hearing impaired or deaf infants parallel normal-hearing infants in several stages; however, deaf infants do not achieve the reduplicated or canonical babbling stage at the same time as normal-hearing infants. Deaf infants do not approach the canonical babbling period until at least 11 months and some do not obtain reduplicated syllables until 2.5 years of age or later. Early intervention appears to positively affect the development of canonical babbling in infants with hearing impairments and increases the likelihood that they will begin the canonical babbling stage at an earlier age than if they remain unaided.

Given that early amplification increases the likelihood of canonical babbling at more nearly normal ages, it is also likely that cochlear implants will increase the likelihood of canonical babbling in young children using cochlear implants.

To examine this possibility, Sharma et al., (2004) examined the relationship in time between the development of central auditory pathways, as measured by P1 latencies, and the development of canonical babbling. The averaged ALLR waveforms recorded during the post stimulation period for children who were cochlear implanted and who were fitted with hearing aid and are given in figure 2.3 & 2.4 respectively.

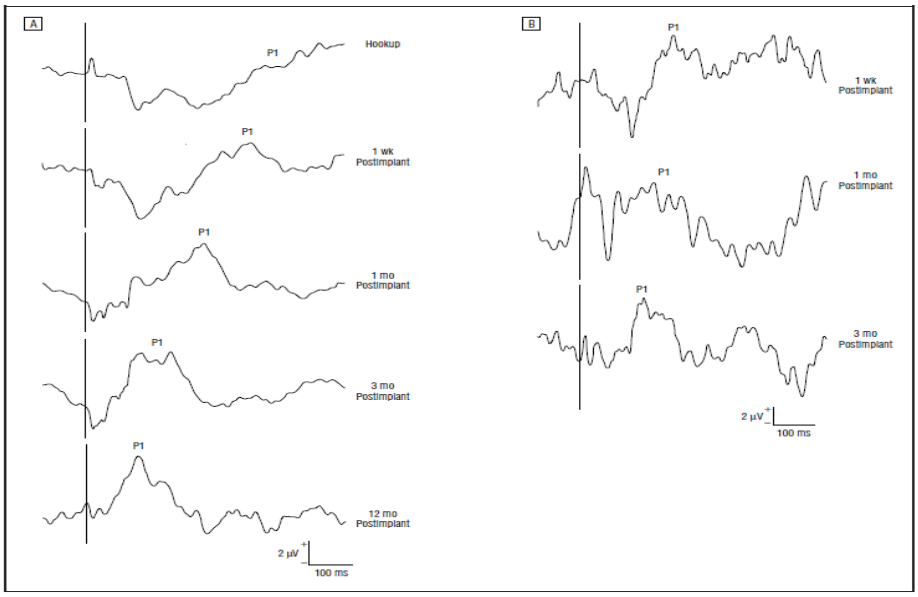

Figure 2.3. Averaged evoked response waveforms are shown for subject 1 (A) and subject 2 (B) at different postimplantation intervals. The P1 response peak is labeled.

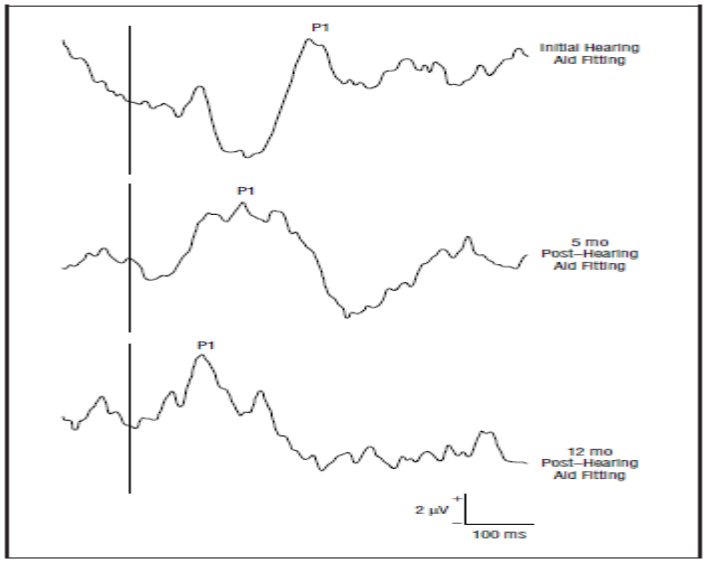

Figure 2.4. Averaged evoked response waveforms are shown for a child with congenital hearing impairment who was first fitted with a hearing aid at age 11 months. The waveforms are shown for different time intervals after hearing aid fitting. The P1 response peak is labeled.

And they reported that the P1 latency and vocalization data for the 2 subjects in this study suggest that the development of P1 response latencies and the development of early communicative behavior may follow a similar developmental trajectory. That is, after relatively minimal experience with the implant, rapid changes in P1 latency were observed in parallel with a major change in vocalization behavior from pre canonical to speech like canonical babbling. Although preliminary, these findings suggest that the development of early communication behaviors following implantation may be promoted by changes in central auditory pathways.

Jang et al., (2010) assessed the P1 latency in Koreans with normal hearing according to the age during development and reported that there was a statistically significant negative correlation between the P1 latency and age and the findings support that the maturation of the central auditory pathways could be measured objectively using the P1 latency. They even reported an increase in scores under infant-toddler meaningful auditory integration scale (IT-MAIS) post cochlear implantation.

ALLR in Auditory neuropathy spectrum disorder (ANSD):

Sharma, Cardon, Henion, and Roland (2011) reported that the P1 CAEP responses may be: (i) A useful indicator of the extent to which neural dys-synchrony disrupts cortical development, (ii) A good predictor of behavioral outcome in children with ANSD.

Children with Auditory neuropathy / Auditory dys-synchrony ***(***AN / AD) fell into three distinct groups based on their P1 CAEP results. These were: (1) children with normal P1 waveform morphology, latency, and amplitude; (2) children with normal

waveform morphology, but delayed P1 latency and decreased amplitude; and (3) children with grossly abnormal waveform morphology, for whom P1 latency and amplitude could not be computed. And they found that 71 % of participants presented with present CAEP (of whom 38 % had normal P1 latencies and 33 % had delayed P1 latencies) and 29 % of participants had absent CAEP responses. That is, the majority, but not all, subjects demonstrated recordable cortical potentials. This finding is consistent with reports of previous studies that have measured CAEPs in pediatric patients with AN/AD (Starr, Picton, Sininger, Hood & Berlin, 1996; Narne & Vanaja, 2008;).

ALLR in children with learning problem (LP)

Gilley, Sharma, Dorman, and Martin (2006) reported that the majority of children with LP had abnormal CAEP responses. These children fell into distinct categories based on the abnormalities in maturational patterns of their CAEP responses.

Bishop and McArthur (2004) described immature CAEP morphologies in response to tone pairs in children with LP. Results from that study also indicate delayed development of the central auditory system in children with LP. In language-impaired (Tonnquist-Uhlen, 1996), and dysphasic children (Korpilahti & Lang, 1994), the N2 was smaller in amplitude and longer in latency than in their healthy peers.

From the review, it is evident that ALLR can indicate language development. These studies also indicate that since the P1 latency and amplitude developments are consistent within the first 3 years of life, while, the N1-P2 components are still in the developmental stages till the early adulthood. P1 biomarkers may provide useful information regarding maturation of the central auditory pathways in children with

hearing impairment. Thus, the above studies indicate that the P1 latency can also monitor the development of central auditory pathways after the child has been fitted with the hearing aid, as a potential biomarker for the development of central auditory pathways (Sharma, Gilley, Martin & Dorman, 2005). However these studies have not correlated between language age and P1 latency. Hence, research is required to study the maturation of P1 latency across various language age groups in normal hearing children and in children who are fitted with hearing aids.

Chapter 3

METHOD

The aim of the present study was to find the relationship between language development and P1 maturation in children with hearing loss and normal hearing children. The study was also aimed at comparing the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children. To accomplish these goals, two groups of participants were taken.

3.1 Participants

A total of 25 participants (12 males & 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

Control group comprised of 12 children (6 males & 6 females) with normal hearing sensitivity in the age range of 1 - 5 years with a mean age of 3.08 years. The participants were further divided into 4 sub groups based on their language age, which are as follows: 1 - 2 years, 2 - 3 years, 3 - 4 years and 4 - 5 years. Three children were considered in each sub group. The chronological and language age of the participants in the control group are given in Table 3.1.

Table 3.1:

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

Selection criteria

Participants who fulfilled the following criteria were included in the control group.

- Pure tone threshold within 15 dB HL at octave frequencies from 250 Hz to 8000 Hz for air conduction and from 250 Hz to 4000 Hz for bone conduction.
- Air bone gap within 10 dB HL.

- Speech Detection Threshold or Speech Recognition Threshold and Speech Identification Scores correlating with the pure tone threshold.
- 'A' type tympanogram with bilateral normal ipsilateral and contralateral acoustic reflex thresholds.
- Presence of Transient Evoked Otoacoustic Emissions (TEOAEs) with a SNR of +6 dB and the response reproducibility and stimulus stability of greater than 80%.
- Presence of Auditory Brainstem Response (ABR) with a wave V at 30 dB nHL.
- No complaint and history of any middle ear pathology in both the ears.
- Age appropriate speech and language development with language age ranging from 1 – 5 years.
- No complaint and history of any observable medical or neurological impairment.

Clinical group

Clinical group comprised of 13 children (6 males & 7 females) with bilateral severe to profound sensorineural hearing loss in the age range of 2.5 - 6 years with a mean age of 4.93 years. The participants were further divided into 4 sub groups based on their language age, which are as follows: 1 - 2 years, 2 - 3 years, 3 - 4 years and 4 - 5 years. Four children in the first sub group and three children were considered in each of the next three sub groups. The chronological and language age of the participants in the clinical group are given in Table 3.2.

Table 3.2:

Chronological and language age of the participants for each sub group in the clinical group.

Sub groups	**Participant**	**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

Selection criteria

Participants who fulfilled the following criteria were included in the clinical group.

- Pure tone threshold varying from severe to profound degree of hearing loss (71 dB HL to 100 dB HL) at octave frequencies from 250 Hz to 8000 Hz for air conduction.
- Air bone gap within 10 dB HL.
- Speech Detection Threshold or Speech Recognition Threshold and Speech Identification Scores correlating with the pure tone threshold.
- 'A' type tympanogram with absence of ipsilateral and contralateral acoustic reflexes, in both the ears indicating normal middle ear function.
- Absence of Transient Evoked Otoacoustic Emissions (TEOAEs) indicating Outer Hair Cell (OHC) dysfunction in both the ears.
- Absence of wave V at 90 dB nHL in the Auditory Brainstem Response (ABR) indicating severe hearing loss.
- No complaint and history of any middle ear pathology in both the ears.
- No complaint and history of any observable medical or neurological impairment.
- Aided audiogram within the speech spectrum with the most appropriate hearing aid fitting.
- Language age ranging from 1 to 5 years; however it was not age appropriate.

3.2 Instrumentation

The following instruments were used for the study

- A calibrated double channel Madsen Orbiter - 922 (version 2) diagnostic audiometer with TDH - 39 headphones and sound field facility, to estimate air conduction thresholds and to establish speech detection threshold or speech recognition threshold and speech identification scores. Radio ear B - 71 bone vibrator to obtain bone conduction thresholds.
- A calibrated Grason Stadler Inc. - Tymp star immittance meter to carry out both tympanometry and acoustic reflexometry.
- A calibrated Otoacoustic Emission system ILO - V6 for measuring Transient Evoked Otoacoustic Emissions (TEOAEs) in order to examine the status of outer hair cells.
- Auditory Brainstem Response (ABR) and Auditory Long Latency Response (ALLR) was recorded using calibrated evoked potential system, Biologic Hearing system (Version 7.0) with ER - 3A insert ear phone and Fostex PM 0.5 MKII loudspeaker respectively.

3.3 Test Materials

- Receptive Expressive Emergent Language Scales 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 group.

- Speech recognition threshold was assessed using the spondee word list developed by Rajashekar (1976) for children in the age range of 4 – 5 years.
- Speech identification test for kannada speaking children, developed by Vandana, (1998) was used to assess speech identification scores for children in the age range of 4 - 5 years.

3.4 Test environment

Testing was carried out in a sound treated room where the ambient noise level was within the specified limits as per ANSI S3.1 (1991).

3.5 Procedure

- ***Stimulus generation***

Stimulus /ba/ spoken by adult male kannada speaker, into an unidirectional microphone was recorded on a PC with 16 bits processor using Adobe Audition 1.5 software, at a sampling rate of 48,000 Hz. The duration of the stimulus was 248.85 ms. Acoustic characteristics and both the waveform and spectrogram of stimulus /ba/ as obtained from reading the sound file using PRAAT software are given in Table 3.3 and in Figure 3.1 respectively.

Table 3.3:

Acoustic characteristics of stimulus /ba/

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

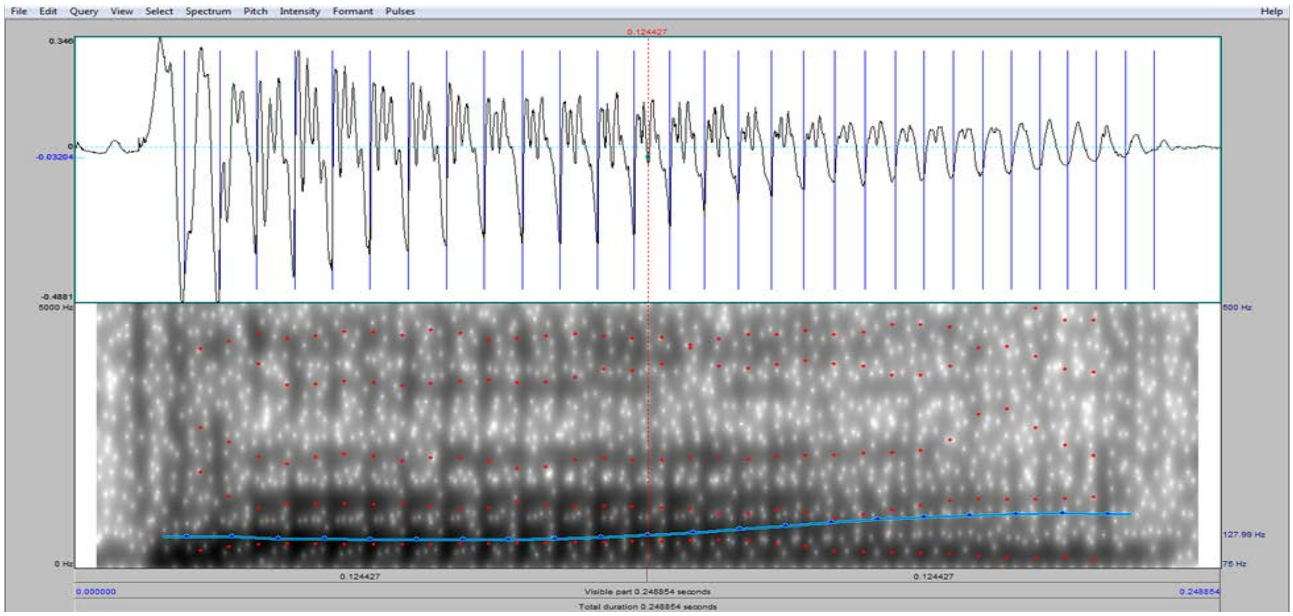

Figure 3.1. The waveform (X – axis represents time in seconds and Y – axis represents amplitude of the sound) and spectrogram of stimulus /ba/ (X – axis represents time in seconds and Y – axis represents frequency in Hertz).

- ***Behavioral Observation Audiometry (BOA):***

  Behavioral Observation Audiometry (BOA) was used to assess the child's responsiveness to sounds, for children in the age range of 1 – 1.6 years. The behavioural responses (minimum response level) of the infants were observed in the free field condition using warble tones from 500 Hz to 4000 Hz separated in octaves and speech stimuli. It was carried out in a double room situation. The infants were seated comfortably on the caregivers lap at a distance of 1 meter from the loud speakers and at an azimuth of 45^0 in the observation room. One clinician was present in the observation room to draw the attention of the infant to the midline and to watch for the unconditioned responses. The other clinician in the test room, presented the test stimuli sequentially with the initiation level being

decided below the level at which the infant is expected to exhibit some kind of auditory behavior, as reported by the parents. The lowest levels of presentation of each of the stimuli, at which the subject exhibited some sort of auditory behavior was noted down.

- ***Visual reinforcement audiometry (VRA):***

VRA given by Liden and Kankkunen (1969) was used to assess pure tone threshold for children in the age range of 1.6 - 2 years. VRA testing was carried out in a sound treated two room situation. The computer used to present stimuli was housed in the tester room while the sound field speaker system was in the patient room. The child was seated comfortably on mother or caregiver's lap in the patient room at a distance of 1 m from the speaker placed at an angle of 45° azimuth. The examiner was seated in front of the child. The reinforcement was provided through the LCD TV monitor placed next to the speaker which delivered the test stimuli. Child's attention was maintained at midline by the examiner using a series of quiet distracters before every stimulus presentation during the training and subsequent testing. A response was defined as a head turn towards the stimulus or the reinforcer (LCD TV monitor) which should occur within 3 seconds of the stimulus presentation. For every response VRA pictures through power point presentation slide show was activated (such that one picture was presented at a time, for one correct response) within the stipulated response interval of 5 seconds. Thresholds were obtained for the warble tones at octave frequencies from 250 Hz to 8000 Hz. Speech stimuli bisyllabic word /papa/ was

presented through loudspeakers and a response was defined as a head turn towards the stimulus or the reinforcer. The minimum intensity at which the child gave head turn response towards the stimulus or the reinforcer 50 % of the time, indicating that the child can just discern the presence of a speech material was considered as the Speech Detection Threshold.

- ***Conditioned play audiometry (CPA):***

CPA was carried out to assess pure tone threshold for children in the age range of 2 – 5 years. The child was conditioned to stack the ring, whenever the child heard the tone. Child was also instructed to pay attention and respond for the softest sound he or she could hear. Thresholds were obtained for the warble tones at octave frequencies from 250 Hz to 8000 Hz. Speech stimuli bisyllabic word /papa/ was presented through headphones and the minimum intensity at which child responded by stacking the ring 50 % of the time, indicating that the child can just discern the presence of a speech material was considered as the Speech Detection Threshold.

- ***Speech audiometry:***

Speech recognition Threshold and Speech Identification Scores were obtained for children above 4 years of age. Spondee words were presented through headphones and the minimum intensity at which the child could recognize 50 % of the speech material from a set of pictures was considered as the Speech recognition Threshold. Speech stimuli (monosyllable list) or monosyllable speech stimuli developed by Vandana, (1998) were presented through headphones

at 40 dB HL above the Speech recognition Threshold. Child was asked to point to the appropriate pictures and the number of stimuli correctly identified was multiplied by four to get the Speech Identification Scores.

- ***Acoustic immittance evaluation:***

  Typmanometry was carried out with 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.

- ***Transient evoked otoacoustic emissions (TEOAEs):***

  TEOAE was measured using the default setting in the instrument (ILO-V6) using non-linear clicks trains presented at 84 ± 3 dB pe SPL of 260 sweeps. Subjects were made to sit on mother or caregiver's lap and silent video was shown to ensure that the children do not move during the testing. Foam tip was properly positioned in the external auditory canal to get a flat stimulus spectrum across the frequency range 500 Hz – 6000 Hz. The presentation mode included a series of four stimuli; three at the same level and of same polarity and the fourth is of three times the level of either of the three and opposite in polarity. This is called nonlinear averaging which was used for artifact reduction during the response acquisition. The responses were considered as emissions based on the reproducibility and signal to noise ratio (SNR). The overall TEOAE amplitude of 6 dB SPL above the noise floor, with the reproducibility of greater than 80% was considered as presence of TEOAE (Dijk & Wit, 1987).

- ***Auditory Brainstem Response (ABR):***

ABR testing was carried out to estimate the threshold for those children in both the control and clinical group in whom reliable pure tone threshold could not be obtained. Electrode sites were cleaned by scrubbing with cotton dipped in skin preparation gel. Silver chloride electrodes were placed on the recording site with a conducting gel. After the electrodes are placed, plasters were used to secure the electrodes in place. It was ensured that the independent electrode impedance was less than 5 KΩ and the inter-electrode impedance was within 2 KΩ. The stimulus and acquisition parameters used to record ABR for threshold estimation is given in Table 3.4 & 3.5 respectively.

Table 3.4:

The stimulus parameters used to record ABR.

Stimulus parameters	Stimuli:	Clicks
	Stimulus duration:	100 µs
	Number of stimuli:	1500
	Intensity:	Variable
	Repetition rate:	30.1/sec
	Stimulus polarity:	Rarefaction
	Transducer:	ER- 3A insert ear phone

Table 3.5:

The acquisition parameters used to record ABR.

Acquisition parameters	Analysis time	15 msec
	Filter settings:	30 Hz - 3000 Hz
	Notch filter:	On
	Artifact rejection:	30 µV
	Number of channels:	Single
	Amplification	1,00,000
	Number of repetitions	2
	Electrode placement	
	Inverting: Test ear mastoid	
	Noninverting: High forehead	
	Ground: non test ear mastoid	

Subjects with presence of wave V at 30 dB nHL were considered as having normal hearing sensitivity and were recruited in the control group. Subjects with absence of wave V at 90 dB n HL 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. The loudspeaker was kept at 45° azimuth and at 1 m distance from the child to present the

stimuli. Child's aided responsiveness to sounds (for warble tones from 500 Hz to 4000 Hz separated in octaves and speech stimuli) was obtained using behavioural observation audiometry (BOA) for children in the age range of 1 – 1.6 years and the lowest levels of presentation of each of the stimuli, at which the subject exhibited some sort of auditory behavior was noted down.

Aided thresholds were obtained using warble tones at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz using either visual reinforcement audiometry (VRA) for children in the age range of 1.6 - 2 years or conditioned play audiometry (CPA) for children in the age range of 2 – 5 years. Aided Speech Detection Threshold was obtained for children below 4 years of age and aided speech identification score was obtained for children above 4 years of age using conditioned play audiometry (CPA).

- ***Language age assessment:***

  Receptive Expressive Emergent Language Scales

Receptive Expressive Emergent Language Scales (REELS) was used to assess Receptive language age (RLA) and Expressive language age (ELA) of all the participants in both the control and clinical group in the age range 0-3 years. REELS test has a total number of 132 test items, under the subtests for receptive language and expressive language. REELS describe language and interactive behaviors that are optimal for infants and toddlers. Questions were asked to the mother, based on the mothers response on to whether the child exhibits that particular behavior or not, based on this scoring was done. The scoring is given in Appendix 1.

If a child scored positive score for all the test items under the last age group, that is, between two years ten months and three years of age, then his or her language age was considered as more than three years and language age assessment was then carried out using Language test in Kannada (KLT).

Language test in Kannada (KLT)

Language test in Kannada (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. The categories under each Part, scoring and the normative table are given in Appendix 2. After obtaining the percentage score the corresponding table was referred depending on whether the child was from urban or rural area and his or her language age was obtained.

- ***Recording of Cortical Auditory Evoked Potential (CAEP):***

Auditory Long Latency Response (ALLR) was recorded for all the participants in both the control and clinical group. Participants were comfortably seated in a reclining chair so that they are relaxed. Care was taken to restrict the movements of head, neck and eyes of the participants during the testing. To keep the participants awake a captioned silent video was shown to them. Older children, above 4 years of age, were advised not to sleep and move during the test. Electrode sites were cleaned by scrubbing with cotton dipped in skin preparation gel. Three Silver chloride disc type electrodes were placed on the recording site with a conducting gel. After the electrodes were placed, plasters were used to secure the electrodes in place. It was ensured that the independent electrode

impedance was less than 5 KΩ and the inter-electrode impedance was within 2 KΩ. The stimulus and acquisition parameters used to record ALLR are given in Table 3.6 & 3.7 respectively.

Table 3.6:

Shows the stimulus parameters used to record ALLR.

Stimulus parameters	Stimuli:	Natural speech stimuli /ba/
	Stimulus duration:	248.85 ms
	Number of stimuli:	300
	Intensity:	65 dB SPL
	Repetition rate:	30.1/sec
	Stimulus polarity:	Alternating
	Transducer:	Loudspeaker

Table 3.7:

Shows the acquisition parameters used to record ALLR.

Acquisition parameters	Analysis time:	500 ms
	Pre stimulus	100 ms
	Filter settings:	1 Hz – 30 Hz
	Notch filter:	on
	Number of channels:	Single
	Recording	Binaural
	Number of repetitions	2
	Amplification	25,000
	Electrode placement	
	Inverting: Nape of the neck	
	Noninverting: Vertex	
	Ground: Forehead	

ALLR was recorded at 65 dB SPL as Tremblay and Tolin (2009) have reported that there was no main effect of tone level on P1 latency in normal hearing adults across the two signal levels that is, at 60 and 75 dB SPL.

Stimulus /ba/ was presented through the loud speaker kept at 0° azimuth and at a distance of 1m from the subject and at the subject's ear level. 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.

Waveform analysis

- The stored waveforms were recalled and analyzed later. P1, N1, P2, and N2 components were identified and marked visually by three experienced audiologist. 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 (SD) 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.
- 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.

Chapter 4

RESULTS AND DISCUSSIONS

The aim of the present study was to find the relationship between language development and P1 maturation in normal hearing children and in children with hearing impairment. Also the aim of the study was to compare the relationship between language development and P1 maturation in normal hearing children and children with hearing impairment. To accomplish these aims, ALLR was recorded using natural stimulus /ba/. Unaided ALLR recordings were obtained from the participants in the control group, while, both unaided and aided ALLR recordings were obtained from the clinical group. The stored ALLR waveforms were later analyzed; P1 was identified and marked visually. The mean and SD were calculated for the P1 latency for both the control and clinical group. The other ALLR components were also marked and are discussed further descriptively.

To analyze the data, following statistical analysis were carried out using SPSS version 17.0 software. Since, the sample size was small; the data was analyzed using the non-parametric statistical test tool.

- Descriptive statistics to obtain the mean and the standard deviation (SD) for P1 latency in both the control and clinical group.
- Kruskal-Wallis Test was carried out as the sample size was small and unequal in the control and clinical group (control group: n-12; clinical group: n-13) to study the effect of language age on P1 latency in the control and clinical group.

- Mann-Whitney Test was carried out as the sample size was small and there was random selection of subjects into their respective group to compare the relationship between language development and P1 maturation in normal hearing children and children with hearing impairment.

The results obtained from different statistical analysis are given below for both the control and clinical group.

4.1 ALLR results in the control and clinical group

4.1.1 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 (SD) of P1 latency for the control group across all the language age groups was calculated. The results are outlined in Table 4.1.

Table 4.1:

Depicts the mean and SD of P1 latency for the Control group.

Parameter	Language age (In years)	Control group	
		Mean (n=3 in each language age group)	SD
P1 latency (In msec)	1-2	315.92	35.77
	2-3	214.95	54.22
	3-4	174.26	46.83
	4-5	121.48	22.88

It is evident from the Table 4.1 that the mean P1 latency decreases 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. This is depicted graphically in the figure 4.1.

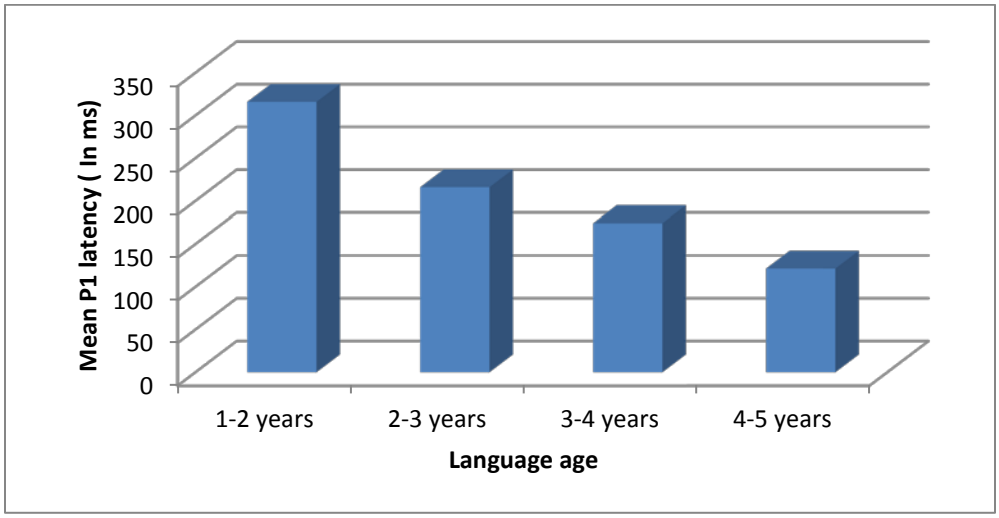

Figure 4.1. Depicts the mean P1 latency for the control group across language age.

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 4.1 and figure 4.1 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., 2002b; Sharma et al., 2005). However, all these studies were carried out on a large sample.

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 – 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 4.2.

Table 4.2.

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	**N1**	**P2**	**N2**
1 – 2 years	Subject 1	1.6 - 2	274.71	299.69	--	--
	Subject 2	1 - 1.6	336.13	410.77	--	--
	Subject 3	1 - 1.6	336.94	367.27	--	--
2 – 3 years	Subject 1	2.6 - 3	176.82	219.50	--	--
	Subject 2	2 - 2.6	274.76	296.62	--	--
	Subject 3	2.6 - 3	193.29	269.29	--	--
3 – 4 years	Subject 1	3.6 - 4	120.46	281.96	351.63	405.46
	Subject 2	3 - 3.6	205.90	348.40	--	--
	Subject 3	3.6 - 4	196.44	275.61	300.94	326.27
4 – 5 years	Subject 1	4 - 4.6	133.06	176.78	218.42	257.98
	Subject 2	4.6 - 5	95.13	161.63	237.10	257
	Subject 3	4 - 4.6	136.27	202.77	228.11	266.11

ALLR results for children with Language age of 1-2 years in the control group

As it can be seen from table 4.2 the ALLR waveform of all the three subjects in the control group with the language age ranging from 1 – 2 years revealed a positive wave P1 followed by a robust negative wave N1. The ALLR waveform of subject 1 in the control group with the language age 1.6 – 2 years is given in figure 4.2.

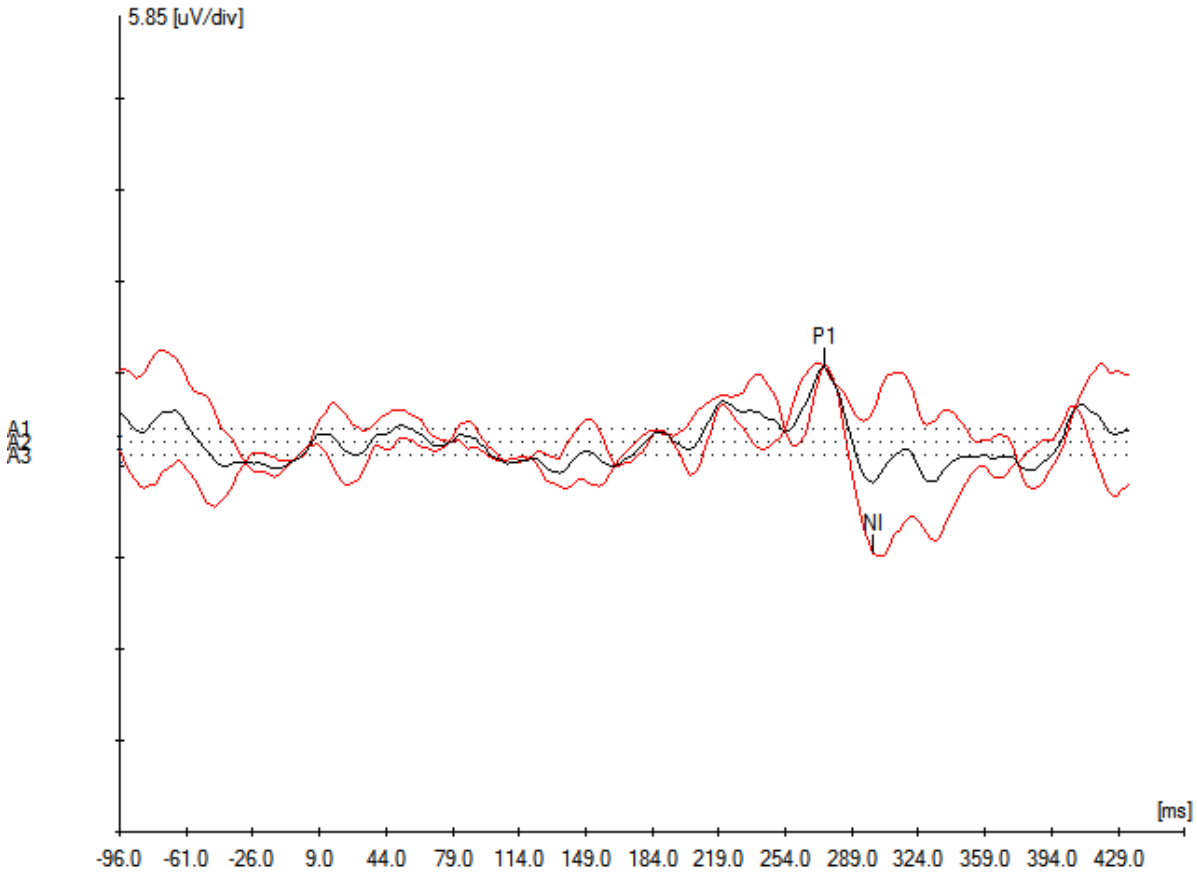

Figure 4.2. Depicts the ALLR waveform of subject 1 in the control group with the language age 1.6 – 2 years.

It is also evident from table 4.2 that the P1 latency of subject 1 is much shorter compared to subject 2 & 3. Also it was noticed that the ALLR waveform of subject 2 and

3 was similar in terms of latency and morphology. The possible reason could be the difference that existed between the subjects in terms of their language age. As it can be noticed from table 4.2, the language age of subject 1 was 1.6 - 2 years and for subject 2 and 3 it was 1-1.6 years. Thus, the difference in language age among the subjects would have resulted in shorter P1 latency in subject 1. Also the ALLR waveform of all the subjects had more negativity following P1 wave. These findings in the present study correlate well with the previous studies (Kushnerenko, 2003; Sharma et al., 1997; Sharma et al., 2002b).

Kushnerenko (2003) reported that during the second half of the first year of life to one year of age, ALLR was characterized by the strong growth of the negative amplitudes (N250 & N450 peaks). Sharma et al., (1997), Sharma et al., (2002b) 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.

ALLR results for children with Language age of 2-3 years in the control group

As it can be seen from table 4.2 the ALLR waveform of all the three subjects in the control group with the language age ranging from 2 – 3 years revealed a positive wave P1 wave followed by a negative wave N1. It is also evident that both P1 and N1 latencies are shorter compared to the subjects in the language age group of 1 – 2 years. The ALLR waveform of subject 1 in the control group with the language age 2.6 – 3 years is given in Figure 4.3.

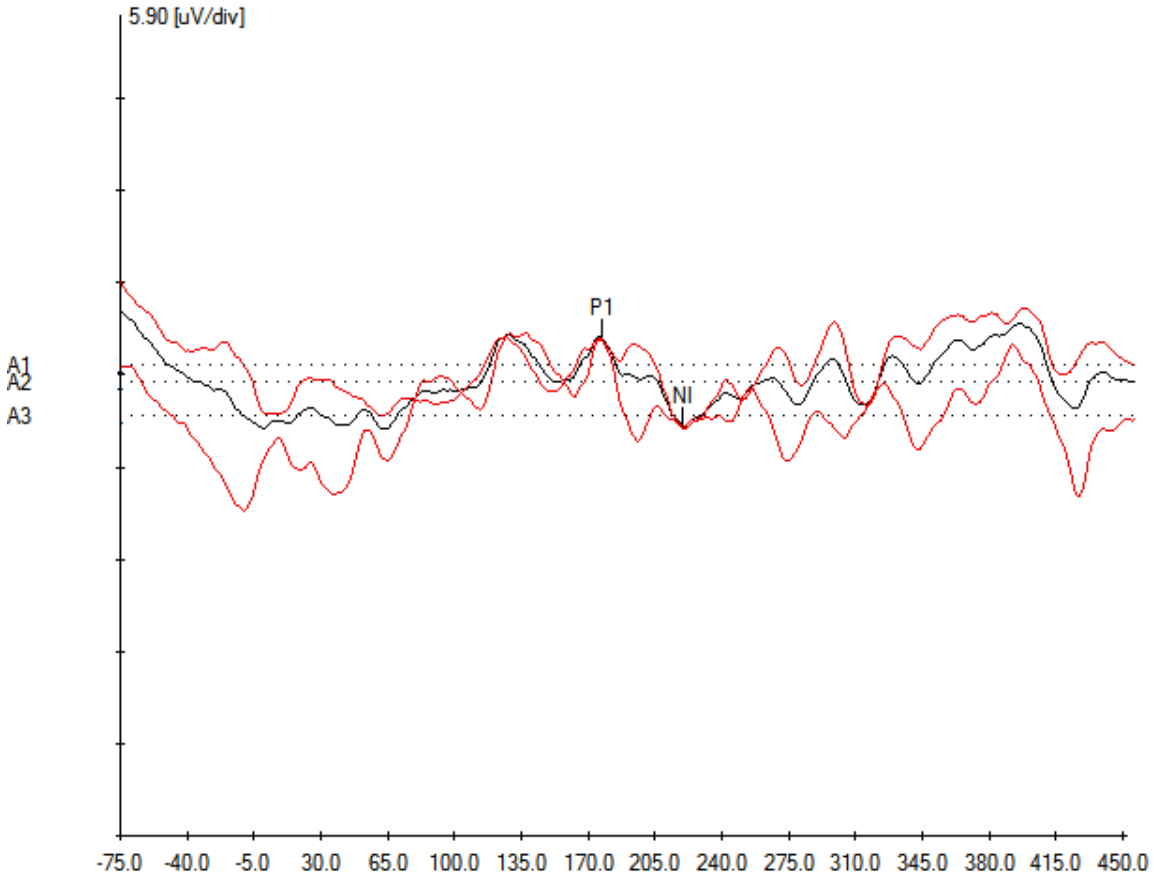

Figure 4.3. Depicts the ALLR waveform of subject 1 in the control group with the language age 2.6 – 3 years.

It is also evident from table 4.2 that the P1 latency of subject 1 and 3 was shorter compared to the P1 latency of subject 2. The possible reason could be the difference in language age that existed between the subjects. The language age of subject 1 and 3 was 2.6 - 3 years and for subject 2 it was 2 – 2.6 years. Thus, the difference in language age among the subjects would have resulted in a decrease in P1 latency in subject 1 and 3. These findings in the present study are in consonance with the previous studies (Courchesne, 1990; Pasman et al., 1991; Pasman et al., 1999; Sharma et al., 2002a).

Courchesne (1990) reported that for children as young as 2–4 years of age the waveform is dominated by a large positivity (P1) followed by a broad negativity (N1b). Pasman et al., (1991), Pasman et al., (1999), Sharma et al., (2002a) recorded CAEPs in young children aged 0 to 4 years and obtained latencies approximately in the range of 150 ms - 200 ms for P1 and 300 ms - 350 ms for the late negativity after P1.

ALLR results for children with Language age of 3 - 4 years in the control group

As it can be seen from table 4.2 the ALLR waveform of all the three subjects in the control group with the language age ranging from 3 – 4 years revealed a positive wave P1 wave followed by a negative wave N1. Subject 1 and 3 had all the P1, N1, P2, and N2 components and P1 and N1 latencies are shorter compared to the subjects in the lower language age groups. The ALLR waveform of subject 1 in the control group with the language age 3.6 – 4 years is given in figure 4.4.

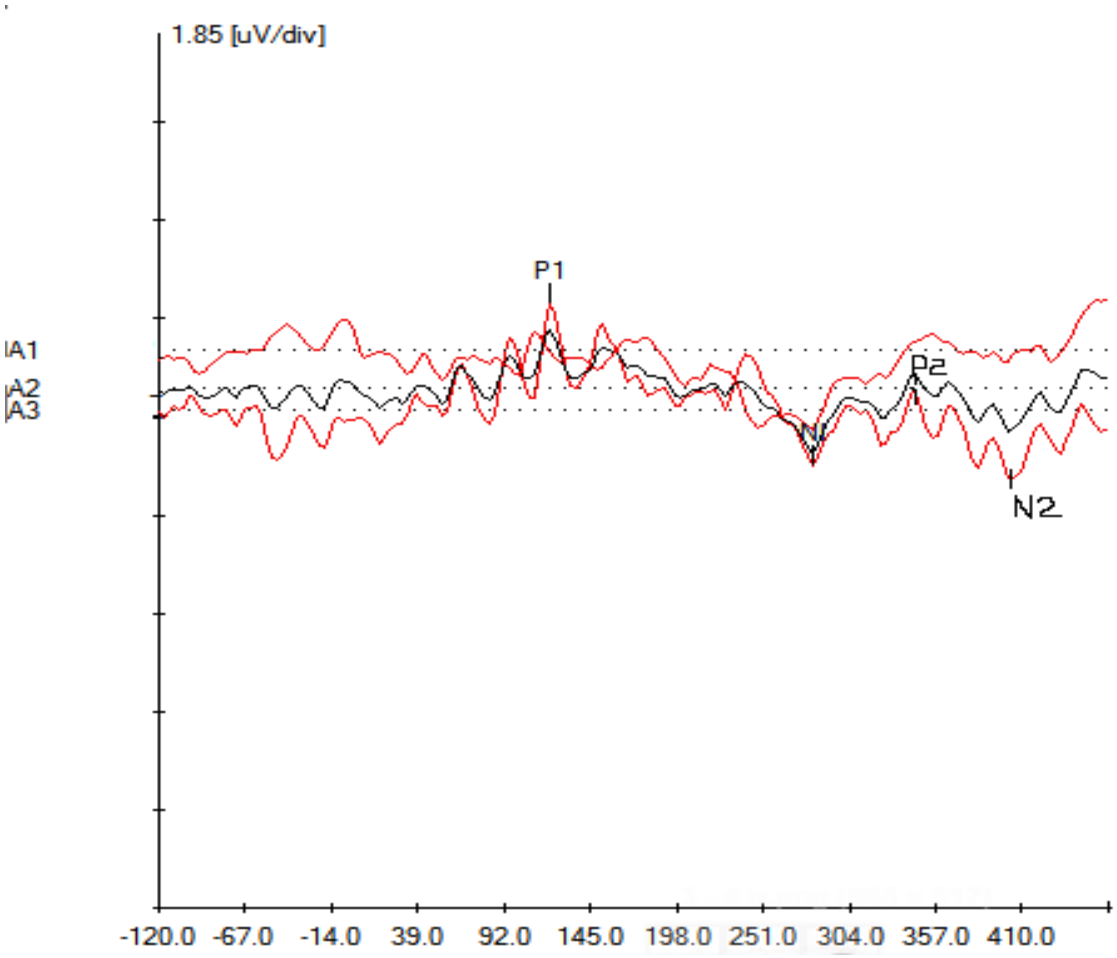

Figure 4.4. Depicts the ALLR waveform of subject 1 in the control group with the language age 3.6 – 4 years.

It is also evident from table 4.2 that the P1 latency of subject 1 and 3 was shorter compared to the P1 latency of subject 2. The possible reason could be the difference that existed in terms of language age between the subjects. The language age of subject 1 and 3 was 3.6 - 4 years and for subject 2 it was 3 – 3.6 years. Thus, the difference in language age among the subjects would have resulted in a decrease in P1 latency and emergence of later peaks namely, P2 and N2 in subject 1 and 3. These findings in the present study are in consonance with the previous studies (Sharma et al., 2005; Sharma et al., 1997; Sharma et al., 2002a).

Sharma et al., (2005) investigated CAEP in 50 normal hearing children aged 3- 12 years, using natural speech syllable /uh/ of 23 ms duration, that was presented through loudspeaker at 70 dB SPL with sequentially decreasing ISIs (offset-to-onset) of 2000, 1000, 560, and 360 ms. They reported that the CAEP waveforms in the two age groups (3 – 4 & 5 – 6 years) showed a robust positivity (P1) at all 4 presentations and many children did have clear N1/P2 responses.

Sharma et al., (1997), Sharma et al., (2002a) reported that the mean P1 latency decreased rapidly over the first 2-3 years to approximately 125 msec at 3 years old and then it gradually decreased into the second decade of life. The possible reason for the difference in P1 latency between this study and the present study (P1 latency varied from 120 ms to 205 ms) could be that, in the present study natural stimulus /ba/ of duration 248.85 ms was used, which is of longer duration compared to synthesized /ba/ stimulus of 90 ms duration that was used in their study. Also, in the present study stimuli was presented at 65 dB SPL through loudspeakers placed at 0° azimuth, whereas in their study stimuli was presented at an higher intensity of 70 dB SPL through loudspeakers placed at 45 ° azimuth (towards the aided ear).

ALLR results for children with Language age of 4 - 5 years in the control group

As it can be seen from table 4.2 the ALLR waveform of all the three subjects in the control group with the language age ranging from 4 – 5 years had all the P1, N1, P2, and N2 components. It is also evident that both P1 and N1 latencies are shorter compared to the subjects in the lower language age groups. The ALLR waveform of subject 1 in the control group with the language age 4 - 4.6 years is given in figure 4.5.

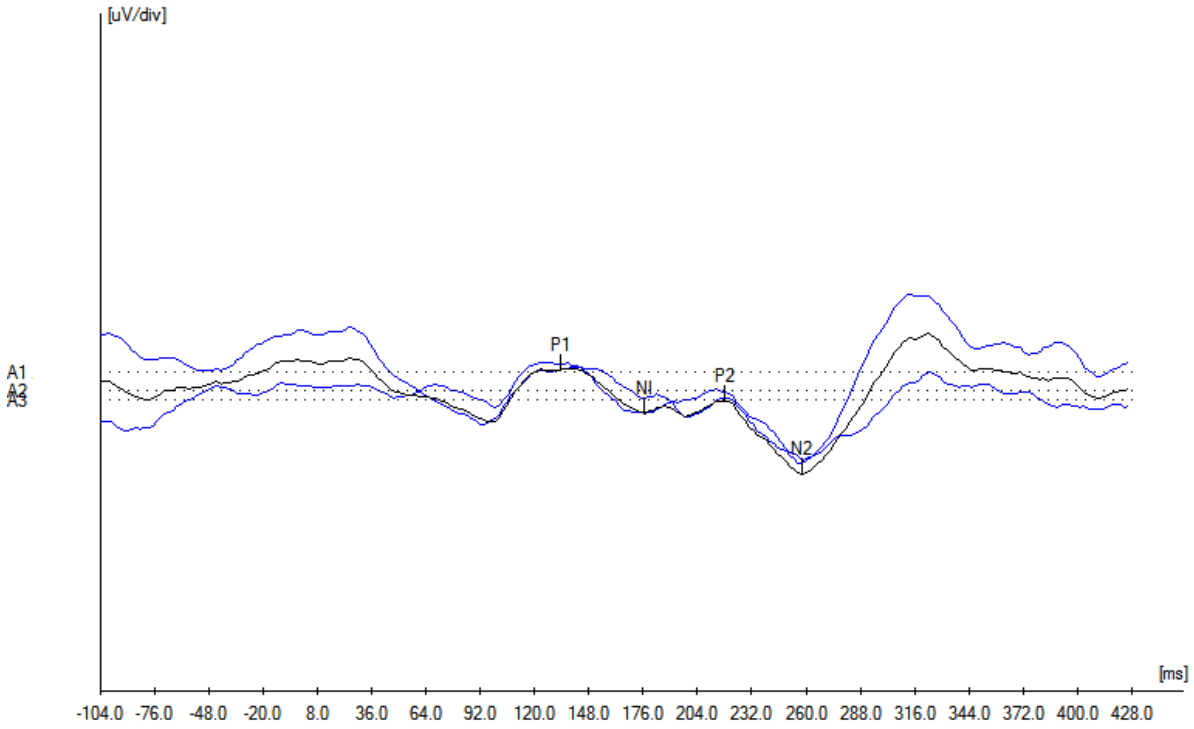

Figure 4.5. Depicts the ALLR waveform of subject 1 in the control group with the language age 4 - 4.6 years.

It is also evident from table 4.2 that the P1 latency of subject 1 and 3 was longer compared to subject 2. The possible reason could be the difference that existed between the subjects in terms of their language age. The language age of subject 1 and 3 was 4 – 4.6 years and for subject 2 it was 4.6 - 5 years. Thus, the difference in language age among the subjects would have resulted in a decrease in P1 and N1 latency in subject 2.

These findings are consistent with the previous study by Ponton et al., (2000), who reported that the latency of P1 is around 80 ms -110 ms in 5-6 year old normal hearing children.

4.1.2 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 dB SPL) that was given was not sufficient enough to stimulate the regions responsible for the generation of ALLR response. The mean and standard deviation (SD) 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 4.3.

Table 4.3

Depicts the mean and SD of P1 latency for all the subjects across language ages for the clinical group.

Parameter	Language age (In years)	Clinical group	
		Mean	**SD**
Aided P1 latency (In msec)	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

As it is evident from table 4.3, the SD is not provided for the first sub group with language age of 1 – 2 years because out of four children tested in this language age group, only one child had ALLR. It is also evident from the table 4.3, that the mean latency decreases as the language age increased from 1 to 5 years in the clinical group. This is depicted graphically in the figure 4.6.

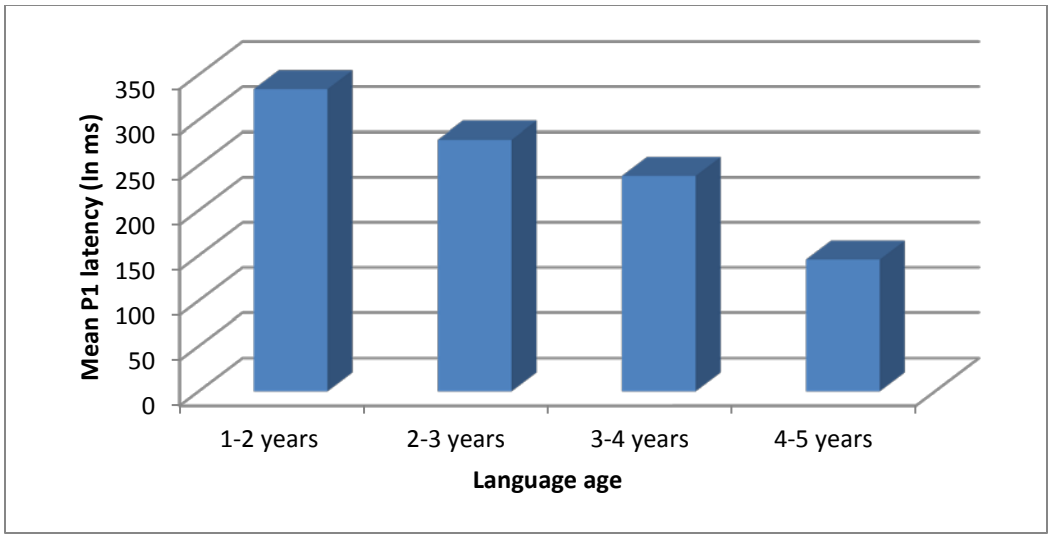

Figure 4.6. Depicts the mean P1 latency for the clinical group across language age.

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 4.3 and figure 4.6 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.

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 duration of 4 month 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 4.4.

Table 4.4:

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	**N1**	**P2**	**N2**
1 – 2 years	Subject 1	1.6 - 2	334.13	413.77	--	--
	Subject 2	1 - 1.6	--	--	--	--
	Subject 3	1 - 1.6	--	--	--	--
	Subject 4	1 - 1.6	--	--	--	--
2 – 3 years	Subject 1	2 - 2.6	299.65	339.20	--	--
	Subject 2	2.6 - 3	272.60	299.66	--	--
	Subject 3	2.6 - 3	262.96	405.46	--	--
3 – 4 years	Subject 1	3 - 3.6	275.61	319.94	--	--
	Subject 2	3 - 3.6	242.40	277.79	297.53	319.39
	Subject 3	3.6 - 4	197.44	276.61	--	--
4 – 5 years	Subject 1	4.6 - 5	107.99	191.27	--	--
	Subject 2	4 - 4.6	152.11	171.11	297.77	319.94
	Subject 3	4 - 4.6	176.82	219.50	254.90	275.75

ALLR results for children with Language age of 1-2 years in the clinical group

As it can be seen from table 4.4, the ALLR (P1 & N1 only) was present for only subject 1 out of four children tested in the language age group of 1 – 2 years. The P1 latency for the subject 1 was observed at around 334.13 ms. The ALLR waveform of subject 1 in the clinical group with the language age 1.6 - 2 years is given in figure 4.7.

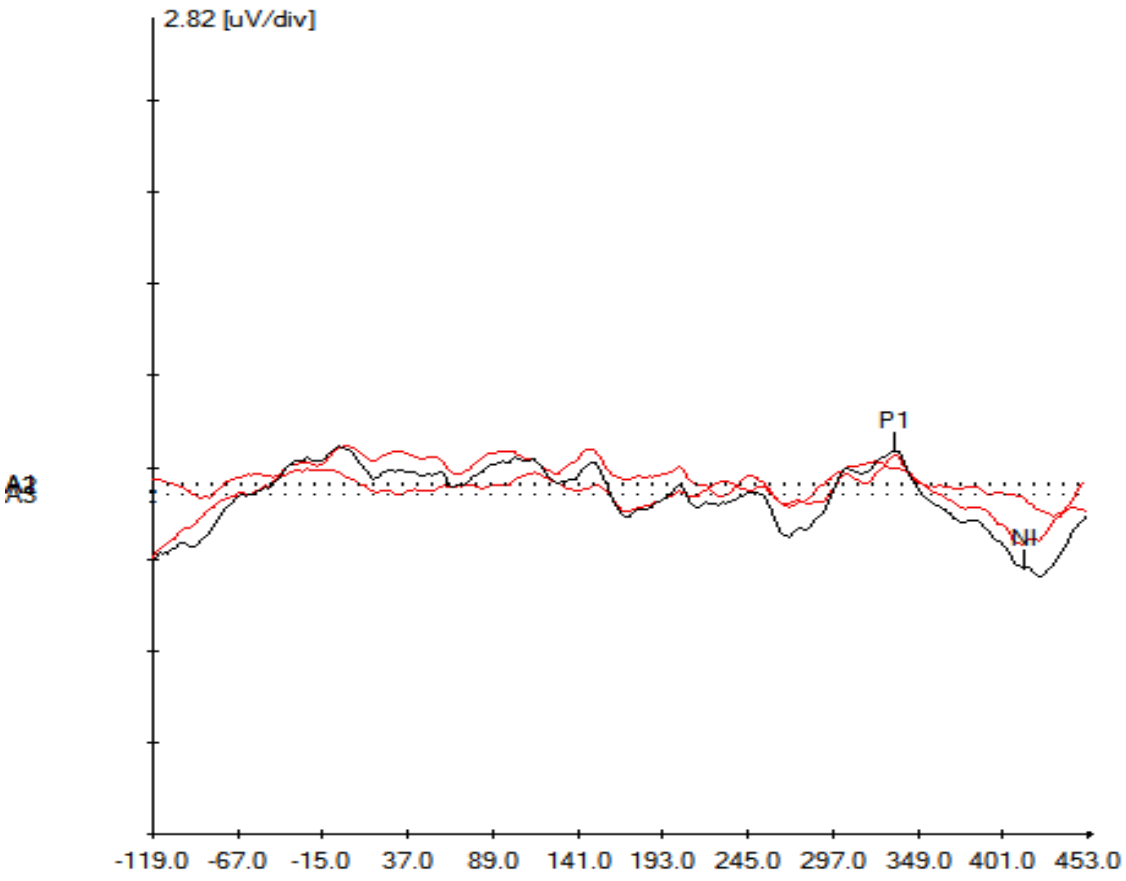

Figure 4.7. Depicts the ALLR waveform of subject 1 in the clinical group with the language age1.6 - 2 years.

The possible reason for the absence of the ALLR in the other three subjects could be that the age of identification and rehabilitation was beyond 3 years of age for subject 2, 3, and 4. While the age of identification and rehabilitation was within 3 years of age

for subject 1. This finding is in consistent with the previous studies (Sharma et al., 2004; Sharma et al., 2002a).

Sharma et al., (2004) examined the development of P1 latency in a child with congenital moderate to severe sensorineural hearing loss who was fitted with hearing aid at the age of 11 months. P1 latencies were outside normal limits at the time of initial fitting with the hearing aid and decreased to within normal limits 5 months after hearing aid use. The P1 latencies continued to develop normally with 12 months of hearing aid use. This change in response morphology was similar to that found for children who were fitted with cochlear implants.

Sharma et al., (2002a) examined the development of P1 response latencies in 104 congenitally deaf children who had been fit with cochlear implants at ages ranging from 1.3 yr to 17.5 yr and three congenitally deaf adults. The results revealed that implanted children, with the longest period of auditory deprivation before implantation by about 7 or more years had abnormal cortical response latencies to speech. Children implanted within 3.5 years evidenced age-appropriate latency responses within 6 months after the onset of electrical stimulation.

ALLR results for children with Language age of 2 - 3 years in the clinical group

As it can be seen from table 4.4 the ALLR waveform of all the three subjects in the clinical group with the language age ranging from 2 - 3 years revealed a positive wave P1 wave followed by a negative wave N1. The ALLR waveform of subject 2 in the clinical group with the language age 2.6 – 3 years is given in figure 4.8.

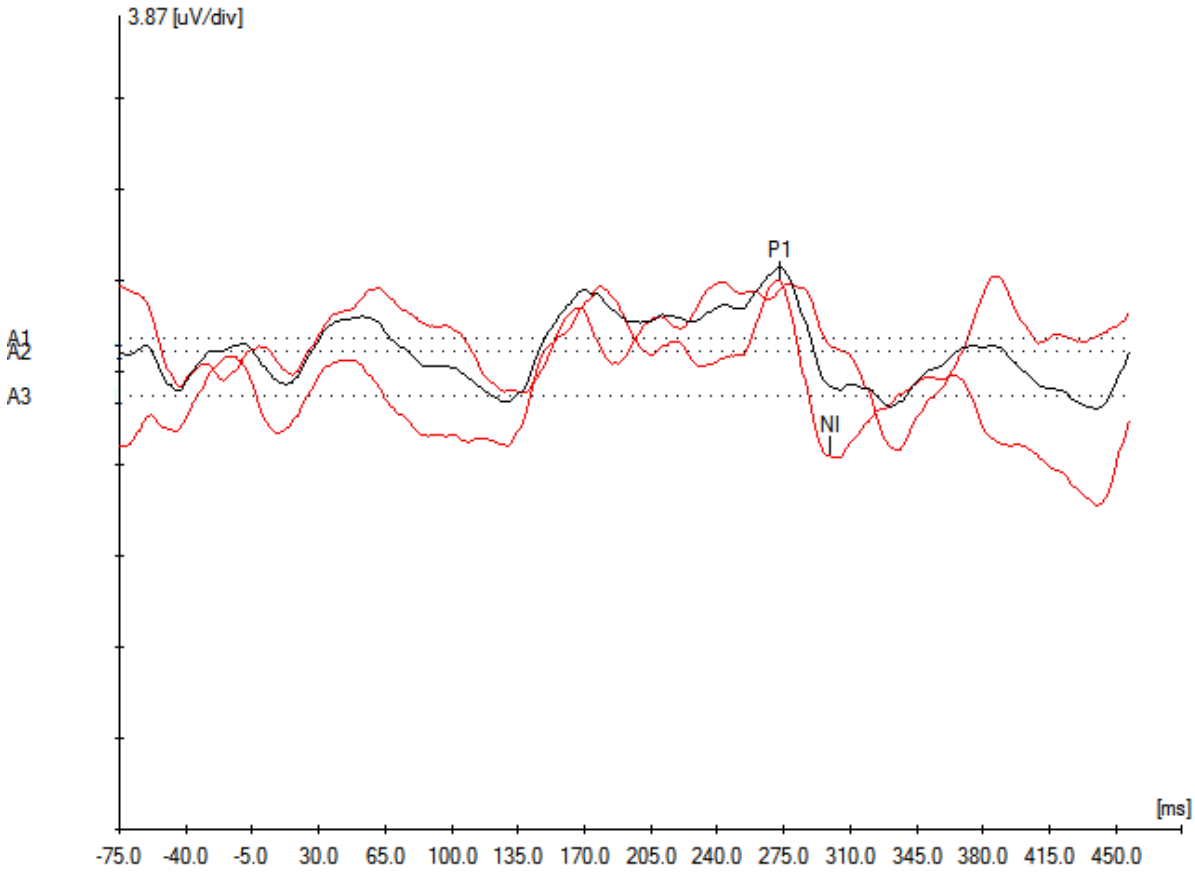

Figure 4.8. Depicts the ALLR waveform of subject 2 in the clinical group with the language age 2.6 – 3 years.

It is also evident that the P1 latency of subject 2 & 3 is evidently less than the P1 latency of subject 1 of the same language age group 2-3 years. The possible reason could be that the language age of subject 2 & 3 was 2.6 - 3 years and for subject 1 it was 2 - 2.6 years. It can also be noted that the P1 and N1 latency have drastically reduced compared to child with language age of 1-2 years. This finding is in accordance with the previous studies (Sharma et al., 2004; Ponton et al., 1996; Dorman et al., 2007).

Sharma et al., (2004) reported that the rapid changes in P1 latency and morphology in the post stimulation period occur in children fitted with hearing aid as

well as those who are fitted with cochlear implant. Ponton et al., (1996) reported that the P1 latency decreases in children who were fitted with cochlear implant. Dorman et al., (2007) reported similar findings in 245 congenitally deaf children who were fitted with cochlear implants.

ALLR results for children with language age of 3 - 4 years in the clinical group

As it can be seen from table 4.4 the ALLR waveform of all the three subjects in the clinical group with the language age ranging from 3 - 4 years revealed a positive wave P1 followed by a negative wave N1. The ALLR waveform of subject 3 in the clinical group with the language age 3.6 – 4 years is given in figure 4.9.

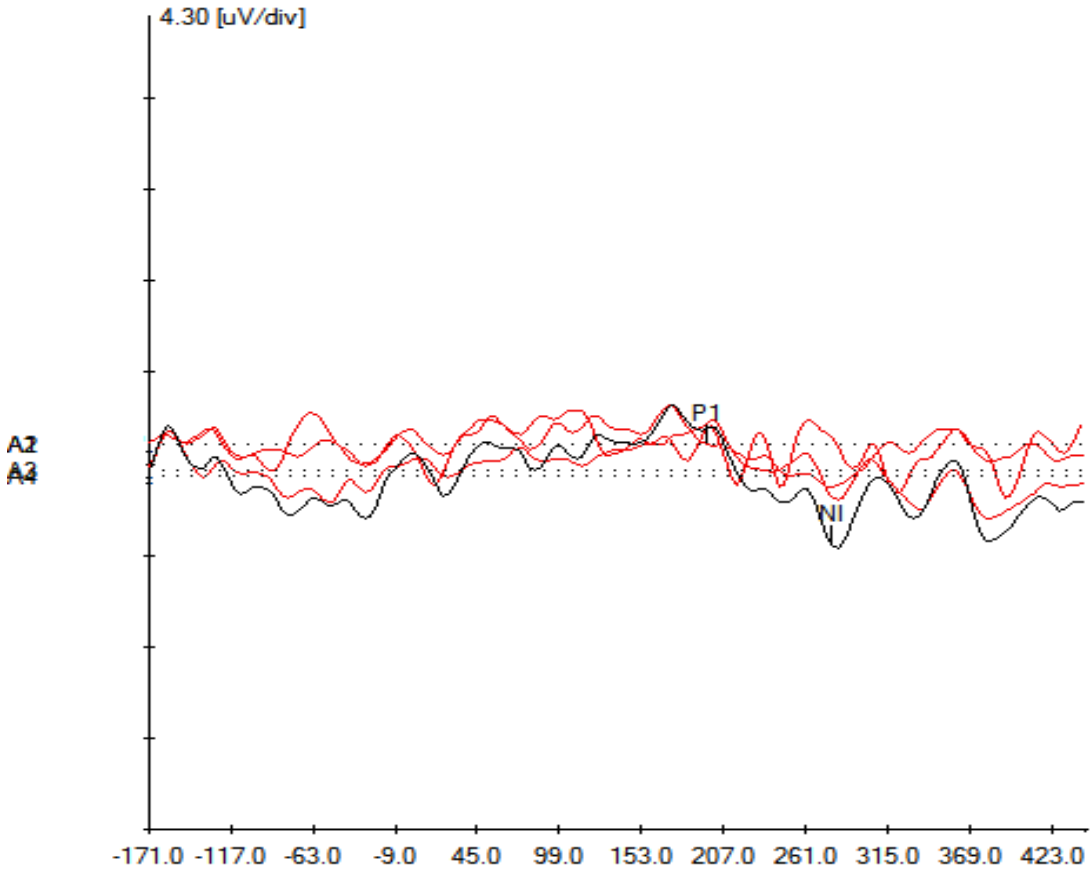

Figure 4.9. Depicts the ALLR waveform of subject 3 in the clinical group with the language age 3.6 - 4 years.

It is also evident from table 4.4 that Subject 2 had all the P1, N1, P2, and N2 components. Also P1 latency of subject 1 & 2 is evidently longer than the P1 latency of subject 3 of the same language age group. The possible reason could be that the language age of subject 1 & 2 was 3 – 3.6 years, and for subject 3 it was 3.6 - 4 years. It can also be noted that the P1 and N1 latency have drastically reduced compared to children in the lower language age groups. These findings of the present study are in consonance with the previous study by Dorman et al., (2005). They have reported a decrease in P1 latency

and improvements in speech and language in children fitted with hearing aid as well as children fitted with cochlear implants.

However, Jang et al., (2010) investigated aided ALLR in 10 children aged 3.3 – 15.5 years in the post cochlear implant stimulation and the results revealed that the P1 latency was between 115 ms – 135 ms in 3 children in the age range of 3 – 4 years of age. The possible reason for the difference in P1 latency between this study and the present study could that in the present study (latency varied from 197 ms to 275 ms) natural stimulus /ba/ of duration 248.85 ms was used, which is of longer duration compared to synthesized /ba/ stimulus of 90 ms duration that was used in Jang, et al., (2010) study. Also, in the present study stimuli was presented at 65 dB SPL through loudspeakers placed at 0° azimuth, whereas in their study stimuli was presented at an higher intensity of 70 dB SPL through loudspeakers placed at 45 ° azimuth (towards the aided ear).

ALLR results for children with language age of 4 - 5 years in the clinical group

As it can be seen from table 4.4 the ALLR waveform of all the three subjects in the clinical group with the language age ranging from 4 - 5 years revealed a positive wave P1 followed by a negative wave N1. The ALLR waveform of subject 1 in the clinical group with the language age 4.6 – 5 years is given in figure 4.10.

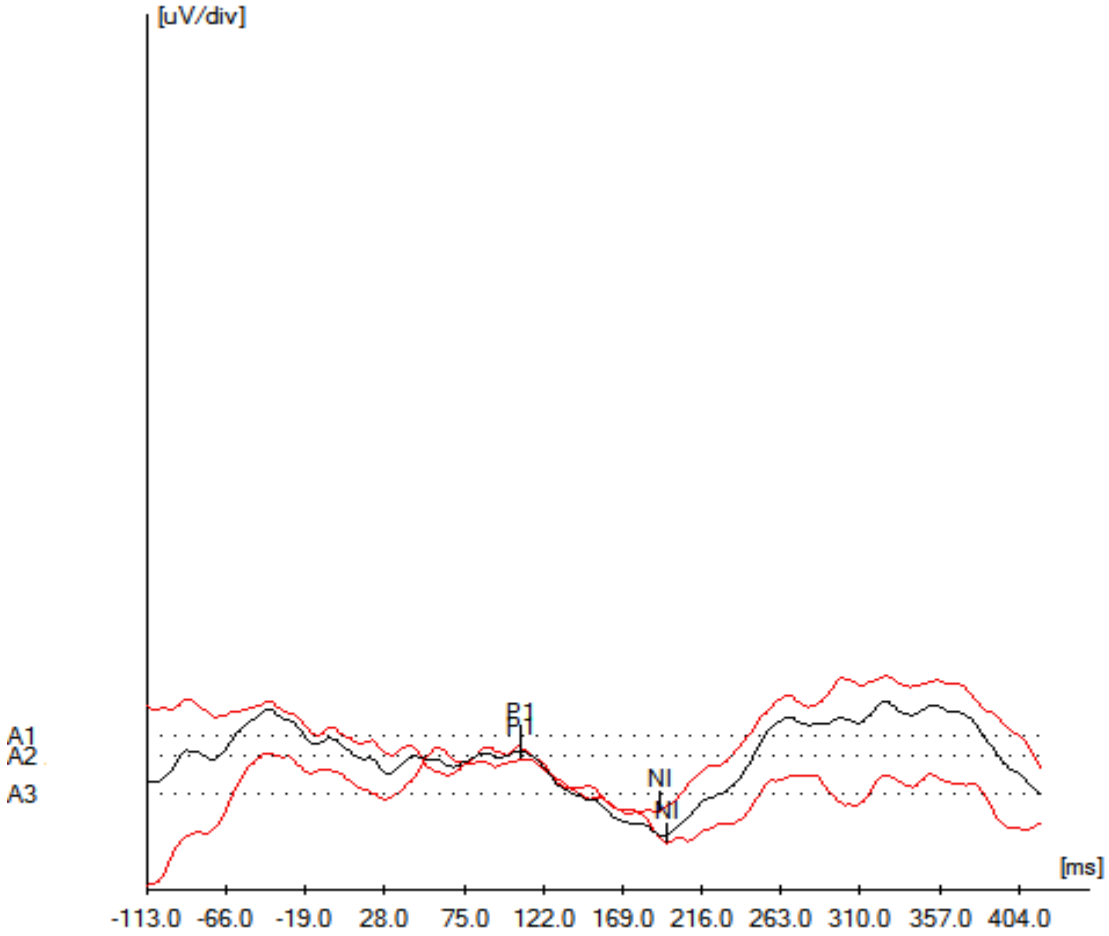

Figure 4.10. Depicts the ALLR waveform of subject 1 in the clinical group with the language age 4.6 - 5 years.

It is also evident from table 4.4 that Subject 2 & 3 had all the P1, N1, P2, and N2 components. Also P1 latency of subject 1 was shorter compared to the P1 latency of subject 2 and 3 of the same language age group 4 - 5 years. The possible reason could be that the language age of subject 1 was 4.6 - 5 years, whereas, for subject 2 and 3 it was 4 – 4.6 years. It can also be noted that the P1 and N1 latency have drastically reduced compared to children in lower language age groups. This finding is in consonance with the previous studies (Jang et al., 2010; Sharma et al., 2004).

Jang et al., (2010) investigated aided ALLR in 10 children aged 3.3 – 15.5 years in the post cochlear implant stimulation and the results revealed that the P1 latency decreased with increased period of auditory stimulation. Sharma et al., (2004) reported a decrease in P1 latency and improvements in speech and language in children fitted with hearing aid as well as children fitted with cochlear implants.

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

The mean and SD of P1 latency for both the control and clinical group across language ages are given in table 4.5.

Table 4.5.

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

Parameter	Language age (In years)	Control group		Clinical group	
		Mean	SD	Mean	SD
P1 latency (In msec)	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

As it is evident from table 4.5, 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 but this 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 finding are consistent with previous studies (Ponton et al., 1996; Sharma et al., 2002a; Sharma et al., 2002b).

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), Sharma et al., (2002b) 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 et al., 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 the P1 CAEP 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 et al., (2008) analyzed CAEP 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, Craig, Raudenbush, Heavner & Zwolan, 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.

Chapter 5

SUMMARY AND CONCLUSION

Central auditory maturation is a constant process from pre natal period to puberty. Development of the peripheral auditory system (ear and auditory brain-stem) 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 et al., 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.

Auditory evoked potentials (AEPs) using speech stimuli especially the P1 latency of the auditory long latency response (ALLR) can give more accurate information and it reflects the developmental status of the central auditory system maturation through changes in their latency, amplitude and morphology (Pang & Taylor, 2000; Gilley et al., 2005; Dorman et al., 2007; Jang et al., 2010). However, there is a dearth of information regarding the relationship between P1 latency and language development in children fitted with hearing aid. Hence, the present study aimed:

- ✓ To know the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children.
- ✓ To compare the relationship between language development and P1 maturation in children with hearing impairment and normal hearing children.

To arrive at the objectives, 12 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 – 5 years. Subjects in both the control and clinical group were tested on a test battery including behavioral observation audiometry (BOA) to obtain the child's responsiveness to sounds for children in the age range 1 – 1.6 years. Participants between 1.6 – 2 years and 2 – 5 years of age were tested using visual reinforce audiometry (VRA) and conditioned play audiometry (CPA) respectively to obtain child's pure tone threshold. Speech detection threshold was obtained from children in the age range of 1.6 – 2 years using visual reinforce audiometry (VRA) and for children between 2 – 4 years of age using conditioned play audiometry (CPA). Speech recognition threshold and speech identification scores were obtained using closed set task for children in the age range of 4 – 5 years. All the participants were tested using immittance and reflexometry, Transient evoked oto-acoustic emissions (TEOAEs), auditory brainstem response (ABR) and auditory long latency response (ALLR). Language age was assessed using Receptive expressive emergent language scales (REELS) and Language test in kannada (KLT) for all the participants in both the control and clinical group.

ALLR was recorded using a natural speech stimulus /ba/ of 248.85 ms duration which was presented through loudspeakers at 0° azimuth at a rate of 1.1 Hz at 65 dB SPL for both the groups. Unaided ALLR was recorded from the control group, whereas both unaided and aided ALLR was recorded from the clinical group. The obtained waveforms were analyzed by three experienced Audiologists to mark P1, N1, P2 and N2 components. Since, the unaided ALLR recording of the clinical group revealed absence of ALLR, only the aided ALLR recordings of the clinical group and the unaided ALLR

recordings of the control group were considered further. Descriptive statistics was used to describe the ALLR waveform morphology of each of the participants in both the control and clinical group. Since it is established that the P1 latency is a biomarker for assessing the maturation of the central auditory system in children, only P1 latency was considered for further analysis. From the obtained date, the mean and the standard deviation of P1 latency were calculated and following statistical analysis were carried out.

- 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.

The results obtained from the various statistical procedures for both control and clinical group are as follows.

ALLR results for the Control group:

- ALLR was present for all the 12 participants.
- The mean P1 latency decreased as the language age increased from 1 – 5 years, 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.
- Children with same language age within each language age group had similar P1 latencies.

ALLR results for the Clinical group:

- ALLR was present for 10 children, out of 13 children.
- The mean P1 latency decreased as the language age increased from 1 – 5 years, 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.
- Children with same language age within each language age group had similar P1 latencies.

Comparison of P1 latency and language age between the control and clinical group:

- P1 latency was found to be longer in clinical group compared to control group across all the language age groups, but this was not statistically significant between the two groups.
- The occurrence of all the P1, N1, P2 and N2 components was lesser in number in the clinical group compared to the control group.
- The decrease in P1 latency with increase in language age was similar to that seen in normal hearing children though the latencies were slightly prolonged.

Conclusion

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 of the study

- 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.

Future research

- 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.

Appendix 1

Scoring of Receptive Expressive Emergent Scales (REELS):

Scoring

+ : When the child consistently exhibits a particular behavior.

- : When the child is not exhibiting a particular behavior.

The testing is stopped at the age group where the child is not exhibiting any of the behavior listed under the age group. The age group under which the child has scored two positive score out of three test items is considered as child's language age.

Appendix 2

Sub categories and scoring of Language Test in Kannada (KLT):

Sub categories:

Part-I: Semantics

1. Naming
2. Semantic Discrimination: expression not tested
3. Lexical category: Reception not tested.
4. Semantic similarity
5. Semantic Anomaly
6. Semantic contiguity
7. Paradigmatic relations
8. Syntagmatic relations
9. Polar questions: expression not tested
10. Antonymy
11. Synonymy
12. Homonymy

Part-II: Syntax

a. Morphophonemic structures
b. Plural forms
c. Tenses
d. PNG (person, number and gender) markers
e. Case marker
f. Conditional clauses
g. Transitive/ Intransitive/ Causative
h. Sentence type

i. Conjunctive & Quotative

j. Comparative

k. Participal construction

Scoring:

For all the categories except Lexical category, Paradigmatic relations and Plural forms, the following scoring procedure was adopted.

1: For each of the correct response.

½: For the emergent behavior which is acceptable but not listed in expected response.

0: For the incorrect or no response.

Lexical category:

1: When the child responded with or gave all the five names

½: When the child responded with or gave two or more but less than five names.

0: When the child responded with or gave single name/ incorrect/ no response.

Paradigmatic relations:

1: For identification of all the four pictures belonging to the specified group.

0: For identification of less than four pictures.

Plural forms:

1: For identifying the plural form correctly

0: For any other response

Separate total scores were obtained for the following:

i. Semantics receptive score

Semantics expressive score

Semantic score= Semantics receptive score + Semantics expressive score

ii. Syntax receptive score

Syntax expressive score

Syntax score= Syntax receptive score + Syntax expressive score

iii. Language receptive score

Language expressive score

Language score= Language receptive score + Language expressive score

$$Percentage\ score\ = \frac{\text{Raw score}}{\text{Total obtainable raw score}}$$

Chapter 6

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