ACOUSTIC CHANGE COMPLEX IN CHILDREN:

7-15 YEARS OF AGE

Register No: 09AUD002

A Dissertation Submitted in Part Fulfilment of Final Year

Master of Science (Audiology)

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ALL INDIA INSTITUTE OF SPEECH AND HEARING,

MANASAGANGOTHRI, MYSORE - 570006

JUNE, 2011

DEDICATED

TO

MY FAMILY,

GANGOTHRI SCHOOL

&

AIISH

CERTIFICATE

This is to certify that this dissertation entitled ***Acoustic Change Complex in children: 7-15 years of age*** is a bonafide work submitted in part of fulfilment for the degree of Master of Science (Audiology) of the student Registration No.: 09AUD002. 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 ***Acoustic Change Complex in children: 7-15 years of age*** 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 ***Acoustic Change Complex in children: 7-15 years of age*** is the result of my own study under the guidance of Ms. Devi N., 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. 09AUD002**

June, 2011

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INTRODUCTION

Auditory function is a complex process that develops and changes throughout life. Auditory skills related to basic perception of frequency, intensity and duration develops early, and reaches maturation by 5 years of age (Olsho, 1984; Collins & Gescheider, 1989; Trehub, Schneider & Henderson, 1995; Werner, 1996). In contrast development of more advanced auditory behavior related to aspects of speech perception and listening in noise progresses throughout the school age (Elliott, 1979). Normal auditory development provides a solid foundation for the acquisition of more complex processes such as speech and language and in turn, academic skills such as reading and written language (Elliott & Hammer, 1988).

Many school-age children have difficulty demonstrating basic proficiency in reading and writing academic areas, and are eventually diagnosed with learning or attention problems. The cause of these problems may lie in auditory perceptual deficits specifically related to processing of complex signals such as speech (Elliott & Hammer, 1988; Kraus, McGee, Carrell, Zecker, Nicol & Koch, 1996; Nittrouer, 1999). Maturation delays in the acquisition of advanced auditory processes may also be a contributing factor.

Speech evoked Cortical Potentials provides information about the biological processes underlying speech processing. The recording of cortical auditory evoked potentials (AEPs) to human speech sounds in infants have value as an index not only of the maturational state, but of the functional integrity of those regions of the cortex which process acoustically complex stimuli that are critical for the development of normal

speech and language. The assumption is that speech perception is dependent on the neural processing of the frequency, amplitude and timing cues contained within the speech signal (Kraus, McGee, Carrell, Zecker, Nicol & Koch, 1996).

Cortical auditory evoked potentials (CAEPs) have been recorded using a wide range of stimuli including tones, clicks and complex stimuli like speech stimuli. Woods, Alain, Covarrubias and Zaidel (1993), Verkindt, Bertand and Perrin (1995), Roberts and Poepple (1996), Crottaz-Herbette and Ragot, (2000) have shown differences in CAEPs latencies for different stimulus frequencies recording using conventional evoked potentials and magnetoencephalography techniques. There is some evidence that CAEPs in infants evoked by different speech phonemes differ in latency and morphology (kurtzberg, 1989). CAEPs differences between speech stimuli are an indication of different underlying neural representations of speech sounds and suggest that the information needed to differentiate the stimuli is available to the listener. There has been increasing interest in the use of cortical potentials to investigate the neural encoding of speech (Tremblay, Billings, Friesen & Souza, 2006).

Acoustic Change Complex (ACC) is a negative-positive complex that is elicited by a change that occurs during an ongoing acoustic stimulus (Martin & Boothroyd, 1999). In appearance and timing, the ACC is similar to the N1-P2 complex that occurs in response to stimulus onset (Onishi & Davis, 1968; Hillyard & Picton, 1978; Naatanen & Picton, 1987; Naatanen, 1992; Pantev, Euliz, Hampton, Ross & Roberts, 1996).

ACC has been used to study the neural detection of consonant vowel (CV) transitions (Kaurkonata, Hari & Lounasamaa, 1987; Ostroff, Martin & Boothroyd, 1998),

periodicity changes (Martin & Boothroyd, 1999), amplitude envelope and speech spectral content variation (Martin & Boothroyd, 2000). It can be recorded reliably in individuals by two variants of stop consonants and fricatives, and results are consistent with the reliability of CAEP's in response to tones (Pekkonen, Rinne & Naatanen, 1995; Vitanen, Ahveninen, Ilmoniemi, Naantanen & Pekkonen, 1998), and synthetic speech stimuli.

Need for the Study

A thorough characterization of the AEP changes that continue into adolescence is a first step in establishing whether a relationship exists between physiological maturation and the prolonged development of some psychophysical abilities (Litovsky, 1997; Schneider & Trehub, 1992; Marshall, Brandt, Marston & Ruder, 1979; Elliott, 1979; Palva & Jokinen, 1975). Maturation of the CAEPs is an extended process with profound effects on the appearance and disappearance of some components, and on the amplitude and latency of other components (Ponton, Eggermont, Kwong & Don, 2000). ACC could possibly help us to quantify the neuromaturation for complex speech signals. ACC are to hold promise as a clinical tool for assessing the neural detection of time varying cues contained in speech, as well as longitudinal changes in neural activity (Tremblay, Friesen, Martin & Wright, 2003).

A comprehensive description of age-related ACC changes in neurologically intact and normal-hearing children will provide a useful reference for assessing suspected neuromaturational deficits or central auditory processing disorders in children. These reference data may also be useful in evaluating children with hearing disorders (e.g. unilateral deafness) or profoundly deaf children fitted with cochlear implants (Ponton &

Don, 1995). So the present study was carried out to get the reference in diagnosing normal from disordered population. Electrophysiological ACC responses can provide a non behavioral means of investigating the processing of speech sound. These changing complexes can be used in individuals who neither comprehend nor participate in a behavioral task.

Wunderlich and Cone-Wesson (2006), reports that examining childhood development of the CAEP has mostly included children aged from 4 years through to adolescence and early adulthood. The span of years examined varies from study to study but there is considerable overlap from the later years of childhood (7 years onwards) up to early adolescence (about 15 years) so that this period is relatively well understood. By comparison, there is a dearth of literature on the developmental patterns of Acoustic Change Complex (ACC) in age range of 7-15 years. A study of the same might lead to a better understanding of the neuromaturation of the auditory system to complex signals in this age group.

Aim

To study the variations of Acoustic Change Complex (ACC) in children between 7 to 15 years of age for /sa/ and /si/ stimuli.

REVIEW OF LITERATURE

The responses from all auditory system structures following presentation of an acoustic stimulus can be simultaneously recorded from the cochlea, auditory nerve, auditory brainstem, medial geniculate body, and auditory cortex activating multiple brain regions at the same time. However, it would be true that the more caudal structures would have shorter onset latencies than the more rostral structures. This latency is the result of the finite action potential conduction velocity and the delay as the activity passes through chemical synapses. As we have no noninvasive way of recording from these various auditory nuclei directly, it is possible to record a series of responses from scalp (using non invasive surface electrodes) that have latencies from one-thousandth to several tenths of a second. Due to the progressive latency increase of responses from rostral auditory structures, it is popular to classify AEPs by their response time following the onset of a transient stimulus (Katz, Medwetsky, Burkard & Hood, 2009).

Different Auditory Evoked Response Potentials have been used to reveal the encoding and neural maturation taking place with the age in growing children. Auditory Evoked Response Potentials (ERPs) are bioelectrical time locked responses elicited by sound stimuli, and are classified as:

Cochlear Potentials

Electrocochleography (ECoG)

Electrocochleography refers to the responses from the cochlea and the auditory nerve, using a recording electrode located in close proximity to the inner ear. Two

responses arise from the hair cells: the Cochlear Microphonics (CM) and the Summating Potential (SP) (Dallos, 1973; Davis, 1976). Each has a very short latency (1ms or so), which is basically the delay from stimulus onset to hair cell excitation.

The CM has the same waveform as the stimulus. The SP is a Direct Current (DC) response, which continues for the duration of the eliciting stimuli (Dallos, Schoeny & Cheatham, 1972). The response from the acoustic portion of the eighth cranial nerve is called either the Whole-Nerve Action Potential (WNAP) or the Compound Action Potential (CAP). The CAP has latency roughly 1ms longer than the CM or SP, which is the result of the synaptic delay from hair depolarization to the onset of the auditory nerve fiber discharge. Unlike the other AEPs, the ECoG responses are typically NOT measured with scalp electrodes, but rather from electrodes placed in the ear canal, or near the tympanic membrane, or on the promontory (Sohmer & Feinmesser, 1967; Coast & Dickey, 1970).

Brainstem Potentials

Auditory Brainstem Responses (ABR)

ABR waveform is recorded from the brainstem due to synchronization of the nerve fibers. Five peaks are identified and Vth peak is the most prominent peak in adults. Its generation is incomplete at birth, generally only three major components (wave I, III, V) are observed. Interwave latency is initially prolonged. The wave I-V latency interval, for example, is normally about 5 ms at birth (Eggermont, Don & Brackmann, 1980; Moller & Jannetta, 1983).

During first 18 months after birth, other wave components emerge, and waves III and V progressively shorten in latency. After the first 18 months to 2 years, the ABR is essentially adult like in latency and amplitude (Salamy & McKean, 1976; Salamy, 1984).

Explanations for delayed interwave latencies in infants center on CNS anatomy and physiology, specifically incomplete nerve fiber mylenization, reduced axon diameter and immature synaptic functioning. As the maturation of the central auditory pathways takes place waveform becomes adult like. So ABR gives light to the neural maturation and brain plasticity (Moore, Ponton, Eggermont, Wu & Huang, 1996). Newborn wave I latency is prolonged from 0.3 ms (Goldstein, krumholz, Felix, Shannon & Carr, 1979; Jacobson, Morehouse & Johnson, 1982) to 1 ms (Cox, Hack & Metz, 1981) in comparison to adult values.

Frequency Following Response (FFR)

FFR has been used to evaluate aspects related to encoding of complex sounds and binaural processing. The phase-locked activity shows that it is carrying information about certain steady state and time variant acoustic features of speech sounds, pitch relevant information, and cochlear nonlinearity (Moushegian, Rupert & Stillman, 1973).

FFR is used in evaluation of influence of auditory experience in encoding of speech sounds. Children with learning impairment show degraded FFR to speech sounds. Auditory training improves the neural encoding and can be reflected by FFR, suggesting experience induced plasticity at brainstem level. So it is one of the useful tools to study about brain plasticity. The latency of FFR is between 5.5- 7ms to a 500Hz TB.

Slow Negativity-10 (SN10)

A scalp negative wave was observed by Davis and Hirsh (1979) and called Slow Negative response at 10msec (SN10). This component seems to terminate at the brainstem sequence. Its threshold of detection is close to the low frequency behavioral threshold. The SN 10 response at 10msec is generated by more apical cochlear regions than the ABR, as evidenced by the presence of clear response for low frequency and the minimal influence of high frequency hearing impairment. SN10 response varies as a function of intensity and frequency. As intensity and frequency decreases the SN10 latencies increases. An age effect is also seen as latencies are longer for all newborn responses. Latency was approximately 4ms more than the adult group (Hawes & Greenberg, 1981).

Sub-cortical potentials

Auditory Middle Latency Responses (AMLR)

AMLRs are replicable positive and negative peaks that occur between 10 and 50 ms, after the onset of the eliciting signals (Goldstein & Rodman, 1967). It usually consists of 3 positive and 3 negative peaks, which are labeled as No, Po, Na, Pa, Nb, Pb. The wave amplitudes range from 0.5 to 3.0 μv. The Pb component of the MLAEP is often identified as the P1 component of the Late Auditory Evoked Potential.

The Na component receives contributions from sub-cortical regions of the auditory system, specifically the medial geniculate body of the thalamus (Fischer, Bognar, Turjman & Lapras, 1995); and perhaps portions of the inferior Colliculus (Endho, Chiba & Hashimot, 1982). There is general agreement that the Pb component of

the AMLR arises from auditory cortex, perhaps the posterior region of the planum temporale.

The effects of subject's age vary considerably among AMLR. Responses are apparently not adult like until age 8-10 years or even later. Some reports indicate difficulty in obtaining reasonable waveform for neonate (Davis, 1976). Others, however, appeared to demonstrate that AMLRs could be reliably recorded from new-borns and young children (McRandle, Smith & Goldstein, 1974; Mendelson & Salamy, 1981; Wolf & Goldstein, 1978). Amplitude of Pa increases steadily from infancy through late childhood, and then decreases with advancing age.

40 Hz MLR

It is classified as "Steady State Potential (SSP)" or "Event Related Evoked Potential". The 40 Hz Event Related Potential (ERP) is recorded similarly to the AMLR with the exception that stimulus rate is faster, approximating 40/sec (versus the range of 11.1/sec or less). In many respects the 40 Hz ERP is just a variant of conventional AMLRs which will be elicited with somato-sensory as well as auditory stimulation. The 40 Hz ERP has also called a "High rate response" a "High rate driven response" and a "composite response" (Stach, 1986). A stimulus rates of about 40/sec produces an evoked response waveform with a peak every 25 ms or 40repetitions/sec.

Galambos, Makeig & Talmachoff, (1981) speculated on a possible neuroanatomical origin within the polysensory areas of the thalamus. Makela & Hari, (1987) postulate that the 40 Hz responses are at least partly generated in auditory cortex.

Cortical Potentials

The cortical auditory event related potentials are usually classified as two types, obligatory (or exogenous) and cognitive (or endogenous) (Davis, 1976; Picton, Rodriguez, Linden & Maiste, 1985). The obligatory are those whose presence, latency and amplitude are highly dependent upon the acoustic parameters of the stimulus and the integrity of the primary auditory pathway. While the cognitive are those in which patient's status is more important, in terms of cognition and attention.

Late Latency Response (LLR)

The slow "obligatory" cortical P1-N1-P2 evoked potentials occur within about 300ms after stimulus onset in adults. The slow cortical potential is referred to as obligatory because it is primarily determined by the physical properties of the stimulus and it invariably occurs when sound is detected by the subject (Hyde, 1997; Stapells, Tremblay & Yee 2002). The N1-P2 complex can be used to look for neural detection of acoustic cues that are important for speech perception. The complex is made up of N1, a negative peak occurring approximately 100 msec after stimulus presentation, and P2, a positive peak occurring approximately 200 msec after stimulus presentation. These waveforms are thought to represent the synchronous neural activity of structures in thalamic-cortical segment of the central auditory system (Naatanen & Picton, 1987).

The major N1 and P2 components receive contributions from in primary auditory cortex and the supratemporal plane located anterior to this region. The generators of P2 are not well established generator for P2. Based on the topographic recordings, it appears that the P2 wave receives contributions from multiple anatomic sources. The sub cortical

reticular activating system plays a major role in the generation of P2 wave. Auditory cortex is also the possible source including planum temporale and the auditory association regions (area 22) (Cody & Klass, 1968).

Cortical potentials are affected by both arousal level and attention and are typically recorded when the subjects is awake and alert or in a light sleep stage (Cody, Klass & Bickford, 1967). It has been reported that cortical potentials are similar when infants are awake and in "active" sleep (Novak, Kurtzberg, Kreuzer & Vaughan, 1989).

The morphology of the P1-N1-P2 complex is affected by maturation. The complex changes dramatically over the first 2 years of life. The complex begins as a large P1 wave is followed by a broad, slow negativity occurring near 200 to 250 ms after the onset of the sound. A P1-N1-P2 complex similar to that of adults is not seen until approximately 9 to 10 years of age unless stimuli are presented at a very slow rate. ALR latency decreases and amplitude increases as a function of age during childhood, up until about age 10 years (Weitzman, Fishbein & Graziani, 1965). Responses recorded at midline central electrode sites, reflecting contributions from primary auditory cortex, mature more rapidly than those from lateral temporal sites, which reflect maturation of secondary auditory cortex. These potentials continue to mature until the second decade of life and then change again with old age. Prolonged N1 and P2 latencies and amplitude changes have been reported in aging adults.

Speech-Evoked cortical potentials have been studied in children. For P1, N1 and P2, the earlier components were differed in the children. Adults showed the classic, well-defined N1-P2 complex. In children, a large Positive wave (P1) tended to dominate the P1/N1 complex. N1 peak latency was less well defined and P2 was smaller in children, as

compared to adults. Both P1 and N1 latency were significant longer in children (Kraus, McGee, Carrell, Sharma, Micco & Nicol, 1993).

P300

P300 is an event related or endogenous evoked response identified in the 1960's. The P300 is a component within an extended ALR time frame recorded using an oddball paradigm (standard and target signal). Target signal produces a positive peak in the latency of 300ms, which is also called P3. A missing, rare or a deviant signal can elicit P300 response. It is often described as cognitive evoked response as it depends on the detection of the difference between frequent vs. rare signals.

Diverse regions of the brain contribute to the generation of P300 including sub cortical structures – hippocampus, other structures within the limbic system and the thalamus, auditory regions in cortex, frontal lobe (Naatanen & Michie, 1979).

P300 is a positive deflection in the waveform within latency region of 250 - 400ms and up to 800ms for infants. Actual latency values for individual subjects differ due to inter-subject variability and a host of measurement parameters such as the test paradigm, passive or attending, stimulus intensity, relevance of stimulus, recording electrode site etc and subject related factors such as age, gender, and cognitive status. Latency is calculated from the onset of the stimulus. P300 wave is often broad and characterized by multiple positive peaks. Response amplitude is generally within the range of 10-20μV (Dalebout & Robey, 1997).

Passive P300 can be used with infants & young children. From 6 yrs to late adolescence, P300 amplitude increases, latency reduces, morphology improves (Squires

& Hecox, 1983). The relation between age from 6yrs up to 15 yrs and latency is defined by an average change in P300 latency as a function of age of approximately 19ms/year. P300 latency changes of 20ms/yr over the age range of 5 to 13yrs (Pearce, Crowel & Tokioka, 1989).

Mismatch Negativity (MMN)

The Mismatch Negativity Response is a negative wave elicited by a combination of standard and deviant stimuli, and occurring in latency region of about 100 to 300ms. MMN is elicited with an odd ball paradigm in which the infrequently occurring deviant sounds are embedded in a series of frequently occurring standard sounds. MMN reflects the central code of stimulus change, its amplitude and latency are related to the degree to which the deviant stimuli differs from the standard stimuli not the absolute level of standard or deviant stimuli (Stapells, Tremblay & Yee 2002).

Generation of the MMN is a reflection of several simultaneous or sequential and fundamental brain processes, including pre-attentive analysis of features of sound (frequency, intensity, duration, speech cues), extraction or derivation of the invariance within multiple acoustic stimuli, a sensory memory trace in the auditory modality that represents the sound stimulation, and ongoing comparison of the invariant (standard) stimuli versus different (deviant) stimulus. In order for the MMN system to recognize that a deviant is different from the standard, there must be a memory of the standard. Naatanen (1992), considered the relevant memory to be auditory sensory memory.

Generator site of MMN is suggested to be, bilateral generators in the supratemporal plane (auditory cortex) and frontal cortex (Naatanen & Michie, 1979).

Among cognitive evoked responses, the MMN response is the first to be detected in infancy. MMN has been detected in 50-70 % of infants studied when elicited by frequency changes and speech sound stimuli, including durational changes within speech sounds, even in premature neonates at 30 – 34 weeks of gestational age (Cheour – Luhtanen, Alho, Kujala, Sainio, Reinikainen & Renlund, 1996). Prematurity & other neonatal risk factors may have long term consequence for the MMN response. Maturation of the MMN response evoked with the speech sound stimuli (e.g., /da/ and /ga/) appears to proceed through preschool years (Cheour, Ceponiene, Lehtokoski, Luuk & Alho, 1998).

The MMN response elicited with simple stimuli (e.g. frequency differences between standard and deviant stimuli) is remarkably stable in childhood, only minor maturational changes in response latency from infancy (241 ms) to school age (207 ms) (Kurtzberg, Vaughan, Kreuzer & Fleigler, 1995).

Although the MMN response elicited by simple stimuli is adult like by age 6 years with no further changes through the age 16 years (Kraus, McGee, Carrell, Zecker, Nicol & Koch, 1996) developmental trends in MMN for speech sound stimuli occur throughout school age.

MMN has been studied between school aged children (7-11 years) and adults (17-29 years). The results showed that, there was no difference in the latency of MMN in children, compare to adults. No significant differences were found in peak latency, onset, offset or total duration (Kraus, McGee, Carrell, Sharma, Micco & Nicol, 1993).

Contingent Negative Variation (CNV)

The Contingent Negative Variation is an increasing negative shift of the cortical electrical potentials associated with an anticipated response to an expected stimulus and indicative of a state of readiness or expectancy. It is a cognitive event related to a potential. Generated in specific, nonspecific and association areas of the thalamus and within the midbrain reticular formation.

The CNV occurs approximately 400ms following stimulus onset, with a beginning of the CNV occurring at about 450-470 ms and lasting for approximately 500ms. A negative offset to the baseline of about 30 – 50μV characterizes the CNV. CNV or expectancy wave (E-wave) was first described by Walter, Cooper, Aldridge, McCallum and Winter (1964) and is related to the 'readiness' of a subject to make a response. The amplitude of the CNV appears to vary with psychological states related to meaning, motivation and expectation (Walter, 1964).

N400-P500

This is a negative potential that occurs at about 400ms and was first described by Kutas and Hillyard (1980), as being present during the presentation of semantic material. That means the N400 and P500 which appear to be related to language and linguistic features of speech. The earlier negative potential, above 400ms is related to semantic differences between the context of a sentence and the ending word of the sentence that is semantic priming. The greater the semantic mismatch, the more robust the response. This is in contrast to a positive response at about 500 ms that occurs when the ending word is different from that of the preceding word. The response is highly related to decision

making processes and higher level processing tasks (Fisher, 1984). The N400-P500 has been used to study language and the linguistic features of speech.

P600

It's a syntactic component. It occurs in syntactic reanalysis process (i.e. conscious reanalysis of sentences). ERPs have also been associated with syntactic violations within sentences. The P600 (Osterhout & Holcomb, 1995) is a widely distributed positive wave beginning at about 500 ms following a syntactically incorrect word within sentence, with a typical peak latency of about 600 ms. This P600 appears to be quite distinct from the N400 elicited by semantically incorrect words. (Kutas, 1997) violations of phrase structure, apparent sub-categorization violations have been associated with the P600. The cognitive events underlying the P600 are not yet known and there is little evidence that this response is a direct manifestation of sentence comprehension. P600 can be elicited by highly attention.

T-COMPLEX

The T-complex is characterized by a positive peak occurring between 80-90 msec and a negative peak occurring between 120-140 msec. Wolpoaw and Penry (1975) have suggested that the T-complex has two components:

T (a) occurring as a positive peak at 90-100msec

T (b) occurring as a negative peak at 140-160msec.

Cacace, Satya-Murti and Wolpaw (1990) have suggested that the T-complex is a useful tool in the evaluation of hemispheric asymmetries. This response also is sensitive

to several drugs, the processing of non-verbal stimuli and schizophrenia, the positive component (Ta) has larger amplitude ipsilaterally and slightly shorter latencies contra-laterally. The negative component (Tb) has both larger amplitude and shorter latencies contra-laterally (Connolly, 1993). The response changes with stimulus. The latencies of the response show an inverse relationship with intensity of stimulation. The amplitude of the T-complex directly follows changes in the intensity of stimulation. Specifically, amplitude increases and latency decreases as the intensity increases from about 20-80 dBSL. The latency of the negative component of the T-complex shows hemispheric asymmetry and is shorter over the left hemisphere and over the hemisphere contra-lateral to the side of stimulation. Amplitude of the T-complex is greater over the right hemisphere and over the hemisphere contra-lateral to the side of stimulation. This observation supports the temporal cortex origin of the T-complex in secondary auditory cortex on the lateral surface, probably Broadman's areas 22 and 42 (Wolpaw & Penry, 1975).

Processing Negativity (Nd)

The processing negativity is a broad, slow negative response occurring between 80 to 600 msec. It has a bimodal peak at approximately 100 and 300 msec. Processing negativity is described as a neural indication of stimulus selection where by the stimuli are categorized for additional processing (Hillyard, Mangun & Luck, 1994). It is affected by attention and is concurrent with other auditory evoked and event related potentials. The processing negativity is a derived response in that, the response to the unattended stimuli is subtracted from the response of the attended stimuli, and produces a Negative Difference Wave (Nd). Processing negativity is an endogenous potential, highly related

to memory & cognition. The latency of the processing negativity increases with increasing difficulty of discrimination.

Processing negativity originates in the auditory cortex. The earlier components (90-200 msec) reflect primary sensory processing in the specific sensory system of auditory areas related to the acoustic stimuli whereas the later components (200-500 msec) reflect perceptual and cognitive processes of the non specific sensory system of audition related to attention, memory, recognition of stimulus.

Acoustic Change Complex (ACC)

Acoustic Change Complex (ACC) is a negative-positive complex that is elicited by a change that occurs during an ongoing acoustic stimulus (Martin & Boothroyd, 1999). In appearance and timing, the ACC is similar to the N1-P2 complex that occurs in response to stimulus onset (Onishi & Davis, 1968; Hillyard & Picton, 1978; Naatanen & Picton, 1987; Naatanen, 1992). It has been demonstrated that both amplitude and frequency modulation during an ongoing sound can evoke an N1-P2 complex (Clynes, 1969; Spoor, Timmer & Odenthal, 1969; McCanless & Rose, 1970; Kohn, Lifshitz & Litchfield, 1980; Yingling & Nethercut, 1983), as can an acoustic change during a sustained speech sound (Kaukornata, Hari & Lonasma, 1987). In sustained speech sound (syllables), it occurs in response to transition from consonantal segment to vocalic segment (Hari, 1991; Imaizumi, Mori, Kiritani & Yumoto, 1996; Ostroff, Martin & Boothyard, 1998). Multiple responses evoked by the speech stimuli /shee/, in normal hearing listeners, the first N1 response signals the change in acoustic energy (from silence to sound) coinciding with the onset of consonant. The second N1 reflect a change

in acoustic energy corresponding to the onset of the vowel (Tremblay, Friesen, Martin & Wright 2003).

ACC provides important insight into the brain's capacity to discriminate the acoustic features of speech present in the signal. First, the ACC has been recorded in response to consonant-vowel syllables, in which the acoustic change include frequency, amplitude, and periodicity cues similar to those found in normal conversational speech (Kaukornata, Hari & Lonasma, 1987; Ostroff, Martin & Boothyard, 1998). The ACC has also been seen in response to isolated acoustic cues that often differentiate speech sounds as well as to combinations of these acoustic cues. For example, it had been recorded to a change from a harmonic tonal complex to a noise-band stimulus with the same spectral envelope (Martin & Boothroyd, 1999), and amplitude and formant frequency changes within a vowel (Martin & Boothroyd, 2000). Finally it shows reasonable agreement with behavioral psychophysical discrimination threshold (Ostroff, Martin & Boothyard, 1998; Martin & Boothroyd, 2000) indicated that the ACC was elicited for F2 changes that were detected with confidence. Martin & Boothryod (2000) demonstrated that the ACC was present in response to +2 or -3 dB of intensity change, which dovetails nicely with the behavioral intensity discrimination literature.

N1-P2 complex in response to periodic and aperiodic stimuli has been studied. The response of the noise-only and tone-only stimuli showed a clear N1-P2 complex to the onset of stimulation followed by sustained potential that continued until the offset of stimulation. The noise-tone and tone-noise stimuli elicited an additional N1-P2 acoustic change complex in response to the change in periodicity occurring in the middle. The

acoustic change complex was larger for tone-noise than for noise-tone stimulus (Martin & Boothroyd, 1999).

ACC has been recorded in normal hearing individuals and also checked for test-retest reliability with-in an eight-day period. Results showed that ACC by naturally produced speech sounds were reliably recorded in individuals. Also, naturally produced speech tokens, representing different acoustic cues, evoked distinct neural response pattern (Tremblay, Friensen, Martin & Wright, 2003)

The ACC was obtained from eight adults in response to change of amplitude and/or spectral envelope at the temporal center of a three-formant synthetic vowel lasting 800 ms. In the absence of spectral change, the group mean waveforms showed a clear ACC to amplitude increment of 2 dB or more and decrement of 3 dB or more. In the presence of a change of second formant frequency (from perceived /u/ to perceived /i/) amplitude increments increased the magnitude of the ACC but amplitude decrement had little or no effect. The fact that just detectable amplitude changes is close to the psychoacoustic limits of the auditory system argues well for the clinical application of ACC. The failure to find a condition under which the spectrally elicited ACC is diminished by a small change of amplitude supports the conclusion that the observed ACC to change of spectral envelope reflects some aspects of cortical frequency coding. Taken together, these findings support the potential value of ACC as an objective index of auditory discrimination capacity (Martin & Boothryod, 1999).

ACC can be recorded reliably in individuals by two variants of stop consonants and fricatives, and results are consistent with the reliability of CAEP's in response to

tones (Pekkonen, Rinne & Naatanen, 1995), and synthetic speech stimuli. Cortical evoked potentials, such as N1 response, show deviation in waveform morphology that is associated with poor speech perception. e.g., simulated hearing loss (Martin, Kurtzberg & Stapells, 2002), & sensorineural hearing loss (Oates, Kurtzberg & Stapells, 2002; Tremblay, Billings & Rohilla, 2004).

ACC has been studied in children (8-10 years) and adults (18-35 years) for stimulus /shu/ and /su/. N1P2 responses were elicited and there was significant difference between the N1P2 amplitude for both the stimulus, indicative of presence of ACC response in adults. In children it was only present in 50% of the subjects in whom LLR was present; different N1P2 amplitude was noticed across the stimulus. So ACC can be used as an electrophysiological tool for the encoding of spectral changes in adults and children if LLR is present (Karthik, 2005).

ACC is clearly elicited in individuals with moderate sensorineural hearing loss, the ACC decreases in amplitude and increases in latency as the amount of second formant frequency change decreases, and ACC thresholds show good agreement with behavioral thresholds (Martin & Boothryod, 2000).

ACC has been studied in Adult Cochlear Implant listeners (age 37 to 80 yr) and it shows that, it can be reliably recorded in individuals wearing Cochlear Implant. Furthermore, naturally produced CV syllables, /si/ and /shi/ evoked distinct ACC patterns. All individuals showed high test-retest reliability which was conducted within three months of each other (Friesen, & Tremblay, 2006).

ACC of amplified sounds has been studied to see the reliability in individuals, the response pattern and to see if different amplified speech sounds evoke different neural patterns. The results reveal that ACC can be recorded reliably in both aided and unaided conditions. Hearing aid that provide a mild high-frequency gain only subtly enhance peak amplitudes relative to unaided cortical recordings, and if the consonant-vowel boundary is preserved by the hearing aid, it can also be detected neutrally, resulting in different neural response patterns for different speech stimuli (Tremblay, Billings, Friesen, & Souza, 2006).

It has not yet been determined whether the onset P1-N1-P2 and the ACC share the same generators and tap identical processes. The ACC shows the same morphology as the P1-N1-P2, and it might be argued that the onset of P1-N1-P2 is a response to acoustic change from silence to sound. There is some evidences, however, that these response may index different processes. For example, the N1 evoked by a sudden change in pitch or timbre has been shown to have more posterior scalp distribution than the N1 evoked by tone onset. In addition, some acoustic change in speech could conceivably not produce an observable ACC if they result in waveform component overlap and cancellation.

Despite these possible limitations, the ACC has the potential to provide audiologist with important information regarding the initial stages of speech processing. When elicited, the ACC indicates that the brain, at a cortical level, has detected changes within a speech sound and the patient has the neural capacity, given intact higher neural centers, to discriminate the sounds. Therefore the ACC serve as an index of speech discrimination capacity.

Advantages of ACC are that it should be easy to elicit in a clinical setting, can be obtained even in the absence of attention, and requires relatively few stimulus presentations to record a response with a good signal to noise ratio. Most importantly, the ACC is elicited consistently in individual subjects with good test-retest reliability (Tremblay, Friesen, Martin & Wright, 2003), these factors are positive for the potential clinical application of ACC, because generally it is possible to assess the neural representation of multiple sounds within a single session.

METHOD

Participants

45 participants were taken for the present study and they were further divided into three groups (15 participants in each group) based on their age.

The three groups were as follows:

Group A: 7 to 9.11 years of age (26 ears)

Group B: 10 to 12.11 years of age (29 ears)

Group C: 13 to 15.11 years of age (30 ears)

Participant selection criteria

The criteria for inclusion of participants in the study were as follows:

- All participants had normal hearing sensitivity as revealed by Pure Tone Audiometry with Air Conduction (250-8000Hz) and bone conduction (250-4000 Hz) thresholds within 15 dBHL.
- All participants had Speech Identification Scores of 90% and above.
- All participants had normal middle ear function as revealed by Tympanometry with 'A' type Tympanogram and reflexes present at 500, 1K and 2 KHz both ipsi and contralaterally.
- All participants passed in Screening Checklist for Central Auditory Processing (SCAP), developed by Yathiraj and Mascarenhas (2002).

- All participants had no relevant Otological or neurological history and illness on the day of testing.
- For all participants, informed consent of parents/caregiver was obtained.
- All participants were native speakers of Kannada.

Instrumentation

- A calibrated diagnostic audiometer (OB-922) was used for pure tone and speech audiometry with signal matched headphones, TDH 39 and Radio ear B71 bone vibrator for measurement of the BC thresholds.
- GSI Tympstar was used to carry out the tympanometry and acoustic reflexes.
- A unidirectional microphone connected to the computer, and Adobe Audition software was used to record the speech stimuli.
- A Sound Level Meter SLM 824 LND was used to calibrate the stimulus output.
- An evoked potential system (Bio-logic Navigator Pro) was used to record cortical evoked auditory responses, Acoustic Change Complex (ACC) using /sa/ and /si/ stimuli.

Test environment

All the audiological evaluation and recording was carried out in a sound treated room (ANSI 1991; S3.1)

Procedure

- Written consent from the parents was taken for their children to participate in the study, and SCAP was administered with the help of teacher or parents. The health conditions of the children were asked from the teacher and parents.
- The behavioral thresholds in octave frequencies from 250 Hz to 8 kHz for air conduction and 250Hz to 4 kHz for bone conduction were obtained. The thresholds were traced using modified Hughson and Westlake method (Carhart and Jerger, 1959).
- Speech recognition thresholds (SRT) were found using spondees and speech identification scores (SIS) were obtained at MCL using Mayadevi (1974) test material.
- Tympanometry and Acoustic reflexes were carried out to rule out any possibility of middle ear pathology using 226Hz probe tone, and reflexes at 500, 1K and 2 KHz both ipsi and contralaterally.

Recording of ACC

Phase I (Stimulus development)

Speech stimuli /sa/ & /si/ were used to record ACC. These syllables were spoken by an adult male Kannada speaker with normal vocal effort, and were recorded by a unidirectional microphone, kept at distance of approximately 10 cm from the speaker, connected to the computer. The recording was done using Adobe Audition software

(version 2), with a sampling rate of 48000 Hz and 16 bit resolution. The stimuli duration was 248msec. for syllables /sa/ and /si/.

The best recorded signals were given to ten listeners and asked to rank them for the clarity, stimuli marked as best in the clarity were taken as the test signals. Pitch and formant frequency of the signal taken were; 106.1 Hz, F1-573.6Hz, F2-1479 Hz For stimulus /sa/; and 120.4 Hz, F1- 388.3 Hz, F2- 2647 Hz for stimulus /si/ at vowel midpoint. When analyzed, speech stimulus /sa/ found to have 133 ms portion of /s/ and 115 msec portion of vowel /a/, and stimulus /si/ found to have 147 msec portion of /s/ and 101 msec portion of vowel /i/.

Further the files were loaded in biologic system for ACC recording. Intensity calibration was done with SLM 824 LND for the stimulus to be equivalent to 80 dBSPL. Value obtained was 75dBnHL for /sa/ and /si/ stimulus for both the ears.

Waveforms of /sa/ and /si/

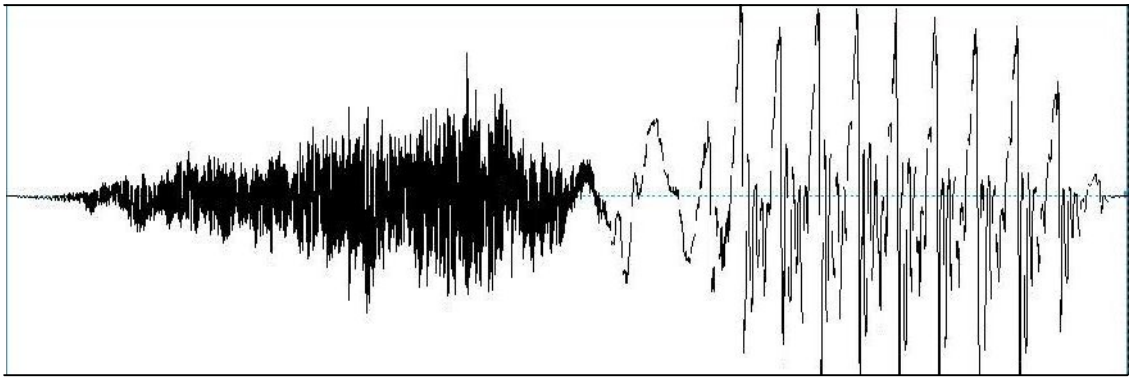


Figure 1*: waveform of stimulus /sa/ used for recording of ACC*

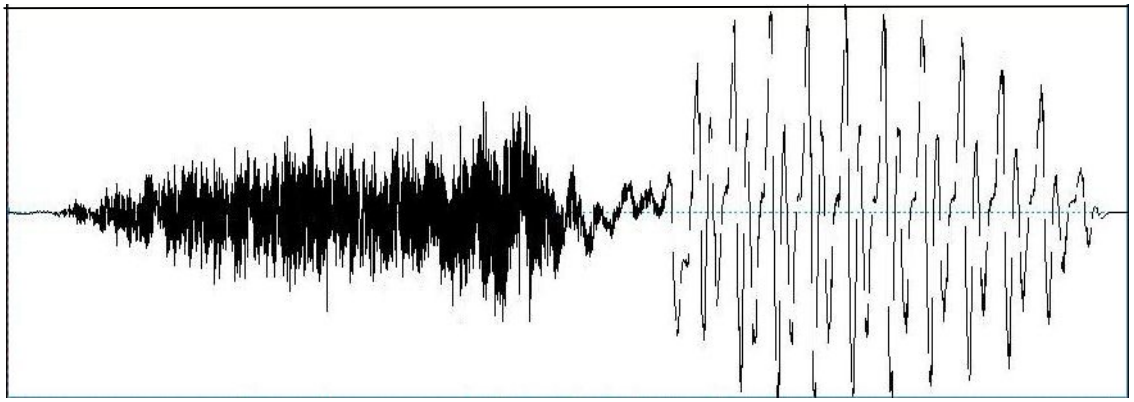


Figure 2*: waveform of stimulus /si/ used for the recording of ACC*

Phase II (Recording of the ACC)

Electrode sites were cleaned by using abrasive gel. AgCl electrodes were used and placed on different sites by applying conduction gel. Different sites for electrode placements were; inverting electrode on the test ear, non-inverting on the vertex and common on the contra-lateral mastoid. Intra electrode impedance was maintained <5 kilo-ohm, and <2 kilo-ohm inter electrode impedance.

Subjects were instructed to be awake and not to move while testing is carried out, as well as a mute cartoon video was played.

Protocol

Table 1

Stimulus and acquisition parameter used for the recording of ACC

Stimulus parameter		***Acquisition parameters***	
Stimulus	/sa/	Mode of stimulation	Ipsi
	/si/	Electrode montage	Cz, M1, M2
Duration	248msec.	Filter setting	1-30 Hz.
	For stimulus /sa/	Transducer	ER-3A
	and /si/	Analysis window	799.5 msec.
Number of sweeps	200	Notch filter	On
Stimulus rate	1.1/sec.	No. of channels	Single
Intensity	75dBnHL	Amplification	50,000
Polarity	Alternating	No. of repetitions	2

Data Analysis

Both N1-P2 complexes were identified and analyzed with respect to latency and peak to peak amplitude. Latencies and amplitude were marked visually by two experienced audiologists.

Latencies were marked as follows:

First positive peak as P1 latency, first negative peak as N1 latency, second positive peak as P2 latency, second negative peak as N2 latency, third positive peak as P3 latency. All the latencies were calculated in msec.

Amplitudes were marked as follows:

Peak to peak amplitude of N1P2 and N2P3 complexes, Amplitudes were calculated in μv.

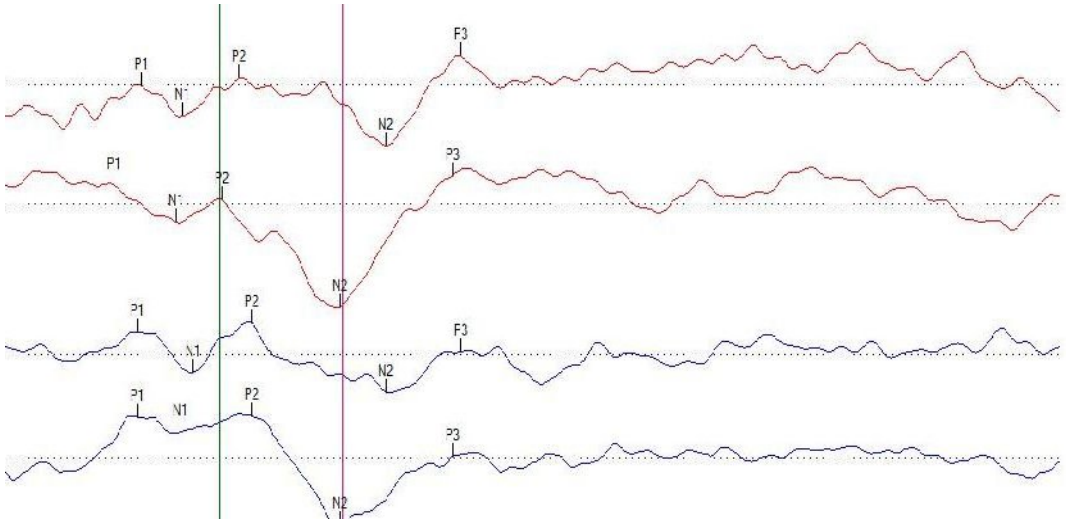


Figure 3*: Marking of latencies P1, N1, P2, N2 and P3, and peak to peak amplitude for N1P2 and N2P3*

- Latencies of P1, N1, P2, N2 and P3 were analyzed for group A (7-9.11 years), group B (10-12.11 years), and group C (13-15.11 years).
- Amplitudes of N1P2 and N2P3 were analyzed for group A (7-9.11 years), group B (10-12.11 years), group C (13-15.11 years).

RESULTS AND DISCUSSION

The latencies P1, N1, P2, N2, P3 and amplitudes N1P2 and N2P3 of different ACC components were analyzed with SPSS version (10 and 17) software, within and across the age groups. The different analyses which were done are as follow:

1. ***Waveform morphology analysis for both the stimulus /sa/ and /si/ across the age groups***

   Waveforms of ACC were analyzed and all the components were marked visually by two Audiologists.

2. ***Age related changes for ACC components elicited by stimulus /sa/ and /si/***

   a) **To obtain the mean and standard deviation for all the parameters elicited by both the stimuli, across all age groups**

      I) Mean latencies in msec and standard deviation for P1, N1, P2, N2, and P3 were calculated.

      II) Mean amplitude in μv and standard deviation for N1P2 and N2P3 complexes were calculated.

   Descriptive statistical analysis was used to calculate the mean latency and mean amplitude values along with the standard deviation for each ACC component.

   b) **To find if there is any interaction for group and stimuli**

   Mixed ANOVA was done for both the stimuli to see the interaction between stimuli, group and stimuli, and group for each ACC component.

c) **To check the significant difference among any two groups for each ACC component**

If interaction seen in Mixed ANOVA; Duncan's Post Hoc test was administered to see the significant difference among any two groups for each ACC component.

d) **To find for which of the stimuli groups were different**

Multiple Analyses Of Variance was done to find out for which of the stimulus the groups were showing the difference.

e) **To check the significant difference for each of the stimuli across the age groups for each ACC component:**

If difference was seen for the stimulus across age group, Duncan's Post Hoc Analysis was done and significant difference among any two groups was checked for particular stimulus for each ACC component.

f) **To find the significant difference between the two stimuli with-in the group:**

Paired T-test was administered to see the significant difference between stimuli /sa/ and /si/ with-in the group for each ACC component.

1. Waveform Morphology for both the stimulus across the groups

The ACC waveforms obtained for different stimuli /sa/ and /si/ were marked for each component P1, N1, P2, N2 and P3.

ACC recorded for Group 'A' (7-9.11 years):

ACC was done on 15 subjects (26 ears). ACC recorded for this group for stimuli /sa/ and /si/ has been shown in figure 4.

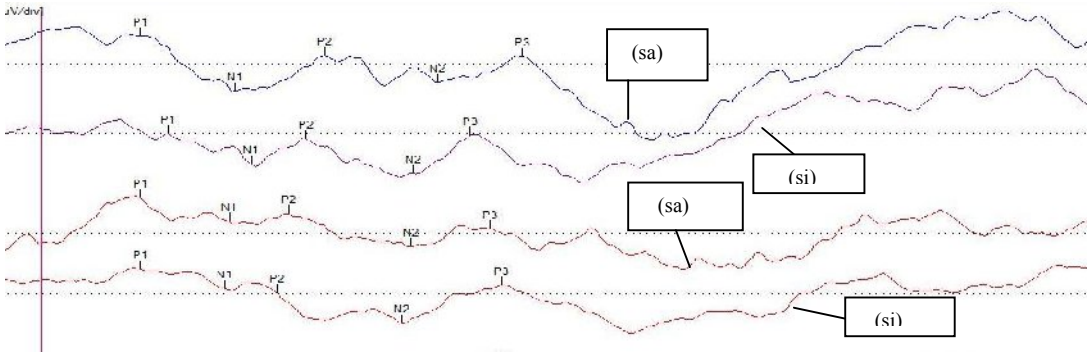


Figure 4*: ACC recording for Group A (7-9.11 years) for /sa/ and /si/ stimuli*

ACC recorded for group 'B' (10-12.11 years):

ACC was done on 15 subjects (29 ears). ACC recorded for both the stimuli /sa/ and /si/ has been shown in figure 5.

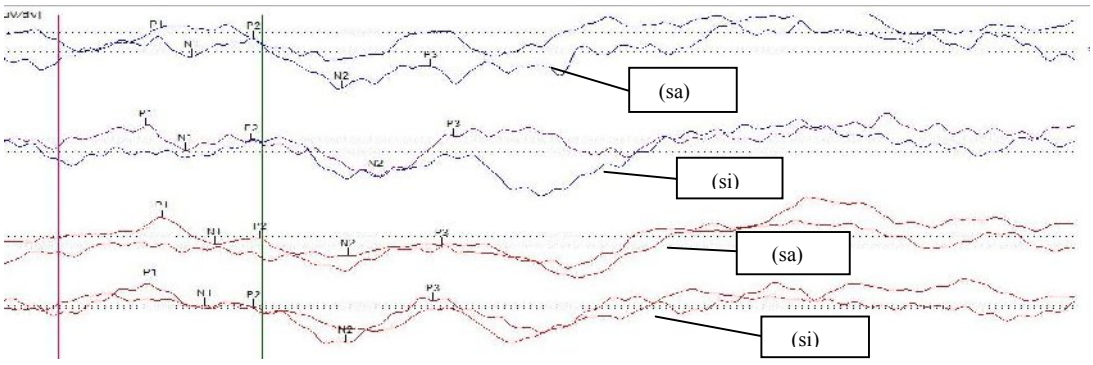


Figure 5*: ACC recording for Group B (10-12.11 years) for /sa/ and /si/ stimuli*

ACC recorded for Group C (13-15.11 years):

ACC was done on 15 subjects (30 ears). ACC pattern for both the stimuli has been shown in figure 6.

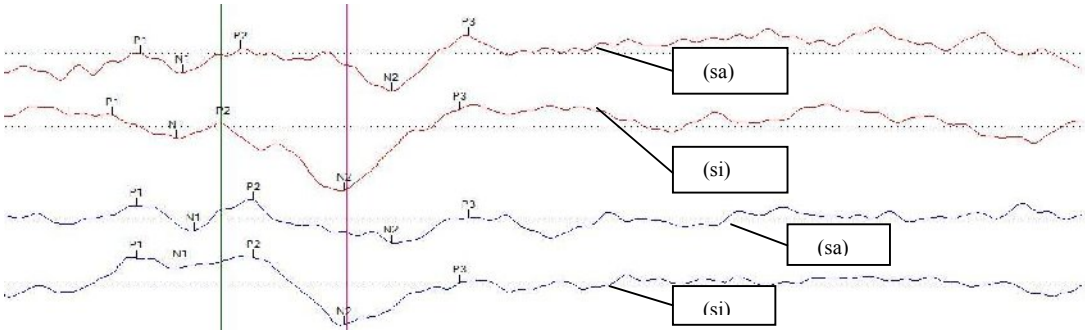


Figure 6*: ACC recording for Group C (13-15.11 years) for /sa/ and /si/ stimuli*

2. Age related changes for ACC elicited by stimulus /sa/ and /si/:

The age related changes for ACC obtained for different stimuli /sa/ and /si/ are discussed under each component P1, N1, P2, N2, P3 in terms of latency and N1P2, N2P3 in terms of amplitude separately.

P1 component

a) Mean latency and standard Deviation:

The latency for P1 component of ACC was measured across age group for both the stimuli. The mean and standard deviation were calculated by descriptive analysis.

Table 2

Mean and standard deviation of P1 latency observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimulus	Group	Mean latency (msec)	Standard deviation
P1 (Sa)	7-9.11	122.72	16.98
	10-12.11	105.55	9.67
	13-15.11	94.35	12.16
P1 (Si)	7-9.11	126.05	18.74
	10-12.11	109.71	9.15
	13-15.11	95.41	10.94

The table 2 shows that as the age increases the latency of P1 reduces for both the stimuli. Group A (7-9.11 years) has longer latency compared to group B (10-12.11 years), and group C (13-15.11 years) has shortest latencies across the three age groups.

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and the groups. Mixed ANOVA did not reveal any interaction for the stimulus [$F(1, 83) = 4.34$, $p > 0.05$]; stimuli and group [$F(2, 83) = 0.46$, $p > 0.05$]. Mixed ANOVA showed a significant interaction for the groups [$F(2, 83) = 45.32$, $p < 0.05$].

c) Significant difference among any two groups:

As the mixed ANOVA showed a significant interaction for the groups, Duncan's Post Hoc analysis was done to see which of the group had significant difference.

Duncan's post Hoc analysis revealed a significant difference between the group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($P < 0.05$), group B (10-12.11 years) and group C (13-15.11 years) ($P < 0.05$). So, all the groups were significantly different from one another.

d) Difference for the stimulus /sa/ or /si/ or for both stimuli across the age groups:

In order to find out for which of the stimulus, the groups were different, Multiple Analysis of Variance (MANOVA) was done. MANOVA revealed a significant difference for the /sa/ stimulus [$F (2, 83) = 33.44$, $p<0.05$] and /si/ stimulus [$F (2, 83) = 37.07$, $p<0.05$]. So, the groups were different for both the stimuli.

e) Significant difference among any two groups for stimuli /sa/ and /si/:

To understand, the significant difference for each of the stimulus across the groups Duncan's post Hoc was done. Post Hoc analysis revealed a significant difference between group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p < 0.05$), group B (10-12.11 years) and group C (13-15.11 years) ($p < 0.05$) for stimulus /sa/ and /si/.

It could be due to maturational changes which makes one group significantly different from the other. Also similar results were seen for speech-evoked cortical potentials in children (Kraus, McGee, Carrell, Sharma, Micco & Nicol, 1993).

f) Significant difference between two stimuli /sa/ and /si/ with-in the group:

Significant difference between /sa/ and /si/ stimuli for P1 component was noticed only in the group B (10-12.11 years) [t (27) =2.82, $p<0.05$].

N1 component

a) Mean latency and standard Deviation:

The latency for N1 component of ACC was measured across age group and the mean and standard deviation were calculated by descriptive analysis.

Table 3

Mean and standard deviation of N1 latency observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimulus	Group	Mean latency (msec)	Standard Deviation
N1 (Sa)	7-9.11	186.11	32.49
	10-12.11	147.63	12.90
	13-15.11	138.25	16.47
N1 (Si)	7-9.11	188.42	38.27
	10-12.11	150.00	10.56
	13-15.11	135.33	8.66

The table 3 shows that as the age increases the latency of N1 reduces for both the stimuli for both the ears. Group A (7-9.11 years) has longer latency, group B (10-12.11 years) has shorter latencies compared to group A (7-9.11 years), and group C (13-15.11 years) has the shortest latencies across the groups.

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and the groups. Mixed ANOVA did not reveal any interaction for the stimulus [F (1, 76) =.11, p >0.05]; stimuli and group [F (2, 76) =1.13, p >0.05]. Mixed ANOVA showed a significant interaction for the groups [F (2, 76) =42.05, p<0.05].

c) Significant difference among any two groups:

As the mixed ANOVA showed a significant interaction for the groups, Duncan's Post Hoc analysis was done to see which of the group had significant difference. Duncan's post Hoc analysis revealed a significant difference between the group A (7-9.11 years) and group B (10-12.11 years) (P <0.05), group A (7-9.11 years) and group C (13-15.11 years) (P <0.05), group B (10-12.11 years) and group C (13-15.11 years) (P <0.05). So, all the groups were significantly different from one another.

d) Difference for the stimulus /sa/ or /si/ or both the stimuli across the age groups:

In order to find out for which of the stimulus, the groups were different, Multiple Analysis of Variance (MANOVA) was done. MANOVA revealed a significant difference for the /sa/ stimulus [F (2, 76) =34.28, p<0.05 and /si/ stimulus [F (2, 76) =39.39, p<0.05]. So, the groups were different for both the stimuli.

e) Significant difference among any two groups for stimuli /sa/ and /si/:

To understand, the significant difference for each of the stimulus across the groups Duncan's post Hoc was done. Post Hoc analysis revealed a significant difference

between group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p < 0.05$) for stimulus /sa/ and /si/, perhaps for group B (10-12.11 years) and group C (13-15.11 years) there was a significant difference for stimulus /si/ ($p < 0.05$) but no significant difference for stimulus /sa/ ($p > 0.05$).

f) Significant difference between two stimuli /sa/ and /si/ with-in the group:

There was no significant difference between /sa/ and /si/ stimuli for N1 component for any of the age group.

P2 component

a) Mean latency and standard Deviation:

The latency for P2 component of ACC was measured across age group for both the stimuli and the mean and standard deviation were calculated by descriptive analysis.

Table 4

Mean and standard deviation of P2 latency observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimulus	Group	Mean latency (msec)	Standard Deviation
P2 (Sa)	7-9.11	246.68	46.54
	10-12.11	197.64	17.55
	13-15.11	181.95	14.19
P2 (Si)	7-9.11	259.56	63.26
	10-12.11	196.74	11.76
	13-15.11	177.95	13.07

The table 4 shows that as the age increases the latency of P2 reduces for both the stimuli in both the ears. Group A (7-9.11 years) has longer latency compared to group B (10-12.11 years), and group C (13-15.11 years) has shortest latencies across the three age groups.

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and the groups. Mixed ANOVA did not reveal any interaction for the stimulus [$F(1, 75) = .43$, $p > 0.05$]; stimuli and group [$F(2, 75) = 1.45$, $p > 0.05$]. Mixed ANOVA showed a significant interaction for the groups [$F(2, 75) = 54.49$, $p < 0.05$].

c) Significant difference among any two groups:

As the mixed ANOVA showed a significant interaction for the groups, Duncan's Post Hoc analysis was done to see which of the group had significant difference. Duncan's post Hoc analysis revealed a significant difference between the group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p < 0.05$), and for group B (10-12.11 years) and group C (13-15.11 years) ($p < 0.05$).

d) Difference for the stimulus /sa/ or /si/ or both the stimuli across the age groups:

In order to find out for which of the stimulus, the groups were different, Multiple Analysis of Variance (MANOVA) was done. MANOVA revealed a significant difference

for the /sa/ stimulus [F (2, 75) =35.10, p<0.05] and also for /si/ stimulus [F (2, 75) =36.89, p<0.05].

e) Significant difference among any two groups for stimuli /sa/ and /si/:

To understand, the significant difference for each of the stimulus across the groups Duncan's post Hoc was done. Post Hoc analysis revealed a significant difference between group A (7-9.11 years) and group B (10-12.11 years) (p <0.05), group A (7-9.11 years) and group C (13-15.11 years) (p <0.05) for stimulus /sa/ and /si/, perhaps for group B (10-12.11 years) and group C (13-15.11 years) stimulus /sa/ showed a significant difference (p <.005) but no significant difference for stimulus /si/ (p >.005).

f) Significant difference between two stimuli /sa/ and /si/ with-in the group:

There was no significant difference between /sa/ and /si/ stimuli for P2 component for any of the age group.

N2 component

a) Mean latency and standard Deviation:

The latency for N2 component of ACC was measured across age group and the mean and standard deviation were calculated by descriptive analysis.

Table 5

Mean and standard deviation of N2 latency observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimulus	Group	Mean latency (msec)	Standard Deviation
N2 (Sa)	7-9.11	306.37	61.10
	10-12.11	283.77	15.86
	13-15.11	275.18	15.37
N2 (Si)	7-9.11	306.45	52.73
	10-12.11	279.59	12.63
	13-15.11	263.93	10.54

The table 5 shows that as the age increases the latency of peak N2 reduces for both the stimuli. Group A (7-9.11 years) has longer latency, group B (10-12.11 years) has shorter latencies compared to group A (7-9.11 years), and group C (13-15.11 years) has shorter latencies even from group B (10-12.11 years).

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and groups. Mixed ANOVA showed interaction for the stimulus [$F(1, 84) = 4.83$, $p < 0.05$]; but no significant interaction between stimuli and group [$F(2, 84) = 2.04$, $p > 0.05$]. Mixed ANOVA showed significant interaction for the groups [$F(2, 84) = 9.58$, $p < 0.05$].

c) Significant difference among any two groups:

As the mixed ANOVA showed a significant interaction for the groups, Duncan's Post Hoc analysis was done to see which of the group had significant difference.

Duncan's post Hoc analysis revealed a significant difference between the group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p < 0.05$), but no significant difference for group B (10-12.11 years) and group C (13-15.11 years) ($p > 0.05$).

d) Difference for the stimulus /sa/ or /si/ or both the stimuli across the age groups:

In order to find out for which of the stimulus, the groups were different, Multiple Analysis of Variance (MANOVA) was done. MANOVA revealed significant difference for the /sa/ stimulus [$F(2, 84) = 5.45$, $p < 0.05$] and for /si/ stimulus [$F(2, 84) = 13.5$, $p < 0.05$].

e) Significant difference among any two groups for stimuli /sa/ and /si/:

To understand, the significant difference for each of the stimulus across the groups Duncan's post Hoc was done. Post Hoc analysis revealed a significant difference between group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p < 0.05$), but no significant difference between group B (10-12.11 years) and group C (13-15.11 years) ($p > 0.05$) for stimuli /sa/ and /si/.

The possible reason could be, the onset response elicited by vowel portion matures by age of 10 years. So there might be no significant difference in the N2 responses beyond 10 years of age. Also supported by other studies in which it has been seen that significant negativity could be traced back to the youngest age group of 10 years (Kummer, Burger, Schuster, Rosanowoski, Eysholdt & Hoppe, 2007)

f) Significant difference between two stimuli /sa/ and /si/ with-in the group:

Significant difference between /sa/ and /si/ stimuli was noticed only in the group C (13-15.11 years) [t (29) =3.03, $p<0.05$]. It could be because of the maturational changes, seen in the group age of 10 years and above, as they can detect the different stimulus onset with different latencies as the duration of consonant and vowel changes for both the stimuli are different even though the overall duration is same for both the stimulus.

P3 component

a) Mean latency and standard Deviation:

The latency for P3 component of ACC was measured across age group and the mean and standard deviation were calculated by descriptive analysis.

Table 6

Mean and standard deviation of P3 latency observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimulus	Group	Mean latency (msec)	Standard Deviation
P3 (Sa)	7-9.11	372.87	51.32
	10-12.11	352.15	18.95
	13-15.11	352.23	18.76
P3 (Si)	7-9.11	374.39	50.71
	10-12.11	343.54	21.94
	13-15.11	332.78	18.14

The table 6 shows that as the age increases the latency of P3 reduces for both the stimuli. Group A (7-9.11 years) has longer latency compared to group B (10-12.11 years), and group C (13-15.11 years) has shortest latencies across the three age groups taken for stimulus /si/ but for the stimulus /sa/ group B (10-12.11 years) and group C (13-15.11 years) showed similar latencies.

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and groups. Mixed ANOVA revealed interaction for the stimulus [$F(1, 84) = 14.95$, $p < 0.05$]; stimuli and group [$F(2, 84) = 7.00$, $p < 0.05$]. Mixed ANOVA showed significant interaction for the groups [$F(2, 84) = 8.07$, $p < 0.05$].

c) Significant difference among any two groups:

As the mixed ANOVA showed a significant interaction for the groups, Duncan's Post Hoc analysis was done to see which of the group had significant difference. Duncan's post Hoc analysis revealed a significant difference between the group A (7-9.11 years) and group B (10-12.11 years) ($p < 0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p < 0.05$), but no significant difference for group B (10-12.11 years) and group C (13-15.11 years) ($p > 0.05$).

d) Difference for the stimulus /sa/ or /si/ or both the stimuli across the age groups:

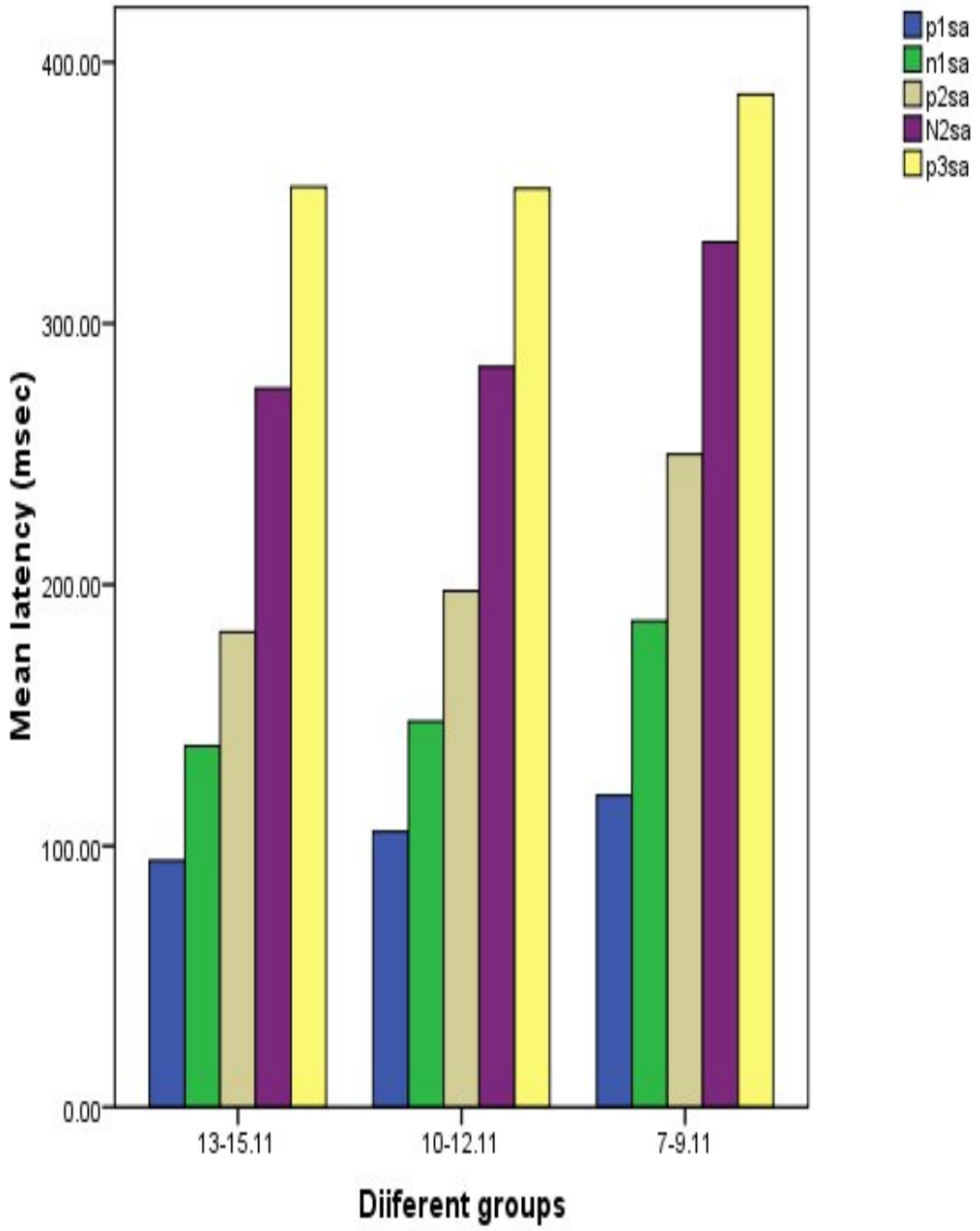
In order to find out for which of the stimulus, the groups were different, Multiple Analysis of Variance (MANOVA) was done. MANOVA revealed a significant difference for stimulus /sa/ [$F(2, 84) = 3.73$, $p<0.05$] and /si/ [$F(2, 84) = 12.15$, $p<0.05$)].

e) Significant difference among any two groups for stimuli /sa/ and /si/:

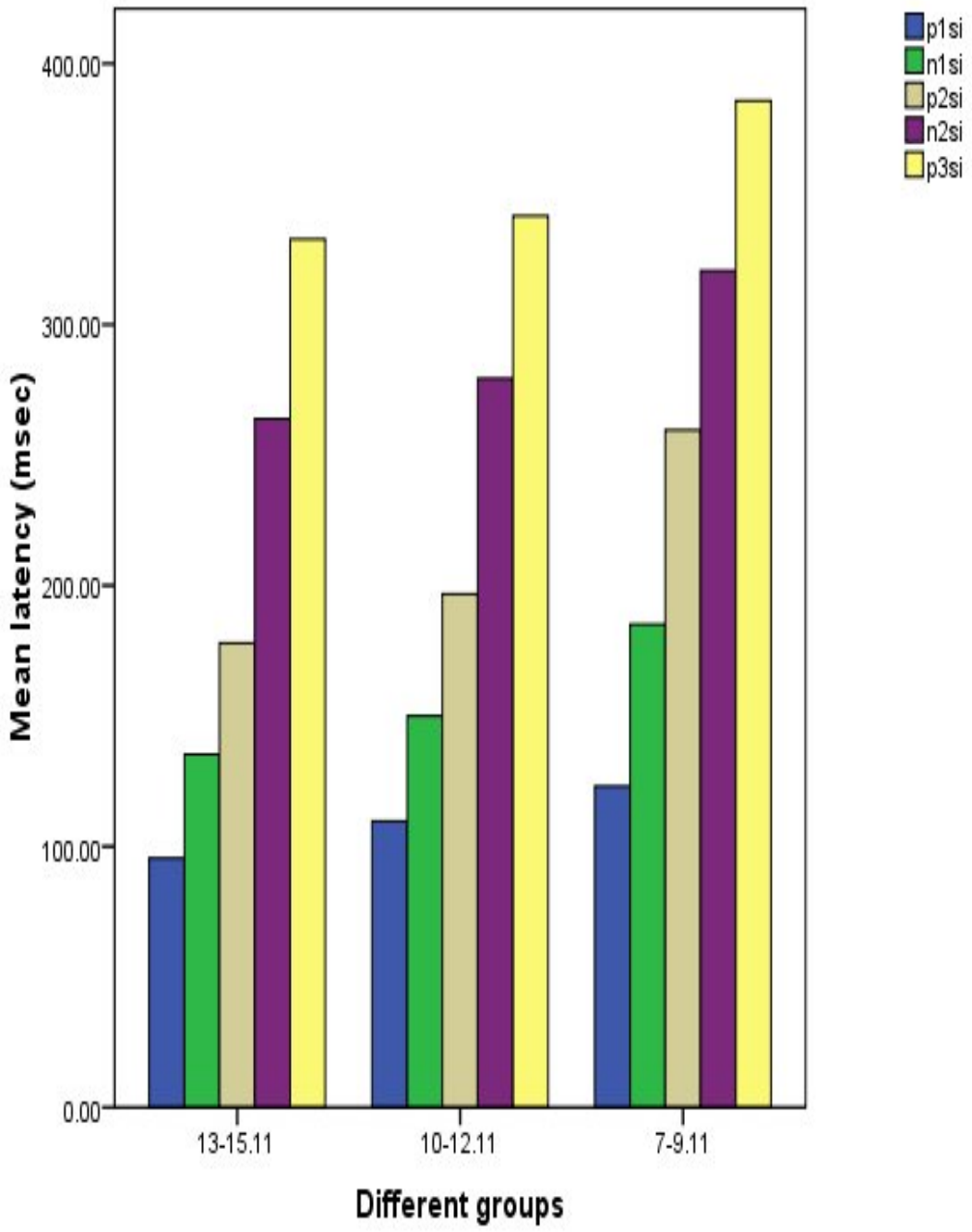
To understand, the significant difference for each of the stimulus across the groups Duncan's post Hoc was done. Post Hoc analysis revealed a significant difference between group A (7-9.11 years) and group B (10-12.11 years) ($p <0.05$), group A (7-9.11 years) and group C (13-15.11 years) ($p <0.05$), but no significant difference between group B (10-12.11 years) and group C (13-15.11 years) ($p>0.05$) for stimulus /sa/ and /si/. The possible reason could be no more maturational changes for the onset responses to ongoing stimuli for more than 10 years of age.

f) Significant difference between two stimuli /sa/ and /si/ with-in the group:

Significant difference between /sa/ and /si/ stimuli was noticed in the group C (13-15.11 years) [$t(29) = 4.03$, $p<0.05$] and group B (10-12.11 years) [$t(28) = 2.47$, $p<0.05$].



Graph 1*: Mean latencies of P1, N1, P2, N2, and P3 for stimulus /sa/*



Graph 2: *Mean latencies of P1, N1, P2, N2, and P3 for stimulus /si/*

N1P2 Amplitude

a) Mean amplitude and standard Deviation:

The amplitude of N1P2 component of ACC was measured peak to peak across age group and the mean and standard deviation were calculated by descriptive analy

Table 7

Mean and standard deviation of N1P2 amplitude observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimuli	Group	Mean amplitude (µv)	Standard Deviation
N1P2 (Sa)	7-9.11	1.44	0.78
	10-12.11	1.92	0.73
	13-15.11	1.90	0.79
N1P2 (Si)	7-9.11	1.83	0.89
	10-12.11	1.88	0.62
	13-15.11	1.80	0.69

As shown in the table 7, the amplitude of N1P2 peak increases with age for stimuli /sa/ and /si/. But for group B (10-12.11 years) and group C (13-15.11 years) amplitudes were similar.

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and groups. Mixed ANOVA did not reveal any interaction for the stimulus [F (1, 76) =0.63, p >0.05]; stimuli

and group [F (2, 76) =1.93, p >0.05]. Mixed ANOVA also showed no significant interaction for the groups [F (2, 76) =1.34, p>0.05].

As N1P2 complex is the first complex to appear it might be possible that it gets mature by 7 or 8 years of age so no significant changes are taking place in terms of amplitude but it shall be further investigated with more number of subjects.

N2P3 Amplitude

a) Mean amplitude and standard Deviation:

The amplitude of N2P3 component of ACC was measured peak to peak across age group and the mean and standard deviation were calculated by descriptive analysis.

Table 8

Mean and standard deviation of N2P3 amplitude observed at 75dBnHL for both syllable /sa/ and /si/ across the groups

Stimulus	Group	Mean amplitude (µv)	Standard Deviation
N2P3 (Sa)	7-9.11	3.94	1.78
	10-12.11	4.69	1.46
	13-15.11	5.16	1.56
N2P3 (Si)	7-9.11	3.73	1.85
	10-12.11	4.42	1.77
	13-15.11	5.02	1.39

In the table 8, it can be seen that amplitude of N2P3 increases with age for both the stimuli /sa/ and /si/.

b) Interaction for stimuli and group:

Mixed ANOVA was done to see the interaction for the stimuli and groups. Mixed ANOVA did not reveal any interaction for the stimulus [F (1, 84) =1.23, p >0.05]; stimuli and group [F (2, 84) =0.04, p >0.05]. Mixed ANOVA showed significant interaction for the groups [F (2, 84) =5.92, p <0.05].

c) Significant difference among any two groups:

As the mixed ANOVA showed a significant interaction for the groups, Duncan's Post Hoc analysis was done to see which of the group had significant difference. Duncan's post Hoc analysis revealed a significant difference between the group A (7-9.11 years) and group C (13-15.11 years) (p <0.05), but no significant difference for group B (10-12.11 years) and group C (13-15.11 years) (p >0.05), and group B (10-12.11 years) and group A (7-9.11 years) (p >0.05).

d) Difference for the stimulus /sa/ or /si/ or both the stimuli across the age groups:

In order to find out for which of the stimulus, the groups were different, Multiple Analysis of Variance (MANOVA) was done. MANOVA revealed a significant difference for the /sa/ stimulus [F (2, 84) =4.24, p <0.05] and /si/ stimulus [F (2, 84) =4.26, p <0.05].

e) Significant difference among any two groups for /sa/ and /si/:

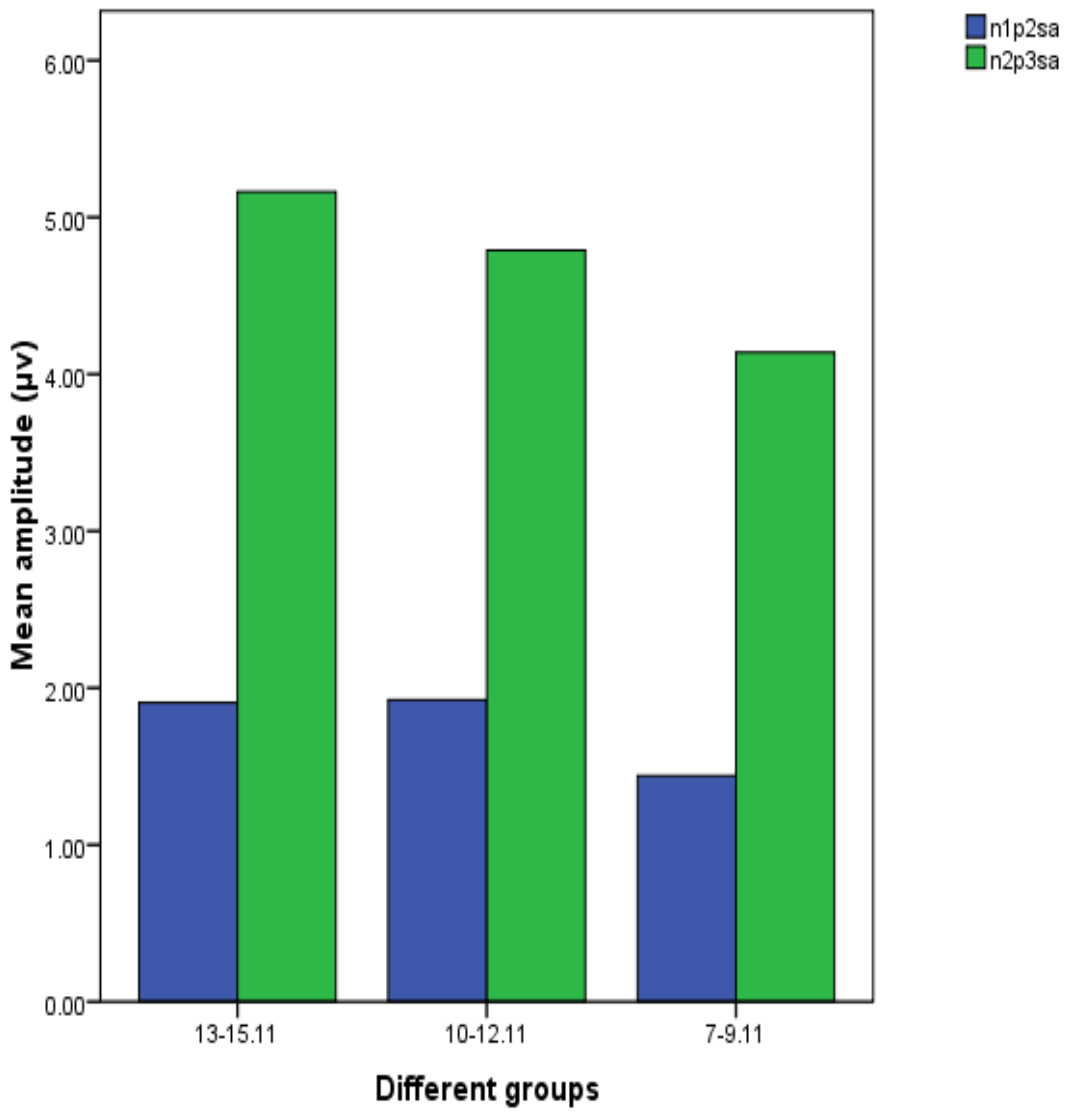
To understand, the significant difference for each of the stimulus across the groups Duncan's post Hoc was done. Post Hoc analysis revealed a significant difference

between group A (7-9.11 years) & group C (13-15.11 years) ($p < 0.05$), but no significant difference between group B (10-12.11 years) & group A (13-15.11 years) ($p > 0.05$); and group B (10-12.11 years) & group C (13-15.11 years) ($p > 0.05$) for stimulus /sa/ and /si/.

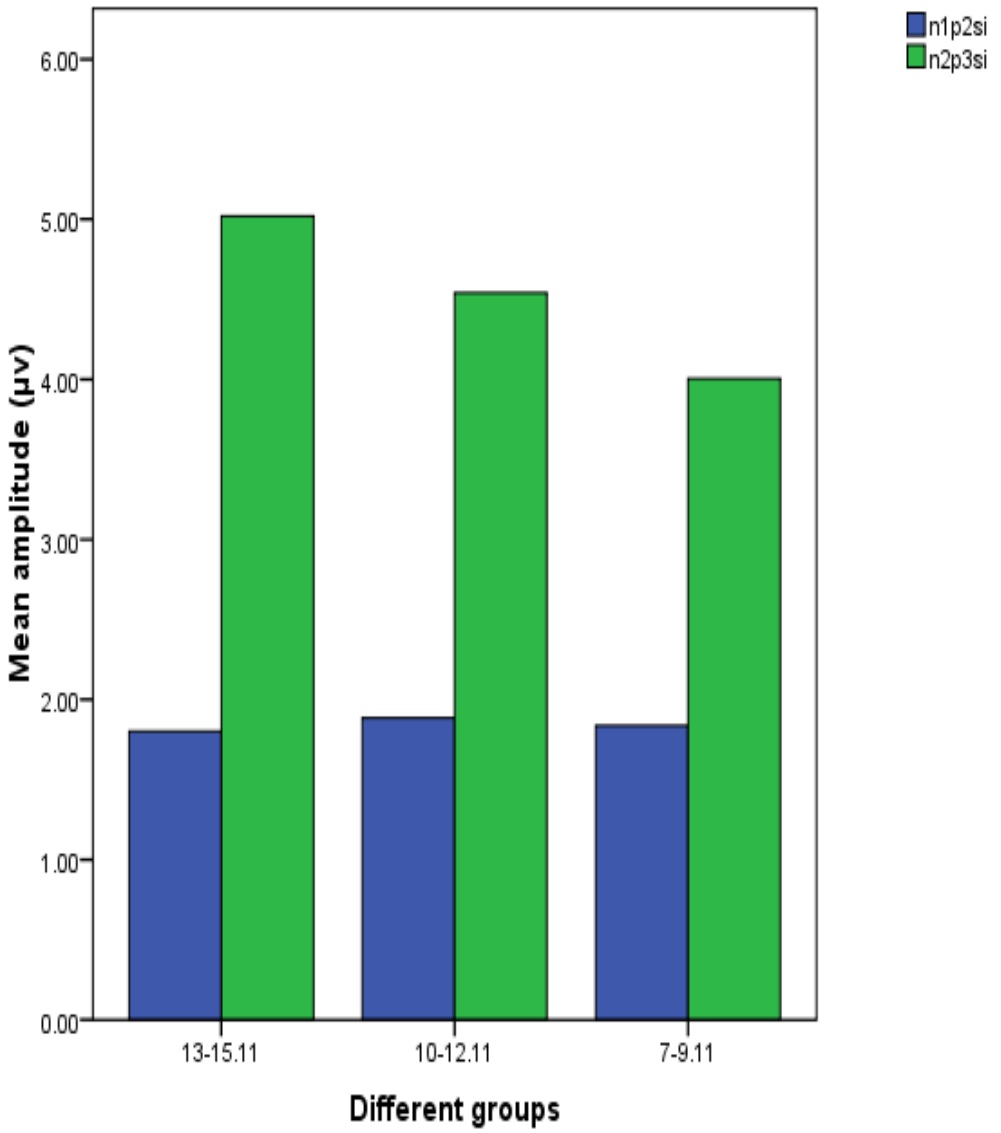
It could be possible due to maturation changes which effects the amplitude of the second complex of ACC. The second complex keeps changing in amplitude till 15 years of age, and is significant different from what is seen till 8-9 years of age. It shall be investigated further to see when it becomes adult like in amplitude.

f) Significant difference between two stimuli /sa/ and /si/ with-in the group:

There was no significant difference between /sa/ and /si/ stimuli for N2P3 amplitude for any of the age group.



Graph 3: *Mean amplitudes of N1P2 and N2P3 complexes for stimulus /sa/*



Graph 4*: Mean amplitudes of N1P2 and N2P3 complexes for stimulus /si/*

Similar results as noticed in the study are also seen in other studies, the younger group of children showed longer latencies than older group children and morphology was also better in older group. These findings are consistent with findings in CAEP that the latency decreased with increasing age (Kurtzberg, Hilpert, Kreuzer & Vaughan, 1984; Little, Thomas & Letterman, 1999; Sharma, Kraus, McGee & Nicol, 1997; Shucard,

Shucard & Thomas, 1987; Weitzman & Graziani, 1968); and the positive negative peak component of the CAEP becomes more clearly defined with age (Ponton, Eggermont, Kwong & Don, 2000). The waveform for speech stimuli for 14 years of age showed adult-like complexes. With decreasing age, P1 and N1 latencies distinctly increased and their amplitudes appeared to decrease (Kummer, Burger, Schuster, Rosanowski, Eysholdt & Hoppe, 2007).

So, ACC could possibly help us to quantify the neuromaturation for complex speech signals. ACC can be promising as a clinical tool for assessing the neural detection of time varying cues contained in speech, as well as longitudinal changes in neural activity, also supported by Tremblay, Friesen, Martin and Wright (2003).

SUMMARY AND CONCLUSION

A thorough characterization of the AEP changes that continue into adolescence is a first step in establishing whether a relationship exists between physiological maturation and the prolonged development of some psychophysical abilities (Litovsky, 1997; Schneider & Trehub, 1992; Marshall, Brandt, Marston, & Ruder, 1979; Elliott, 1979; Palva & Jokinen, 1975).

ACC could possibly help us to quantify the neuromaturation for complex speech signals. ACC are to hold promise as a clinical tool for assessing the neural detection of time varying cues contained in speech, as well as longitudinal changes in neural activity (Tremblay, Friesen, Martin, & Wright, 2003). There is a dearth of literature, on the ACC in developing age groups.

Hence, the present study was taken up with the objective of investigating the effect of age on latency and amplitude of different components of ACC elicited by the speech stimulus /sa/ and /si/. To arrive at the aim of the present study 45 participants were taken between the age ranges of 7-15 years.

They were categorized as:

- Group A: had 15 participants (26 ears) from age range of 7-9.11 years.
- Group B: had 15 participants (29 ears) from age range of 10-12.11 years.
- Group C: had 15 participants (30 ears) from age range of 13-15.11 years.

The subjects participated in the study had normal hearing sensitivity, good speech identification scores, passed in Screening Checklist for Central Auditory Processing,

normal middle ear function, no illness on the day of testing and no relevant history of Otological symptoms.

ACC was recorded for all the age groups for the speech stimuli /sa/ and /si/. The latencies were marked as P1 for first positivity, N1 for first negativity, P2 for second positivity, N2 for second negativity, P3 for third positivity. The latencies of all the components were measured in msec. Peak to peak amplitude was marked for N1P2 and N2P3 complexes and was measured in μv.

Age related changes for ACC components elicited by stimulus /sa/ and /si/ were seen in the following way:

a) **To obtain the mean and standard deviation for all the parameters elicited by both the stimuli, across all age groups:**

   III) Mean latencies in msec and standard deviation for P1, N1, P2, N2, and P3 were calculated.

   IV) Mean amplitude in μv and standard deviation for N1P2 and N2P3 complexes were calculated.

   Descriptive statistical analysis was used to calculate the mean latency and mean amplitude values along with the standard deviation for each ACC component.

b) **To find if there is any interaction for group and stimuli:**

   Mixed ANOVA was done for both the stimuli to see the interaction for stimuli, between stimuli and group, and for group for each ACC component.

c) **To check the significant difference among any two groups for each ACC component:**

If interaction seen in Mixed ANOVA; Duncan's Post Hoc test was administered to see the significant difference among any two groups for each ACC component.

d) **To find for which of the stimuli groups were different**

Multiple Analyses Of Variance was done to see the difference for the two stimuli across the age group for each ACC component.

e) **To check the significant difference for each of the stimuli across the age groups for each ACC component:**

If difference was seen for the stimulus across age group, Duncan's Post Hoc Analysis was done and significant difference among any two groups was checked for particular stimulus for each ACC component.

f) **To find the significant difference between the two stimuli with-in the group:**

Paired T-test was administered to see the significant difference between stimuli /sa/.

Different statistical analyses showed that there were systematic age related changes in latency and amplitude for different components of ACC, which are P1, N1, P2, N2, P3 and N1P2, N2P3 respectively elicited by speech stimulus /sa/ and /si/. The changes noticed were as following:

- The younger participants had longer latencies and lesser amplitude compare to older participants.
- There were systematic changes in the amplitude of N1P2 and N2P3 complexes.

- Amplitude of N2P3 complex showed significant age effect, as increased with the age, while N1P2 did not show significant interaction across age groups.
- There was difference in the morphology and it was clearer in older children compare to younger children.

These results noticed are consistent with the results for CAEP measurements, that are the younger group of children showed longer latencies than older group children and morphology was also better in older group. These findings are consistent with findings in CAEP that the latency decreased with increasing age (Kurtzberg, Hilpert, Kreuzer & Vaughan, 1984; Little, Thomas & Letterman, 1999; Sharma, Kraus, McGee & Nicol, 1997; Shucard, Shucard & Thomas, 1987; Weitzman & Graziani, 1968); and the positive negative peak component of the CAEP becomes more clearly defined with age (Ponton, Eggermont, Kwong & Don, 2000).

These reference data obtained may also be useful in evaluating children with hearing disorder or children fitted with cochlear implants or hearing aid.

It can be concluded from the study that ACC could possibly help us to quantify the neuromaturation for complex speech signals. It could also hold promise as a clinical tool for assessing the neural detection of time varying cues contained in speech, as well as longitudinal changes in neural activity.

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