ACOUSTIC CHANGE COMPLEX (ACC): AN ELECTROPHYSIOLOGICAL INDEX FOR SPEECH PERCEPTION IN CHILDREN AND ADULTS

Register No. A0390007

A dissertation submitted in part fulfillment for the degree of M. Sc. (Audiology), University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING, MANASAGANGOTHRI, MYSORE- 570006.

MAY – 2005

Dedicated to the almighty,

Amma and Appa,

Adi,

&

Deepu...

CERTIFICATE

This is to certify that this dissertation entitled **"ACOUSTIC CHANGE COMPLEX (ACC): AN ELECTROPHYSIOLOGICAL INDEX FOR SPEECH PERCEPTION IN CHILDREN AND ADULTS"** is the bonafide work in part fulfillment for the degree of Master of Science (Audiology) of the student with **(Reg. No. A0390007).** This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any university for the award of any other degree or diploma.

Place: Mysore
May 2005

Prof. M. Jayaram
Director
All India Institute of Speech & Hearing
Naimisham Campus
Manasagangothri
Mysore 570006

CERTIFICATE

This is to certify that this dissertation entitled **"ACOUSTIC CHANGE COMPLEX (ACC): AN ELECTROPHYSIOLOGICAL INDEX FOR SPEECH PERCEPTION IN CHILDREN AND ADULTS"** has been prepared under my supervision and guidance.

Place: Mysore.
May 2005

Dr.C. S. Vanaja
Guide
Lecturer,
Department of Audiology
All India Institute of Speech & Hearing
Naimisham Campus
Manasagangothri
Mysore 570006

DECLARATION

This dissertation entitled **"ACOUSTIC CHANGE COMPLEX (ACC): AN ELECTROPHYSIOLOGICAL INDEX FOR SPEECH PERCEPTION IN CHILDREN AND ADULTS",** is the result of my own study under the guidance of **Dr. C. S. Vanaja,** Lecturer, Dept of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any university for any other Diploma or Degree.

Place: Mysore. **Reg. No. A0390007**

May 2005

ACKNOWLEDGEMENT:

I would like to express my sincere gratitude to ***Professor M. Jayaram****, Director, All India Institute of Speech and Hearing, Mysore. I have always admired him and cherished the desire to develop a charismatic personality like him someday. Sir! I thank you for everything, this institute has offered me.*

I would like to thank ***Dr. Asha Yathiraj****, Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore. Ma'am you have tremendously influenced me as an exemplary, in terms of dedication towards the profession.*

I am indebted to my guide ***Dr. C. S. Vanaja****, Lecturer, Department of Audiology, AIISH, for her invaluable guidance and extreme patience throughout the completion of the project. Thank you ma'am for your motivation and critical insights throughout the course of the dissertation.*

Thank you for opening a door for me,
Having the desire to share your joy.
A teacher gives much more than thought and skill,
Nor is her heart the least she might employ.

I would also like to thank all my teachers in AIISH, viz. ***Ms. P. Manjula, Mr. Animesh Barman, Dr. R. Manjula, Dr. S. R. Savithri, Dr. N. Sreedevi, Ms. Pushpavati, Dr. Basanti Devi, Dr. Shyamala Chengappa, Mr. S. Goswami****, and other clinical faculty for the faith and self-confidence instigated in me.*

Teachers are the sculptors of young lives.
How fortunate our child was shaped by you!
A year in your class taught him how to study,
Knowing now the paths each thought derives.

I especially thank all ***my subjects*** *for their extreme cooperation, without which, this study would never have been possible.*

I thank ***Ms. Vasanthalakshmi,*** *Biostatistician, Department of Speech Pathology, AIISH, and* ***Mr. Lancy D'Souza*** *for help with the statistics.*

I am grateful to ***my parents****, who are wholesomely responsible to whatever I am. May their blessings and goodwill support me enroute my life. Special thanks to* ***Deepu****, for fervently lifting my spirits and being his own mischievous self. His support & fragrance measures immensely in my life.*

Thank you all for all that you have done.
All this we managed with you by our side,
Nor could we otherwise this course have run.

Upon your love we now proudly stand.

*Heartfelt thanks to **Aditee**, for instigating confidence and morale time and again; and being a wonderful companion throughout. Her effervescent self has brightened my life, like never before.*

Standing by, All the way.
Here to help you through your day.

Holding you up, When you are weak,
Helping you find what it is you seek.

Catching your tears, When you cry.
Pulling you through when the tide is high.

- Brittani Kokko –

*Special thanks to **Thatha, Pati, Chitti, Chittappa** and **Sunil** for making my stay at Mysore so comfortable; and their immense hospitality throughout.*

Words cannot express the feeling in our hearts.
Thank you for being with us during this difficult time.
Your thoughts, prayers and words of sympathy will
always be remembered

*My heartfelt gratitude to **Dr. Arthur Boothroyd, Dr. Brett Martin, Dr. Ostroff, Dr. Glenis Long, Dr. Naatanen, Dr. Mari Tervaniemi**, and **Dr. Kelly Tremblay** for sharing some of the splendid and invaluable reprints and internet resources.*

*I would also like to thank **Mr. Reddy Sivaprasad**. Sir! Your inspiration, encouragement and support have been an unparalleled stuff to me.*

*I also thank **Mr. Ajith, Mr. Vinay, Mr. Santosh, Sai Ram** and **Mr. Arun B. T.** Sir! Without you people I couldn't have been successful. Your critical advices and timely guidance were indispensable.*

I believe in angels,
The kind that heaven sends,
I am surrounded by angels,
But I call them friends.

- Aizabel Parinas –

*I would like to acknowledge my classmates; **Radha, JK, KD, Sudhakar, Sandeep, Hari,** and **Sujeet**; my **B.Sc batch mates (Kushum** and **Dhananjay**) and **Venkat**. They own a very special place in my heart. I would also like to thank **Tanushree, Ashley, Amy,***

Deema, Divya, Mili, Bhuvana, Rajani, Pooja, Sujita, Sonia, Devi *and* ***Geetha****. I will definitely cherish the wonderful moments we shared together.*

Friendship is like a mustard seed;
planted by God
and watered by men
- Muda Saint Michael –

A very special thanks to all juniors, ***Pandian, Pradyumn, Ramu, Sanjay, Noor, Sudipt, Prafful, Vinay, Sumesh, Shibashis, Uday, Yatin, Ankit, Manuj, Chandramouli, Ashish*** *and* ***Nikhil.***

These past few years have not been easy for me.
How I made it through them, I can't say.
All your love has helped me reach this day.

The library & library staff *of AIISH, merit special thanks.*

I would also like to thank the ***hostel cooks****, i.e.* ***Mr. Ramaswamy*** *and* ***Mr. Mahadeva*** *for their kitchen delicacies.*

Finally, very special thanks to ***Mr. Shivappa*** *for the compilation of the project.*

Thanks god for everything on earth……..

Thank you for all that you have given:
Happiness and terror, love and death,
Agony and pleasure, pulse and breath,
You gave us, us, by pain and passion riven,
One brief, bright burst of need and glory!!!!!

TABLE OF CONTENTS

CONTENTS	PAGE NUMBER
a. LIST OF TABLES	i
b. LIST OF FIGURES	ii
c. INTRODUCTION	1
d. REVIEW OF LITERATURE	5
e. METHOD	38
f. RESULTS AND DISCUSSION	42
g. SUMMARY AND CONCLUSIONS	56
h. REFERENCES	59

LIST OF TABLES

TABLE	DESCRIPTION	PAGE
Table 1	Protocol for recording of ACC (N1P2 complex)	39
Table 2	The mean and SD values for amplitude and latency parameters of N1 and P2 peaks in adults.	43
Table 3	Results of paired sample statistics (t-test) in adults (30 subjects).	44
Table 4	Amplitudes and latencies in response to /sh/ and /shu/ stimuli for 8 to 9 year old children will mean and SD.	47
Table 5	Results of Wilcoxon Signed rank test administered on 8 to 9 years old children.	48
Table 6	Amplitudes and latency values of N1 and P2 peaks and Z values of Wilcoxon singed rank test.	50
Table 7	Summarizes the N1P2 response amplitudes (normative values) across children and adults.	53
Table 8	Results of Kruskal Wallis test for significance across children and adults	54

LIST OF FIGURES

FIGURE	DESCRIPTION	PAGE
Figure 1	Depicts the stimulus waveforms for |shu| and |sh| stimuli used for eliciting an ACC response.	40
Figure 2	A sample waveform depicts the classical N1 P2 (or ACC) response that could be noted for /shu/ in comparison to /sh/ stimulus.	45
Figure 3	A sample waveform depicts the classical N1 P2 (or ACC) response that could be noted for /shu/ in comparison to /sh/ stimulus in Group II a.	49
Figure 4	A sample waveform depicts the classical N1 P2 (or ACC) response that could be noted for /shu/ in comparison to /sh/ stimulus in Group II b.	52

CHAPTER 1

INTRODUCTION

In recent years, there has been an increasing interest in the use of cortical potentials to assess speech perception capacity in clinical population. There is a need to explore a specific electrophysiological response in terms of its ability to demonstrate peripheral discrimination ability. Such tests would contribute to the objective evaluation of subjects, who for reasons of age, hearing loss, lack the auditory, linguistic and/or cognitive pre-requisites for behavioural speech perception tests (Tyler, 1993; Boothroyd 1991).

With the advent of cochlear implants, increasing number of young children are becoming candidates for cochlear implantation. Considerable success has been reported for the use of cochlear implants for children who have profound prelingually acquired deafness (Geers and Moog, 1994). As a result, there is increasing pressure to implant children with lesser degrees of hearing loss. This pressure creates an acute need for reliable objective tests of auditory speech perception capacity that remains valid when used with very young, pre phonological hearing impaired children. The availability of such tests would reduce the possibility that children with severe hearing losses will be given implants that provide no more auditory capacity that could have been obtained with properly fitted hearing aids.

Most of the previous research in this area has involved mismatch negativity (Naatanen, Gaillard and Mantysalo, 1978). Mismatch negativity is a negative ongoing, cortical event related potential that occurs in response to an occasional deviant stimulus

train of repeated standard stimuli. There are however, potential limitations to the use of mismatch negativity as a clinical tool in pediatric population. The amplitude of MMN is low in relation to background EEG activity (Picton, 1995), which, in young children is likely to be higher than adults. In addition, there is a practical limit to the amount of response averaging that can be done to increase signal to noise ratio. The limit occurs for two reasons. First, there is a limit to the time that a young child will remain immobile and cooperative. Second, the number of deviant stimuli can only be a small proportion of the total, which limits the amount of signal averaging that, can be done to improve S/N ratio for the deviant response. There is however, another event related potential that might serve in this application, namely the N1P2 complex.

The N1P2 complex is best known as an onset response (Hilyard & Picton 1978; Naatanen, 1992; Naatanen and Picton, 1987; Onishi and Davis 1968; Pantev, Eulitz, Hampton, Ross and Roberts, 1996). It has been shown that the N1P2 complex can be elicited by changes in an ongoing acoustic stimulus. Changes in ongoing acoustic stimulus will result in increase in the amplitude of the negative-positive complex and is termed as – Acoustic change complex (ACC) by Martin and Boothroyd (1999). In appearance and timing, the ACC is very similar to the conventional N1P2 complex.

Since, ACC has been elicited using stimulus having a change of spectral envelope; it is believed that ACC has potential clinical utility as a means of assessing the phonetic contrast perception in children who are yet to develop phonology and in difficult to-test population (Ostroff, Martin and Boothroyd, 1998).

Need for Study

The administration of (Acoustic change complex) ACC in adults has been evaluated by exploratory reports and has been found to yield diagnostically significant results (Ostroff, Martin & Boothroyd, 1998; Ostroff, 1999; Martin and Boothroyd, 1999; Martin & Boothroyd, 2000). None of the studies have addressed the issue of evaluating the clinical efficacy of this potential in children. Rather, they have concluded that ACC could be used as a potent clinical tool to evaluate the speech perception ability of children. This tool could aid the assessment of children who are possible candidates for cochlear implants, pre-phonologic children and difficult-to-test population. But, as a contraindication the role of maturation of auditory late latency responses (ALLR) could have an impact on ACC. Published reports state that that ALLR maturation is complete only by 12 to 15 years of age (Ceponiene, Rinne & Naatanen, 2002). This electrophysiological measure could possibly reflect the speech perception abilities, which gets expressed at a younger age in a behavioral task. Hence, there is an urgent need to administer ACC in pediatric population and validate this tool.

Additionally, there is dearth of literature, comparing and contrasting the ACC responses across age groups, i.e., pediatrics versus adults. It could possibly help us to objectively quantify the effects of neuromaturation. The auditory system is complete and functional by birth, but myelinization continues for several years in the higher auditory systems (Salamy, 1994). Though electrophysiological experiments indicate that infants display discrimination and recognition ability, psychoacoustic performances does not reach adult levels for several years (Werner and Rubel, 1992; Boothroyd 1997). This pattern of anatomical maturation is reflected in latency and amplitude of

electrophysiological responses. This accounts for the variability of responses between pediatrics and adults. This has to be determined before making critical decisions for the clinical utility of ACC.

Thus, ACC could help the hearing scientists to quantify and critically reflect upon the adequacy of speech perception ability of children, to make decisions on management. This brings us to the purpose of the study.

Aims of the Study: The present study was designed with the following aims:

1) Establish normative data on ACC for |shu| and |sh| stimuli in pediatric population and in adults

2) Investigate the effect of age on the ACC responses

CHAPTER 2

REVIEW OF LITERATURE:

Auditory perception is defined, as the interpretation of sensory evidence, derived from sound, in terms of the objects and events that caused the sound (Boothroyd, 1997; Laipply, 1990; Boothroyd, 1971). Like other kinds of perception, it involves the use, not only of sensory evidence, but also of contextual evidence, prior knowledge, memory, attention and processing skills. Attempts have been made to use electrophysiological methods to evaluate speech perception abilities with the hope that it will aid in evaluating speech perception in difficult-to-test population. A brief review of such studies is discussed in this chapter.

Evoked potentials as a measure of speech perception

Brainstem potentials

In the auditory system, the brainstem is uniquely organized to encode rapid timing changes in auditory signals with exquisite accuracy that differences in neural representation on the order of tenths of a millisecond are clinically significant (Josey, 1985; Musiek, 1991). While substantial data have been obtained revealing how brainstem neurons encode simple acoustic signals like clicks and tones, little research has been done to assess the accuracy of brainstem representation of timing events of complex signals such as speech.

King, Warrier, Hayes & Kraus (2002), compared speech evoked auditory brainstem responses in normal and children with learning impairment to determine if

there are any neurophysiologic differences between these two populations. They took 33 normal children and 54 children diagnosed as having learning impairment in the age range of 8-12 years. ABRs were elicited by formant transition of a synthesized speech syllable |da| and by an acoustic click presented at 80 dB SPL at a stimulus rate of 11.1 per second. The results revealed that all subjects had normal responses to click stimuli with no latency difference between the normal and learning impairment group. For |da| stimulus to compare the peak latencies of the normal and learning impairment groups they determined which peaks in the waveform could be measured reliably in normal children. Peaks C and F are part of FFR (Frequency following response). Peak A was significantly different between the two groups. These findings indicate that at least some of the learning impaired have abnormalities in the acoustic representation of the speech sound as low as the auditory brainstem. 20 out of 54 learning impaired children had delayed onset latencies to |da| (7.47 - 7.60 msecs) even though they had normal ABR to click stimuli. All learning impaired subjects with delayed onset latencies also had delayed latencies for peaks C and F of FFR. This was one of the earliest exploratory attempts to identify subtle auditory processing deficits as seen in children with Learning disability, at brainstem level. However, there are no reports with respect to comparison across speech stimuli.

Following this Reddy, Kumar & Vanaja (2004), studied ABR evoked by speech bursts of a stop consonant and compared the responses with that elicited by click. For this ABRs were recorded for clicks and stop bursts of |k|, |p|, |t| and |th| presented at 20, 40, 60 and 80 dB nHL for both ears of 10 normal hearing adults (18-20 years). Results indicated that overall wave morphology of ABR evoked by speech bursts was good. Amongst

other, ABR evoked by |t| and |th| had better morphology. There were robust VI and VII peaks with better morphology that that of a click. This could be because of neural excitation at the nuclei central 1 to generator of wave V. Each sound had different V peak latencies in the order of click, followed by ABRs for |k|, |t|, |th| and |p|. The difference in latencies was attributed to the spectro-temporal differences in stimuli. Lastly, slopes of Latency-Intensity (L-I) function were parallel for all stimuli except for |p|, which was steeper. This could be because of low frequency spectral energy concentration in |p|. The other L-I trends were similar to click evoked ABR. However, it is not known if the subject perceived the burst as speech or non-speech stimuli.

Another recently accomplished exploratory study which has opened up avenues for the clinical utility of speech burst ABR was carried out by Khaladkar, Kartik and Vanaja (2005). This study was undertaken on similar lines as the earlier studies, with their focus being to study the perceptual deficits in individuals with sensorineural hearing loss. They considered 20 ears with mild to moderate degree of sensorineural hearing impairment. Two stimuli were used to evoke the auditory brainstem responses, viz. an acoustic click and burst portion of syllable |t|. The results showed that while click evoked ABRs elicited latency values well within normal limits, speech burst ABRs showed deviant results for the same measure. Thus, the study critically forecasts the possible clinical utility of speech burst ABR in identifying perceptual deficits associated with individuals with sensorineural hearing impairment.

Cortical potentials

1. **Auditory long latency responses (ALLR)**

The ALLR was actually the first auditory stimulated electrical response to be recorded from central nervous system (Hall, 1994). In 1939, Pauline Davis and his colleagues described it as "on-response" to sound in EEG and used the term 'k-complex' to describe it (Davis, 1939). The components of ALLR include P1/P60, N1/N100, P2/P160 and N2/N200. One of the earliest attempts to study aspects of ALLR was undertaken by Sharma, Marsh and Dorman (2000). They tried to examine the relationship between the morphology of N1 component of the CAEP (Cortical auditory evoked potential) and the perception of voicing contrast in syllable initial position. This was an effort to verify by Sharma & Dorman (1999), where they had reported that the first component (N1) occurred in response to the burst at the beginning of each syllable and second component N1 occurred in response voicing onset. 5 males and 5 females native speakers of English in the age range of 20-30 years served as subjects. Two continuums of CV speech sounds varying in VOT, from |ga| to |ka| and from |ba| to |pa| were used as stimuli. The VOT in both were varied in the tokens. The results showed distinct morphological changes related to encoding of VOT in AEP waveforms. For both the |ga-ka| and |ba-pa| continuum, stimuli with short VOT (0-30 ms) elicited a single negativity and long VOT (40-70 ms) elicited two negative components. A significant positive correlation was found between N1 latency and VOT. For N1 component, VOT did not demonstrate main effect. For |ba-pa| continuum, N1 amplitude was significantly negatively correlated with VOT, whereas N1 amplitude did not show a main effect of VOT and same for |ga-ka|. Behaviorally, the subjects revealed a mean category boundary

at a VOT of 46 ms for |ga-ka| and a VOT of 275 ms for |ba-pa| continuum. Therefore the change in N1 morphology (from single to double peak) coincided with change in perception from voiced to voiceless for stimuli from |ba-pa| continuum but not for stimuli from |ga-ka| continuum. Therefore, N1 morphology does not reliably predict phonetic identification of stimuli varying in VOT. The results critically highlight the fact that cortical potentials are associated with high degree of inconsistency, and hence the discrepancy was observed in terms of neurophysiologic representation of speech cues.

Studies have demonstrated the complexity of evoked potentials to a simple syllable. The temporal structure of the evoked potential appears to reflect both the spectral and temporal aspects of the stimulus. Steinschneider, Schroeder, Arezzo and Vaughan (1995), presented evidence that auditory system places constraints upon neural encoding of speech that allows for categorical perception. Multi unit activity (the net potential activity of neuronal ensembles with approximate 100μm in diameter surrounding an electrode), was measured from the auditory cortex of an awake monkey. The stimuli consisted of clicks and syllables with four different voice onset times (VOT); 0ms (/da/), 20ms (/da/), 40ms (/ta/), 60ms (/ta/). Two main responses were produced in relatively equal proportions; (i) “Phase-locked” response patterns to the onset of the syllable followed by response time locked to the F0 of the syllable, (ii) “double on” patterns which contained a peak at the stimulus onset and a 2^{nd} peak at the onset of voicing. Responses to 40ms and 60ms showed this “double-on” response. Responses to 0ms and 20ms showed a peak at the onset of the syllable. The authors suggested that the “double –on” response demonstrates evidence for categorical perception at neuronal level because the categorical boundary is marked when the response gains a second peak.

With the /da/ category, the responses contained only a peak to the single onset voicing and within the /ta/ category two peaks were displayed. The “phase-locked” responses however did not show categorical perception because phase locking to the onset of voicing was progressively delayed as the VOT was increased and there was no indication of a boundary between /da/ and /ta/ categories. They also noted that both response types were equiprobable in the neurons sampled; the psychoacoustic boundary probably represents the average of the temporal activity patterns in the auditory cortex. Results also suggest that various acoustic elements are processed by different populations of cortical neurons and speech stimuli are encoded at different levels of the auditory pathway. This was one of the earliest reports which utilized near field recording, carried out to gain deeper insights of cortical neurophysiology. This study empirically reinstates the idea that each perceptual cue is coded differently by different set of cortical neurons.

Following this study, a number of studies tried to investigate the correlation between definite sets of evoked potential parameters to specific stimulus specific attributes. Tremblay, Friesen, Martin & Wright (2003), conducted a study with 7 normal hearing native English speakers (23-31 years) as subjects. Four tokens from the standardized UCLA version of nonsense syllable test (NST) were used. These tokens were naturally produced CV syllables, i.e. /bi/, /pi/, /i/ and /si/ spoken by a female talker. Results indicated that the voiced stimulus /bi/ evokes a response that is different from /pi/, because negative (N130) and positive peaks (P217) appear large in response to /bi/ stimulus. There was a significant latency difference between /bi/ and /pi/ for these 2 peaks. Waveform patterns evoked by the /si/ and /i/ are also different from each other. N345, P413 and N519 appear earlier in latency in response to the /i/ stimulus. Although

first positive peaks (P242) is larger in amplitude in response to /si/ stimulus this difference is not significant. However N346 and N 519, as well as P413 are significantly earlier when elicited by /i/ stimulus. Therefore naturally produced speech tokens, representing different acoustic cues, evoke distinct neural response patterns. This study clearly highlights the variability in N1, P2 and the later peaks, across a variety of naturally produced stimuli. Similar published reports have been quoted in literature, which have used MMN cortical evoked potential.

2. MMN

The MMN is a frontal negative deflection in the human event-related potential that typically occurs when a repeating auditory sound stimulus changes in some manner (Naatanen, Gaillard and Mantysalo, 1978). It occurs approximately 200 ms after the stimulus onset. There appear to be two components of MMN, both originating in the auditory cortex with contributions from auditory thalamus and hippocampus (Csepe, Karmos and Molnar 1987; Kaukoranta, Sams, Hari, Hamalainen and Naatanen, 1989).

One of the preliminary attempts to study cortical speech sound processing using MMN was carried out Aaltonen, Niemi, Nyrke and Tuhkanen (1987), examined the processing of speech sounds with the MMN. Using oddball paradigm they presented stimuli drawn from the Finnish /i/ to /y/ vowel continuum. The MMN was larger and occurred stronger in response to deviant stimuli when the 2 types of stimuli presented in a man consisted of the pure vowels /i/ and /y/ than when the stimuli consists of the boundary stimulus and one of the pure vowel. This effect occurred equally whether subject ignored or attended the stimuli. The MMN thus varied with the extent of the

acoustic difference between the stimuli, just as with other auditory stimulus like tone bursts. This study gave a deep insight into aspects of perceptual cues affecting the MMN evoked by speech stimuli.

Another study carried out on similar lines was by Sharma, Kraus, McGee, Carell and Nicol (1993), who predicted that if the MMN was reflective of categorically perceived stimuli as a result of phonetic analysis, crossing a phonetic boundary, will yield a MMN with larger amplitude than a MMN to stimuli in the same category. Nine synthetic stimuli in the /da/ to /ga/ continuum varying in the 2nd and 3rd formants were used. No significant differences in latency or amplitude values of MMN responses were evidenced. One of the limitations of the studies being that they have not investigated other possible cues like VOT, burst amplitude, burst spectrum, fundamental frequency and so on. Also, 2nd and 3rd formant transitions are minor cues with respect to speech perception.

In an effort to overcome the demerits of previous studies, and to have a better insight into neurophysiologic mapping of speech features, Maiste et. al. (1995), tried to determine whether there are physiological correlates of categorical perception. For this purpose, they recorded human evoked potentials in response to computer modified speech sounds from a nine stimulus continuum between /ba/ and /da/. In the first experiment, subjects listened to trains composed of 52% /ba/ or /da/ and 60% of each of the other eight stimuli and classified the stimulus as /ba/ or /da/. In second experiment, subjects read a book and ignored trains containing a standard stimulus and 2 deviant speech sounds, one within the same category as the standard and other across the category boundary. The third experiment was similar to the first except that the subject

was reading. The fourth experiment compared the responses to stimuli that deviated from standard in terms of their phonetic category or intensity. The results showed a N2P3 complex evoked by those stimuli in the more improbable category when the stimuli were attended to the first experiment therefore perceived in categorical manner. In the 2nd and 3rd experiments there was a clear MMN for the across category deviant stimuli when the standard stimuli came from the /ba/ end of the continuum. However, when the standard stimulus came from the /da/ end of the continuum, there was no definite MMN. The overall frequency content of the /da/ stimulus was broader than that of the /ba/ stimulus. A deviant stimulus from /da/ end of the continuum, thus contained frequencies which were not present in the /ba/ standard stimuli and these frequencies could elicit a MMN. In the 4th experiment, the MMN evoked by a small change in intensity was much larger than evoked by a change in phonetic challenge. Therefore it could be concluded that N2P3 complex accurately reflects phonetic categorization of speech stimuli. The MMN evoked by changes in speech sounds may indicate the detection of acoustic rather than phonetic changes. Hence, this justifies the claim of MMN as a tool or speech discrimination ability.

Similar studies were also reported in clinical populations. Groenen, Snik and Broek (1996), tried to evaluate the clinical relevance of MMN in normal hearing listeners and cochlear implant users. In the first experiment MMN were conducted in adults with normal hearing, to see the effects of decreasing duration of the interstimulus interval. Shortening ISI duration does not seem to how high impact on the individual MMN quality, whereas it does influence group MMN quality. In the second experiment MMNs were elicited in a group of cochlear implant users using a speech sound contrast, i.e. /ba/-

/da/. A group of good performers produced a significant MMN whereas a group of moderate performers did not. Therefore, there seems to be a relation between speech perception ability and MMN quality. However, other dominant perceptual parameters like, VOT and burst amplitude were not employed across studies.

In due course of time the essence of VOT as a cue for categorical speech perception was realized. Hence, studies started using the VOT parameter more than the other acoustic cues. A study by Sharma and Dorman (1999), tried to examine neural correlates of VOT perception at the level of auditory cortex in human listeners. They measured N1 and MMN cortical auditory evoked potentials in conjunction with behavioral testing using /da/-/ta/ continuum (VOT varied from 0-80 in 10ms steps). 16 adults (11 female and 5 male) in the age range 20-30 years served as subjects. All were native English speakers. They participated in discrimination and identification experiments. Results revealed a robust MMN in the across category condition. A minimal MMN was obtained in within category condition. The results of MMN compliment the results of behavioral discrimination. In N1, for stimuli with short VOTs (0-30 ms), a slight negativity was apparent. However, for stimuli with long VOTs (50-60 ms) distinct negative components (N1 and N2) were apparent. Thus the enhanced MMN responses and the morphological discontinuity in N1 morphology observed in region of the /da/-/ta/ phonetic boundary appear to provide neurophysiologic correlates of categorical perception.

However, future reports, aimed at studying specific aspects of cortical maturation (and/ or plasticity) coupled with associated changes. Shestakova, Huotilainen, Ceponiene & Cheour (2003), investigated learning related changes in auditory event related

potentials (ERPs) of finish speaking 3-6 year old children odd ball paradigm, ERPs to sounds of French language were recorded in 2 groups of healthy children: those who were learning French (Experimental group) and those who were not hearing any foreign language (control press). Brain responses recorded for standard showed that when the children from the experimental group were exposed to foreign language, they automatically developed French specific memory traces that helped them to discriminate, categorize and pronounce utterances of the new language as indicated by the MMN component of ERPs in a previous study. They found that the learning process was also reflected by changes in P3a and late difference negativity (LDN) responses. Unlike, MMN and P3a, LDN has been discovered relatively recently and its functional role remain unclear. Similarly, as the MMN magnitude increased during the learning process, an increase of the P3a (known to reflect the involuntary attention searching toward ads deviant stimuli) and LDN amplitudes was observed. The ERPs of the control peers did not change significantly over the best period. Hence, it was concluded that when phonemes of a foreign language are learned the process is accompanied by increase in MMN, P3a and LDN. Hence, the specific acoustic features adherent to a non-native language gets superimposed on to the perceptual system, which in course of time can be documented electrophysiologically.

A parallel study was reported by Ceponiene, Lepisto, Alku, Aro and Naatanen (2003). They tried to determine the ERP characteristics and ERP indices of central auditory speech sound encoding and discrimination in young children. Auditory sensory event-related potentials and ERP index of auditory sensory discrimination (the MMN) were elicited by vowel stimuli in 3 year old children. In an odd ball paradigm, the

standard stimulus was vowel /a/, one deviant stimulus was vowel /o/ (the across-category change), and the other was nasalized vowel /a/ (within category change). In addition, ERP changes occurring during the 14 min uninterrupted recording were examined. As indexed by the sensory P1, N2 and N4 peaks, the 3 year old children's transient neural encoding of vowels was comparable to 1 year old children, but also showed vowel specific characteristics observed in school age children. The 3 year olds MMN was comparable in amplitude to the school age children's MMN and appeared to be sensitive to the across category aspects of vowel changes. However, its latency was longer in 3 year olds than in school age children. Among the sensory ERPs, only the N4 peak showed significant diminution during the experiments. The across category MMN diminished after 10 min of the recording, however, over frontal areas only. It was concluded that sensory processing of vowels exhibited transitional characteristics in 3 years old children. Also the auditory sensory discrimination in the 3 year olds appeared to be sensitive to the phonemic aspects of stimulus change. This signifies the efficacy of MMN as a tool to monitor the maturational changes in the perceptual system in terms of speech sound processing.

3. **P300**

P300 was first described by Sutton, Braren, Zubin & John (1965) as a component of human auditory evoked potential that is not affected by the physical parameters of the eliciting stimuli. The component appears to reflect the cognitive processing of stimuli

information on the part of the subject. The amplitude of P300 is 15 micro Volts and under certain conditions it may be bimodal (Polich, 1989), i.e. P300a and P300b.

a: - When there is large stimulus difference whether or not the subject is actively attending to stimuli sequence.

b: - Subject is actively discriminating between stimuli. The P300 is elicited by 'Task relevancy', i.e. by a stimulus that requires some response or judgment and whose occurrence is uncertain.

Very limited literature is available in terms of P300 and its utility to study speech perception. Kileny, Boerst, and Zwolan (1997) investigated late and cognitive (mismatch negativity, P300) auditory potentials in 14 children with cochlear implants between the ages of 4 and 12 years. The length of cochlear implant use ranged from 7 to 84 months. Three types of stimulus contrasts were used: (1) a loudness contrast consisting of a 1500 Hz tone burst presented at 75 (standard) and 90 dB sound pressure level (deviant); (2) a frequency contrast consisting of a 1500 Hz tone burst (standard) and a 3000 Hz tone burst (deviant) presented at 80 dB sound pressure level; and (3) a speech contrast consisting of "heed" (standard) and "who'd" (deviant) delivered with a roving loudness paradigm involving a randomized variation of the levels of the standard and deviant stimuli. Latencies and amplitudes of components N1, P2, N2, and P3 and mismatch negativity were measured. Overall, there were very few missing or unidentifiable components. P3 and mismatch negativity components were identified for all subjects and all stimuli. The latencies of most components were affected by stimulus type. There was a trend for longer latencies for the speech contrast compared with the loudness or frequency contrasts. This may be a reflection of the increased processing time required for the

speech stimuli because of its higher complexity. There were several significant correlations between speech recognition and cognitive evoked potential latencies. These results indicate that the clinical use of cognitive evoked potentials (especially P300 and MMN) in children with cochlear implants is feasible and informative. However, specifically, the role of P300 in speech perception evaluation is very much restricted and more emphasis has been laid to other cortical potentials.

However, some studies in literature report of the possibility of using P300 as a tool to aid speech perceptual analysis. Beynon, Snik, and Van den Broek (2002)**,** obtained endogenous P300 and exogenous slow vertex potentials with tone and speech stimuli in a group of five children using a cochlear implant (CI) with poor speech recognition (group A) and compared with those from another group of five children using a CI with good speech recognition (group B). The responses were also compared to those of children with normal hearing (n= 14) and a group of adult CI users (n=9). N1 and P2 latencies of CI group A and group B were prolonged compared to those of normally-hearing children. In group A, P300 was present when contrasts with tone stimuli were used. When speech stimuli were used, P300 potentials were absent or delayed. P300 potentials obtained in group B were no different from those obtained in normally-hearing children. It is suggested that the poor results evoked with speech stimuli in contrast to those evoked with pure-tone stimuli in group A are due to the immaturity of (sub) cortical generators associated with acoustical and phonetic processing. In contrast to the children in group B, all children in group A suffered from congenital deafness.

Another study on similar lines was reported by Groenen, Snik, Beynon and Broek (2001), who tried to study the speech recognition in cochlear implant users using speech,

evoked cortical potentials. They stated that processing in auditory cortex may play a role in the unexplained variability in cochlear implant benefit. P300 and N1P2 were elicited in post lingual deaf cochlear implant users wearing Nucleus 22 multichannel devices. Four sound contrasts were presented (500Hz, /ba/, /da/, /ba/, /pa/, /i/, /a/). N1 and P2 were present in all subjects for all conditions. Prolonged N1, P2 and P300 was found in cochlear implant group compared to a control group of subjects with normal hearing, cochlear implant users show smaller amplitudes of N1 for all speech signals as well as smaller amplitudes of P2 for the consonants compared to controls, P300 results of cochlear implant users were compared to behavioral results of speech recognition testing. A relation was found between P300 amplitude and magnitude for the 500 Hz-1 KHz and /a/-/i/ contrasts and behavioral speech recognition in cochlear implant users. The results suggest that P300 measurements are useful and have additional value to speech recognition evaluations in cochlear implant users.

4. **N400-P500**

Following the P300 a negative potential that occurs at about 400 msecs and was first described by Kutas and Hilyard (1980). This N400 is present during the presentation of semantic material. The negative potential (i.e. at 400 ms) is related to semantic differences between the context of a sentence and the ending word of the sentences. The greater the semantic mismatch, the more robust the response. It has an amplitude of about 6 micro volts and a width of about 80 to 100 msecs. The generator sources of N400-P500 are not known, but the response is highly related to decision-making processes and higher level processing tasks (Fischer et al 1981).

5. **CNV**

The CNV occurs approximately 400 msecs following stimulus onset, with the beginning of the CNV occurring at about 450-470 msec and lasting for approximately 500 msecs. The CNV is characterized by a negative offset to the baseline of about 30 to 50 UV. The appearance of CNV is highly correlated to psychological status of expectancy and motivation. The CNV or expectancy wave (E-wave) was first described by Walter and colleagues and is related to the 'readiness' of a subject to make a response. It is a cognitive ERP. The CNV appears between a warning stimulus and an imperative stimulus. Rohr Baugh and Galliard (1983) argue that there are 2 components to CNV:

I. An earlier response (250 msec after the warning stimulus) that is an orienting response to the warning stimulus and is considered as vertex non specific response.
II. A component of 500 msec that may include the Bereites chafts potential (a slow negative potential that occur prior to onset of movement).

Although there is very limited literature with respect to CNV and speech perception one of the earliest studies was done by Goto, Adachi, Utsunomiya, Nakano, and Chin (1978)**.** They investigated evoked response, P300 and CNV in the condition in which the subject was required to make a differential response to the acoustically and visually presented sentence with or without meaning, and he was able to determine whether the sentence had a meaning or not by understanding the key information. In healthy persons, P300 amplitude to the beginning of information and to the key information was larger than those to the others. P300 latency to the key character was longer. CNV appeared at the beginning of information and continued. The difference between CNVs produced by

meaningful and meaningless sentences was observed after the key information. In aphasia, the difference between CNV's was not observed. In auditory agnosia, the difference between CNVs was not observed to acoustic sentences, but appeared to visual sentences. These results suggest that evoked response, P300 and CNV can be useful for assessing recognition of sentences. But the study had incorporated more of a language based task than focusing on evaluation the role of cortical speech processing.

Subsequent studies went to study speech discrimination ability using CNV. Jacobson, and Gans (1981), tried to investigate how CNV magnitude is influenced by speech discrimination tasks of graded difficulty. Nine young adult subjects performed easy and difficult speech discrimination tasks while cortical activity was recorded from the vertex. Stimuli consisted of consonant-vowel pairs that were presented in a background of noise. Consistently larger CNV responses were obtained for the difficult versus the easy discrimination task in the nonimperative condition. No consistent trend was found for the imperative condition. There electrophysiological results correspond well with both correct and incorrect discrimination behavior. The clinical implications of these findings are not very significant and hence in due course of time research interest in CNV declined.

6. **P600/Syntactic Positive Shift**

The P600/Syntactic positive shift/SPS is a slow positive shift occurring approximately 600 msec after the stimulus. It occurs in response to violations of a range of syntactic rules, including agreement between the subject and verb in a sentence (Neville, Nicol, Barss, Foster & Garett, 1991), phrase structure (sentences containing

violations of syntactic constraints within a language Hagoort et. al. 1993, Neville et. al. 1991) and sub categorization (words that don't fit the syntactic structure of a sentence, Osterhout and Holcomb, 1992). These P600/SPS have been found in response to both spoken and written stimuli (Hagoort and Brown, 2000). The later responses like N400, P500, P600 are not used clinically, rather are recorded in laboratory environments to study the neurobiology of language.

7. **T-Complex**

It is characterized by a positive peak occurring between 80-90 ms and a negative peak occurring between 120-140 msec. Cacace and colleagues (1988) have suggested that the T-complex is a useful tool in the evaluation of hemispheric asymmetries. This response also is sensitive to several drugs (Wolpaw and Penry, 1978), the processing of non-verbal stimuli (Wolpaw and Penry, 1975) and schizophrenia. The positive component (Ta) has a larger amplitude ipsilaterally and slightly shorter latency contra laterally. The negative component (Tb) has both a larger amplitude and shorter latency contra laterally (Connolly, 1993). These responses change with the stimulus. Amplitude of the T-complex is greater over the right hemisphere and over the hemisphere contra lateral to the side of stimulation (Cacace et. al., 1988). Walpaw has suggested that the temporal cortex origin of the T-complex is the secondary auditory cortex on the lateral surface probably Broad man's areas 22 and 42 (Wolpaw et. al., 1975). Further investigation has shown that the T-complex to be generated by a dipole source radically oriented in the primary auditory cortex on the posterior surface of the temporal lobe (Pantev, Hoke & Lukenhoner, 1986).

8. **Processing negativity**

The processing negativity is a broad, slow negative response occurring between 80-600 msec. It has a bimodal peak at approximately 100 and 300 msecs (early and later). Hilyard and colleagues (1995) described it has a neural indication of a stimulus relation whereby the stimuli are categorized for additional processing. It is affected by attention and is concurrent with other auditory evoked and event related potentials. Processing negativity is a derived response in that the response to the unattended stimuli is abstracted from the response of the attended stimuli thus producing a negativity difference wave (N_d). Processing negativity is highly related to memory and cognition, and hence is an endogenous potential. The latency increases with increasing difficulty of discrimination (i.e. categorization).

Acoustic Change Complex (ACC)

ACC is another event-related potential, namely the N1P2 complex that occurs in response to acoustic change during an ongoing stimulus. The N1P2 is best known as an onset response (Hilyard and Picton 1978; Naatanen 1992; Naatanen and Picton 1987; Onishi and Davis, 1968; Pantev, Eulitz, Hampton, Ross and Roberts 1996). If the auditory cortex produces a measurable response to an acoustic change during an ongoing stimulus, it follows that the different acoustic patterns, occurring before and after the change, are represented by different patterns of excitation that are manifested at every level of processing from the cochlea through the cortex. Such, a response would index the arrival, at cortical level of potentially discriminable information.

ACC is thought to be elicited due to change in the spectral envelope or periodicity of the speech stimuli. Review of literature emphasizes on the role of spectral transition for speech perception. Furui (1986) examined the relationship between dynamic spectral features and the identification of Japanese syllables modified by initial or final truncation. The experiment confirmed several main points. "Perceptual critical points", where the percent correct identification of the truncated syllable is a function of the truncation position changes abruptly are related to maximum spectral transition positions. A speech wave of approximately 10ms of duration that includes the maximum spectral transition position bears the most important information for consonant and vowel perception. Consonant and vowel identification scores simultaneously change as a function of truncation position in the short period, including the 10ms period for final truncation. This suggests that crucial information for vowel and consonant identification is contained in same initial part of each syllable. The spectral transition is more crucial than unvoiced and buzz bars for syllable perception, although the later features have some perceptual importance.

Subsequent studies went on to state that acoustic features and acoustic changes are represented at different levels of the auditory system. King, McGee, Rubel, Nicol and Kraus (1995), reported that the central processing of acoustic stimulus change can be observed neurophysiologically in the mismatch negativity auditory evoked potential (MMN). Stimuli differing in interaural phase were used to investigate the contributions of primary and non primary auditory pathways in the encoding of binaural stimuli and to investigate passively elicited measures of binaural processing in experimented animals. In guinea pigs, the MMN was obtained in response to 1000Hz tones embedded in white

noise (S:N = 2:1 dB). Using a modified odd ball paradigm, stimuli were presented binaurally with both tone and noise in phase in the two ears (S0N0) as the standard stimulus and the tone 180° out-of-phase [S{P1)N0] as the deviant stimulus. The responses to the deviant stimulus in the odd ball paradigm were compared to the responses of the same stimulus when presented in a series alone. The responses to S0N0 and S(P1)No collected in a series alone, termed the intrinsic responses, were also compared. Responses were recorded from surface epidural electrodes – one over the posterior midline and one over the left temporal lobe AEPs from this location have shown to reflect the activity of the primary and non primary thalamocortical pathways respectively. A significant MMN was observed at the midline electrode, but no MMN was observed over the temporal electrode (or lobe).

There is a very small body of literature demonstrating the N1P2 potential to a change in an ongoing speech stimulus (Kaukoranta et al 1987). Recent investigations by Ostroff, Martin and Boothroyd 1998, have been able to replicate the findings of Kaukoranta et al 1987. Cortical potentials N1 and P2 were obtained in eight adults with normal hearing. Twice naturally produced speech stimuli were used: (i) the syllable [sei], (ii) the sibilant [s] extracted from the syllable (iii) the vowel [ei] extracted from the syllable. The isolated sibilant and syllable preserved the same time relationships to the sampling window as they did in the complete syllable. Clear responses were observed for both the sibilant and isolated vowel. Although the responses to [s] were weaker than to [ei], both elicited N1 and P2 components with latencies, in relation to sound onset, appropriate to cortical onset potentials. The vowel onset response was preserved in the

response to the complete syllable, although with reduced amplitude. This justifies the fact that N1P2 complex is elicited as a result of ongoing spectral change in the stimuli.

Later it was Ostroff (1999), who tried to study and develop an electrophysiologic test for speech discrimination capacity, by examining the smallest sound formant (F2) change within a vowel stimulus that elicited the ACC and compared those threshold values to behavioural responses. Stimuli were generated by creating a synthetic 800msec 3-formant vowel with 7 separate degrees of F2 change from 1050Hz. The F2 change occurring at 393 msec, were either 9,19,38,75,150,300 or 600Hz. A control condition with no spectral change was also included. Electrophysiologic responses were recorded from 3 adults at 5 scalp positions. The smallest F2 change that elicited an ACC in the group waveform was: (a) 75Hz based on morphology and amplitude criteria, and (b) 28Hz based on curve fit and interpolation technique. Behavioural responses were recorded from 7 out of 8 subjects who demonstrated an ACC in the electrophysiologic experiment. Using the same curve fit and interpolation technique used in the electrophysiologic experiment, the binary choice, change/no change detection task revealed a mean just noticeable difference threshold of around 5Hz. The mean detection threshold was around 20Hz when taken from correct scores at 75%. The range of threshold values for the ACC from the group waveform correspond with mean detection probabilities between 86%-97% correct in the behavioural task. These findings support the conclusion that the ACC can be elicited by a F2 change that is perceived with confidence. An ACC elicited by F2 changes between 30-75Hz is likely sufficient for the detection of F2 changes between English vowels which are in the order of 100Hz. His

data is encouraging in terms of clinical utility of ACC as a tool for speech discrimination performance.

In a comparative study, Martin and Boothroyd (1999), tried to study cortical auditory event related potentials in response to periodic and aperiodic stimuli with the same spectral envelope in normal hearing subjects. They elicited ACC using signals that were concatenated to produce 2 stimuli that changed in the middle (noise-tone, tone-noise). Two control stimuli were created by concatenating copies of the noise and two copies of the tone (noise only, tone only). The stimuli were presented using an onset-to-onset interstimulus interval of 30. For elicitation of the MMN, the tonal complex and the noise band stimuli were presented using an odd ball paradigm (deviant probability = 0.14) with an onset-to-onset interstimulus interval of 600msec. The stimuli were presented via headphones at 80dBSPL to 10 adults with normal hearing. Subjects watched a silent video during testing. The results indicated a clear N1P2 complex for noise only and tone only stimuli, to the onset of stimulation followed by a sustained potential to the offset of stimulation. The noise-tone and tone-noise stimuli elicited an additional N1P2 ACC in response to the change in periodicity occurring in the middle. The ACC was larger for tone noise stimulus. A clear MMN was elicited by both the stimuli. In contrast to ACC, there was no significant difference in amplitude across the two stimuli. The ACC was concluded to be a more sensitive index of peripheral discrimination capacity than the MMN, primarily because its average amplitude was 2.5 times as large.

As an extension of the previous study, Martin & Boothroyd (2000) demonstrated that N1P2 complex could be produced by a formant change in an ongoing quasi-synthetic

vowel stimulus. A single cycle from each of the vowels /u/, /i/ and /u/ were extracted from digitally recorded words, and iterated over 150msec. The quasi-synthetic vowels were equalized for fundamental frequency and rms amplitude. They were then concatenated to produce two stimuli, [ui] and [uU], each with a duration of 300msecs. These stimuli were selected because they permit the evaluation of electrophysiologic responses to a spectral change in the middle of an ongoing stimulus based on an F1 ([uU]) and an F2 ([ui]) contrast. The N1P2 complex was elicited by the spectral change contained within both the F1 and F2 contrasts, although the response to F2 contrast was larger. This could be accounted for by the fact that F2 differences between /u/ and /i/ is much larger than F1 differences between /u/ and /U/. The latency of the response was approximately 150msec in relation to the onset of the change for both the types of formant change.

In summary, it is highlighted from the review of literature that, since the clinical relevance of brainstem and cortical evoked potentials has been realized, a wide variety of electrophysiological measures have been developed. All theses evoked potentials have been evaluated for their role in reflecting the speech perception ability of adults and children. However, except a few potentials which can tap specific aspects of speech sound processing, none of the potentials is a comprehensive reflection of the neurophysiological mapping of speech. For instance, speech evoked ABR taps temporal patterns of speech processing at brainstem level, MMN taps attention independent gross speech sound discrimination and so on. However, it is clear that the N1P2 complex which has been termed as – Acoustic change complex (ACC) can be elicited by a change in the ongoing stimulus and could serve as a diagnostic tool for fine ground speech sound

discrimination (cortical processing). Hence, sticking to the conventionally emphasized comprehensive test-battery approach for assessment, a combination of speech evoked ABR (brainstem level), ACC (cortical level) and a behavioral assessment technique [like, Ling's sound test or (ESP) Early speech perception test] would constitute reasonably sensitive and specific test for speech perception evaluation in children. But, the major limitation of using a cortical evoked potential like ACC would be make considerations for LLR maturation, which is complete only by adolescence (Sharma, Kraus, McGee & Nicol, 1997).

Effect of Maturation on Cortical Evoked Potentials:-

Review of literature, is indicative of significant difference in cortical evoked potentials (or ERPs), thereby re-emphasizing the critical aspect of neuromaturation. Anatomically, it involves myelinisation, dendroganesis and formation of newer synaptic junctions. While, physiologically, it is reflected through enhanced nerve conduction velocities, better dipole source localization of generators and maturation of peaks.

One of the earliest studies was as reported by Goodin, Squires, Henderson and Starr (1978), who studied age related variations in auditory evoked potentials to auditory stimuli in normal human subjects. Auditory evoked potentials were recorded from 47 subjects ranging in age from 6 to 76 years, in order to assess the effects of maturation and aging on the evoked (N1 and P2) and event related (N2 and P3) components. Because of clear differences in the effects of age on the event related components between children (less than 15 years of age) and adults. The subjects were divided into 2 populations for

analysis. For adults there was a systematic increase in latency and decrease in amplitude of each component with age. Also the rate of age-related increase in latency was proportional to the latency of the components. The scalp distributions of both the stimulus evoked and event-related components were found to vary with age yielding a more nearly equipotential distribution for older subjects. For children the latencies of the event related components decreased with age. The stimulus evoked components had latencies which were not significantly different from those predicted from the adult data. In contrast to the adult data, age affected the scalp distribution of the stimulus evoked components differently than the event related components. These results suggest an aging process is reflected in the auditory evoked potential which is not the simple inverse of maturational processes. In due course the interest in studying maturational changes died down.

It got revived nearly a decade later by Johnstone, Barry, Anderson and Coyle (1996), who studied age related changes in child and adolescent event related potential component morphology, amplitude and latency to standard and target stimuli in an auditory odd ball task. Previous studies of auditory event related potentials (ERPs) to an odd ball task in adolescents and children focus on responses to target stimuli and provide little detail of age related changes to standard stimuli. In this study, age related changes in behavioural responding and ERP component morphology, amplitude and latency to standard and target stimuli were examined. Auditory ERPs to an odd ball task were recorded from midline sites (Fz, Cz and Pz) of 50 subjects aged 8 to 17 years 11 months. Behavioural results indicate a decrease in reaction time and errors of omission with age. To standard tones, N2 amplitude and N1 latency show a linear decrease with age while an

increase with age was found for P2 amplitude. For target tones, N1 and N2 amplitude and N1, N2 and P3 latency showed a linear decrease with age and P2 and P3 amplitude showed a linear increase with age. Age related changes in the morphology of the ERP elicited by standard tones (especially N2 and P2 components) as well as concurrent morphological changes in standard and target tones were reported.

Later, studies started incorporating speech stimuli for studying maturational changes. Sharma, Kraus, McGee and Nicol (1997) studied developmental changes in P1, and N1 central auditory responses elicited by consonant-vowel syllables. They examined maturation of central auditory pathways as reflected by age related changes in the P1/N1 components of the auditory evoked potential (AEP). A synthesized consonant vowel syllable /ba/ was used to elicit AEPs (cortical) in 86 normal children ranging in age from 6-15 years and ten normal adults. Distinct age related changes were observed in the morphology of the AEP waveform. The adult response consists of prominent negativity (N1) at about 100msec preceded by a smaller P1 component at about 50msecs. In contrast the child response is characterized by a large P1 response at about 100msecs. This wave decreases significantly in latency and amplitude upto 20 years of age. In children (6 to 10 years), P1 is followed by a broad negativity at about 200msecs which is termed N1b. Many subjects (especially older children) also show an earlier negativity (N1a). Both N1a and N1b latencies decrease significantly with age. Amplitudes of N1a and N1b do not show significant age-related changes. All children have the N1b; however the frequency of occurrence of N1a increases with age. Data indicative that the child P1 develops systematically into the adult response; however the relationship of N1a

and N1b to the adult N1 is unclear. These results indicate that maturational changes in the central auditory system are complex and extend well into the second decade of life.

One of the comprehensive studies which tried to measure the aspects of cortical evoked potential maturation was carried out by Cunningham, Nicol, Zecker and Kraus (2000). They tried to evaluate the maturational progression of speech evoked P1/N1/N2 cortical responses over the life span. They also tried to determine the distinctiveness of these responses in clinical populations like learning problems and elucidate the functional significance of those. The P1/N1/N2 was measured in 150 normal subjects (5 to 78 years) and 86 subjects with learning problems (8 to 15 years) to a synthetic CV syllable. Analyses included description and comparison of the developmental time course in both groups and evaluation of the relationship between P1/N1/N2 and children's performance on speech discrimination tasks. Results revealed significant changes in waveform morphology, latency and amplitude as a function of age. Maturational patterns in the group of children with learning problems did not differ from the normal group. P1/N1/N2 parameters were significantly correlated with standardized tests of spelling, auditory processing and listening comprehension in the learning problem group. The P1/N1/N2 complex changes throughout life from school age to old age. The developmental sequence throughout the school age years is similar in normal and learning problem children. The relationship between P1/N1/N2 parameters and the standardized measures of learning (auditory processing and N2 latency) provides new information about the role of these responses in hearing and highlights the potential value in characterizing auditory processing deficits. This study laid the foundation in terms of establishing a correlation

between behavioral and objective electrophysiological results in normals and clinical population (i.e. Learning disability).

A similar study on Auditory evoked responses (AERs) was conducted by Ponton, Eggermont, Kwong and Don (2000), who studied the maturation of human central auditory system activity evidenced from multichannel evoked potentials. The purpose of the study was to evaluate central auditory system maturation based on detailed data from multielectrode, recordings of long latency auditory evoked potentials (AEP). AEPs were measured at 30-scalp electrode locations from 118 subjects between 5 and 20 years of age. Analysis focuses on age related latency and amplitude changes in the P1, N1 and P2 and N2 peaks of the AEPs generated by a brief train of clicks presented to the left ear. Substantial and unexpected changes that extend well into adolescence were found for both the amplitude and latency of the AEP components. While the maturational changes in latency followed a pattern of gradual change, amplitude changes tended to be more abrupt and steplike. Age related latency decreases were largest for the P1 and N1b peaks. In contrast, P2 latency did not change significantly and N2 peak increased in latency as a function of age. Abrupt changes in P1, P1-N1b and N2 peak amplitude (also RMS amplitude) were observed around age 10 at the lateral electrode locations Cz and Fz. These changes in amplitude coincided with a sharp increase and plateau in AEP peak and RMS amplitude variability from 9 to 11 years of age. These analyses demonstrated that the pattern of AEP maturation depends on the scalp locations at which the responses are recorded. The distinct maturational time courses observed for individual AEP peaks support a model of AEP generation in which activity originates at two or more at least partly independent central nervous system pathways. These results indicate that some

areas in the brain activated by sound stimulation have a maturational time course that extends into adolescence. Maturation of certain auditory processing skills such as speech recognition in noise also has a prolonged time course. This raises the possibility that the emergence of adult like auditory processing skills may be governed by the same maturing natural process that affect AEP latency and amplitude.

However, some of the studies specifically concentrated into children and infants to study developmental patterns. Ceponiene, Rinne and Naatanen (2002), studied the maturation of cortical sound processing as indexed by event related potentials. They noted that children's auditory event related potentials (ERPs) are dominated by P1 and N1 peaks, while the N1 wave emerges between 3 and 4 years of age. The neural substrates and behavioral correlates of the protracted N1 maturation, as well as the 10 year long predominance of N2 are unclear. They utilized high resolution electroencephalography to study the maturation of auditory ERPA from age 4 to adulthood and to compare the sources of N1 and N2 peak in 9 year old children and adults. Three partial tones were delivered with short (700msecs) and long (mean of 5secs) stimulus onset asynchrony (SOA) with only 700msec SOA used with 4 year olds. With short SOA, 4-and 9-years olds also displayed an N1 peak, which was frontal in scalp distribution compared to that in adults who showed P1, N1 and P2 peaks. After filtering out the N2 activity, the N1 was also revealed in the short SOAE data in 9 year old children but not in 4 year old children. In adults and in 9 year olds, the neural scores of N2 and N1 mapped onto the superior aspects of the temporal lobes, the sources of N2 being anterior to N1. The results indicated that the children's N1 is composed of differently weighed components as that in adults, and in both children and adults the N1

and N2 are generated by anatomically distinct generators. A protracted ontogeny of the N1 could be linked with auditory sensitivity and orienting, whereas the P1 and N2 peaks are suggested to reflect auditory sensory process. The study critically highlights the surface scalp distribution, and justifies their claim with evidence based reasoning.

A similar study was reported by Kushnerenko, Ceponiene, Balan, Fellman, Huoitilainen and Naatanen (2002), who studied the maturation of auditory event related potentials during the first year of life. This study examined the maturation of cortical auditory event related potentials (ERPs) from birth until 12 months of age. In the 15 infants studied, all ERP peaks observable at 12 months of age. The P_{150}, P_{250}, P_{350} and P_{450} were identifiable already at birth. As in previous studies, the amplitude of ERP peaks increased and latencies shortened with increasing age. In addition, the time course of the amplitude growth of these peaks differed from each other. It was considered that, the generators of all the infantile ERP peaks are functional already at birth, and that the maturational changes in waveform morphology can mostly be accounted for by the changing relative strengths of different generators. This hypothecation supports the fact that, neurophysiologic maturation of the anatomical generators takes place as a function of age.

As an extension of their previous report, Ponton, Eggermont, Khosla, Kwong and Don (2002), evaluated the central auditory system maturation based on dipole source modeling of multielectrode long latency AEP recordings. Previous studies have shown that observed patterns of auditory evoked potential depend on the scalp location of the recording electrodes. Dipole source modeling incorporates the AEP information recorded at all electrode locations. AEPs were recorded at 30 scalp electrode locations from 118

subjects between 5 and 20 years of life. Regional dipole source analysis, using symmetrically located sources, was used to generate a spatio-temporal source model of age related changes in AEP latency and magnitude. The regional dipole source model separated the AEPs into distinct groups depending on the orientation of the component dipoles. The sagitally oriented dipole sources obtained 2 AEP peaks, comparable in latency to Pa and Pb of the middle latency responses (MLR). Although, some magnitude changes were marked, latencies of Pa and Pb showed no evidence of age related change. The tangentially oriented sources contained activity comparable to P1, N1b and P2. There were various age related changes in latency and magnitude of the AEPs in the tangential sources. The radially oriented sources contained activity comparable to T-complex, including Ta and Tb that showed only small latency changes with age. In addition, a long latency response labeled TP 200 was observed. Hence, it is possible to distinguish 3 maturation groups; one group reaching maturity at age 6 and comprising the MLR components Pa and Pb: P2 and T-complex. A second group that was relatively fast to mature (50% / year) was represented by N2. A third group was characterized by a slower pattern of maturation with a rate of 11-17% / year and included the AEP peaks P1, N1b and TP200. The results also demonstrated the independence of T-complex components, represented in the radial dipoles, from the P1, N1b and P2 components, contained in tangentially oriented dipole sources. In this report, the authors tried to postulate comprehensively, the developmental trends that could be expected in almost all the AEPs.

Tremblay, Billings and Rohila (2004) studied the effects of age, stimulus complexity and stimulus presentation rate of speech evoked cortical potentials in ten

younger and ten older normal hearing adults. A 1 KHz tone burst as well as a speech syllable was used to elicit the N1P2 complex. Three different interstimulus intervals (ISI) were used (510, 910 and 1510 msec). When stimuli were presented at the medium presentation rate (910msecs), the N1 and P2 latencies were prolonged for older listeners in response to the speech stimulus but not the tone stimulus. These age effects were absent when stimuli were presented at a slower rate (1510msecs). Results, from this study suggest that rapidly occurring stimulus onsets, either within a stimulus or between stimuli, result in prolonged N1 and P2 responses in older adults. This is especially true when processing complex stimuli such as speech. One potential explanation for this age effect might be age related refractory differences in younger and older auditory systems. Refractory issues might in turn affect synchronized neural activity underlying the perception of critical time varying speech cues and may partially explain some of the difficulties older experience during understanding speech.

Hence, it will be more clinically meaningful and useful to develop an electrophysiologic objective measure, like ACC, which in combination with speech evoked ABR and a behavioral assessment task could wholesomely tap the speech perception capacity of an individual. It would be a boon in terms of evaluating prephonologic children and difficult-to-test population. And based on ACC results critical decisions; like candidacy for implants, could be incorporated. Hence, a set of normative data for ACC i.e. an immediate need. To lay a foundation, it will be more appropriate to establish normative data for pediatric and adult population, and delineate the effect of maturation in children. Possibly, on standardization with age appropriate norms the test could be also used for monitory benefit of therapy (or auditory training)

and could hold great scope in rehabilitation. Hence, this study will not only enhance the present research insight, rather will also help validating ACC as a clinical tool of speech perception ability.

CHAPTER 3

METHOD

Subjects: Subjects were divided into 2 groups, viz. Group I, and Group II. Group I included a sample of 30 normal hearing subjects (17 males and 13 females), between 18-35 years of age. Behavioural pure tone thresholds were 15dBHL or less at octave frequencies between 250Hz to 8 kHz (ANSI, 1996). Group II included a sample of 20 normal hearing subjects, with their hearing acuity being 15dB HL or less at octave frequencies between 250Hz and 8 KHz (ANSI, 1996). They were further subdivided into two groups.

II. a. 8.1 to 9 years (6 males and 4 females)

II. b. 9.1 to 10 years (7 males and 3 females).

Instrumentation: A calibrated 0B922 clinical audiometer was used for estimation of pure tone thresholds. Calibrated GSI –33 (version – II) middle ear analyzer was used for Immittance measurements. IHS smart EP version 2.39 evoked potential system was used for recording ACC.

Test Environment: All the experiments were conducted in acoustically treated and electrically shielded room.

Procedure: Pure tone thresholds were obtained in octave intervals between 250Hz to 8000Hz for air conduction and between 250Hz and 4000Hz for bone conduction using modified Hughson-Westlake procedure (Carhart and Jerger, 1959). Tympanometry and reflexometry were carried out to rule out any middle ear pathology.

Subjects were made to relax on a reclining chair. ACC was recorded from 2 channels. The site of electrode placement was prepared with skin preparation gel. Silver Chloride (AgCl) electrodes were placed with conducting gel. The non-inverting electrodes were placed at the vertex (Cz) and ipsilateral mastoid (A2), inverting on the nose tip and common electrode on the forehead (Fpz). It was ensured that the interelectrode impedance was less than 5KΩ. The acoustic change complex (N1P2 complex) was recorded using the test protocol shown in Table 1.

Table 1

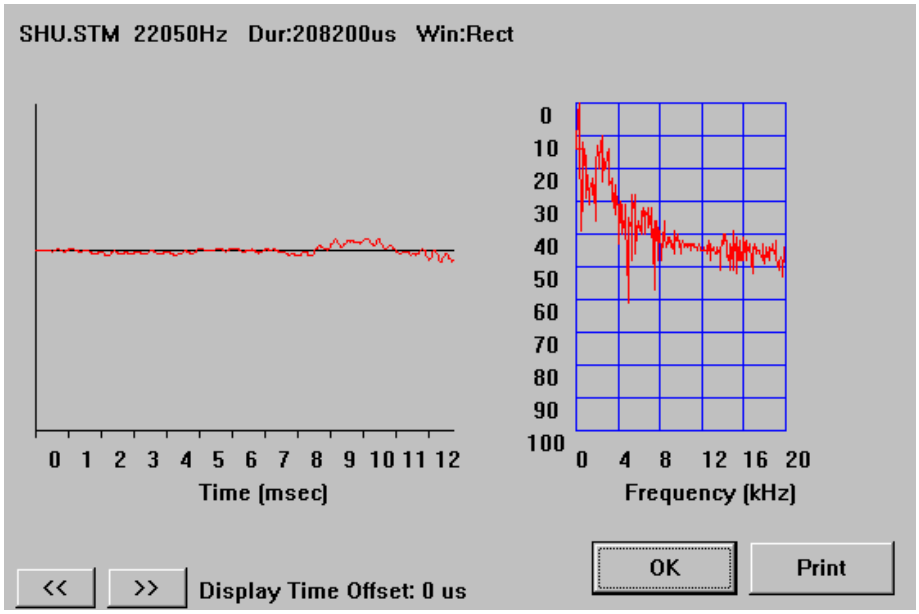
Protocol for recording of ACC (N1P2 complex)

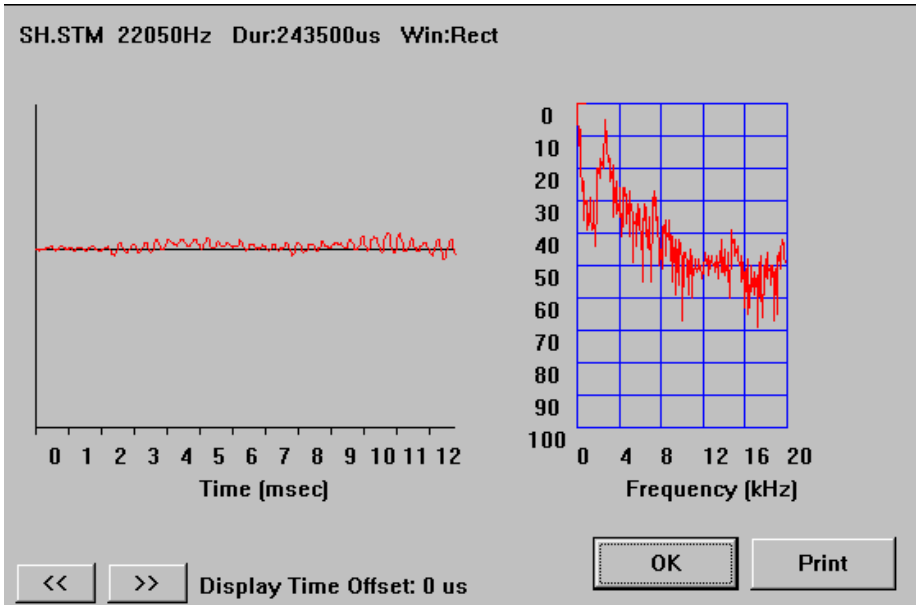
Sl.No	Parameters	Stimuli 1 (|sh|)	Stimuli 2 (|shu|)
1.	Duration of stimuli	243msecs	208msecs
2.	Intensity	80dB nHL	80dB nHL
3.	Transducer	Eartone 3A insert earphones	Eartone 3A insert earphones
4.	Repetition rate	1.1 per second	1.1 per second
5.	Number of samples	200	200
6.	Filter setting	1-30Hz	1-30Hz
7.	Analysis window	400msecs	400msecs
8.	Mode of presentation	Monaural	Monaural

The electrophysiological responses were recorded for speech stimuli, viz. /sh/ and /shu/ individually. The duration of the stimuli was less than 250msecs (|sh|-243msecs, |shu| - 208msecs). The speech stimuli was spoken by an adult and recorded by a unidirectional microphone. The material was scaled using AUDIOLAB software, and then converted to .stm file, using IHS stim conversion software. The presence of a robust N1P2 complex for each of the speech stimuli was confirmed by replicating the responses. The two stimuli, i.e. |shu| and |sh| that were used to elicit an ACC are shown in Figure 1.

Figure 1

Depicts the stimulus waveforms for |shu| and |sh| stimuli used for eliciting an ACC response.




SH.STM 22050Hz Dur:243500us Win:Rect
0 1 2 3 4 5 6 7 8 9 10 11 12
Time (msec)
0
10
20
30
40
50
60
70
80
90
100
0 4 8 12 16 20
Frequency (kHz)
<<
>>
Display Time Offset: 0 us
OK
Print

CHAPTER 4
RESULTS AND DISCUSSION

The ACC (Acoustic change complex) was elicited using two stimuli, viz. /shu/ and /sh/ across adults and children. In children it was elicited across 2 age groups, viz. 8 to 9 year old and 9 to 10 year olds. The latency and amplitude were measured for N1 and P2 peaks. The latency and amplitude values of each group were compared separately across stimuli to measure the effect of spectral transition. Secondly, the difference in relative N1 P2 amplitude across age groups was analyzed to evaluate the maturational trends in ACC responses. To investigate the aims of the study paired sample t-test was used for adults and Wilcoxon matched pair sign rank test was used in children. For across group comparison non-parametric Kruskal Wallis test of significance was done. All statistical analyses were carried out using SPSS software (Statistical package for social sciences, Version 10.0).

ACC in adults

Table 2 shows the mean and standard deviation (SD) values for /sh/ and /shu/ stimuli, in adults. Results are shown for each stimulus separately, for various parameters, viz. N1 peak absolute latency, N1 peak absolute amplitude, P2 peak absolute latency, P2 peak absolute amplitude and N1-P2 amplitude. It was observed that for /shu/ stimuli the latency of N1 ranges from 84 to 128 msec, with a mean of 106.48msec, while amplitude ranges from -4.04 to 3.34 μV with a mean of 0.79 μV. Similarly, the latency of P2 ranges from 132.8 to 199.2 msec, with a mean of 167.2 msec and amplitude ranges between -7.21 to 15.71 μv with a mean of 4.43 μv. For /sh/ stimuli, the latency of N1

Ostroff, Martin and Boothroyd, (1998) reported that in adults, the N1 latency ranged from 92 - 134 msec (Mean=110.79 ms), and amplitude –1.65 to 0.75 μV (Mean=-0.54 μV). But, P2 latency was slightly longer and amplitude lesser in their study. This is probably, due to the difference in stimulus used across the studies.

Paired sample t-test was carried out to investigate if the difference in latency and amplitude for the two stimuli is statistically significant. Table 3 shows the results of paired sample t-test applied across stimuli /sh/ and /shu/ in adults. It is evident from Table 3 that there is a statistically significant difference ($p < 0.01$) for two parameters, viz. N1 P2 amplitude and N1 peak absolute latency across |shu| and |sh| in adults.

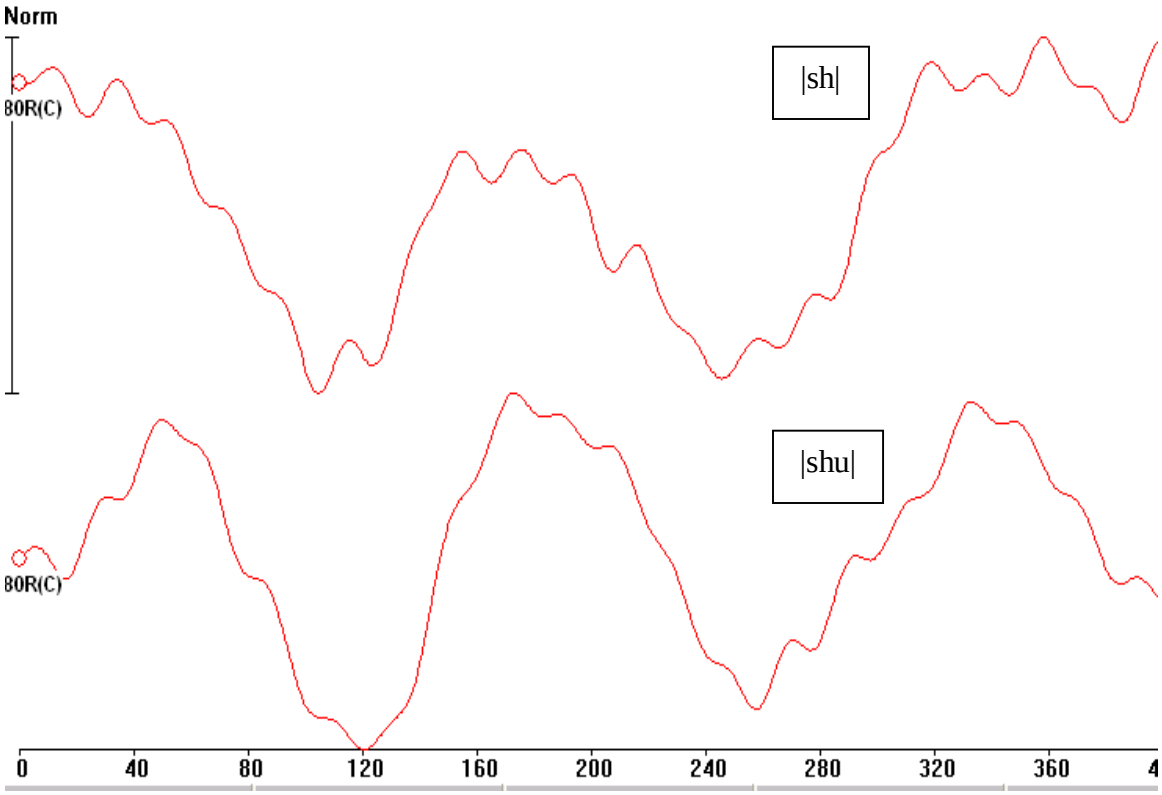
Table 3

Results of paired sample statistics (t-test) in adults (30 subjects).

	Paired Mean	t	df	Sig. (2-tailed)
N1P2 |shu| – N1P2 |sh|	1.3783	3.013	29	0.005**
N1 |shu| – N1|sh| (latency)	-8.48	3.319	29	0.002**
N1 |shu| – N1 |sh| (amplitude)	0.2247	0.226	29	0.822
P2 |shu| – P2 |sh| (latency)	4.8333	1.051	29	0.302
P2 |shu| – P2 |sh| (amplitude)	0.3723	0.665	29	0.511

Figure 2

A sample waveform depicts the classical N1 P2 (or ACC) response that could be noted for /shu/ in comparison to /sh/ stimulus.



The N1-P2 relative amplitude was significantly larger for /shu/ exceeding that of /sh/ by nearly 1 μV. There are inherent spectro-temporal differences as evidenced between the two stimuli, i.e. /shu/ and /sh/ in terms of rate of spectral change, amplitude fluctuations, rate of amplitude fluctuations (or periodicity), rate of formant transitions and energy concentration (Hedrick & Younger, 2003). The obtained results could be justified as an impact of the inherent stimulus characteristics. The demonstration of ACC in response to spectral change, perhaps simply reflects a change of overall synchronous

cortical neuron excitation. Also, it's known that /shu/ stimulus involves a vowel onset preceded by a sibilant. This strongly suggests that the cortical regions activated by the two sounds are not entirely separate, but overlap (Ostroff, Martin and Boothroyd, 1998). And since the net cortical neural excitation decides the response amplitude, a robust N1P2 is elicited.

This can be hypothesized based on previous research. Martin and Boothroyd (2000) obtained ACC in response to changes of amplitude and /or spectral envelope at the temporal center of a three formant synthetic vowel. The spectral change involved an amplitude decrement at one frequency and an amplitude increment at another frequency. They observed that, in the presence of a change of second formant frequency (from |u| to |i|) amplitude increments increased the magnitude of all but amplitude decrements had little or no effect. The resulting ACC may therefore be assumed to reflect a combination of an outset response from one cortical region and an offset response from another. There was no condition under which a small amplitude increment or decrement eliminated or even reduced the amplitude of ACC responses to spectral change. Hence the findings are suggestive of fact that ACC is susceptible to changes in overall spectrum and amplitude. Hence, the findings provide a strong evidence for the dissociation of cortical responses to changes of overall spectrum and amplitude.

The difference in N1P2 amplitude observed in the present study is lesser than reported by Ostroff et. al. (1998). The difference may be due to the complex interplay of various factors like spectral transition, amplitude fluctuations and periodicity. Since the present study did not aim at controlling any of the features, perhaps the aggregate cortical neuron firings are enhanced.

ACC in children:

a) 8-9 year old children

Table 4 depicts the mean and SD values for /sh/ and /shu/ stimuli in 8-9 years old children. It is clear from the table that the latencies of N1 and P2 peaks are in correspondence to the previous reports, like the one reported by Cunningham, Zecker, Nicol and Kraus (2000). Another fact to be noted was that the N1-P2 responses, or rather individual N1 and/or P2 peaks were present only in 5 subjects out of 10 subjects. This shows that LLR was absent in nearly 50% of the subject sample. Two subjects showed N1-P2 response for |shu| and no response fro |sh| stimuli. Only one of the subject, showed an atypical N1-P2 response for /sh/ stimuli, but no LLR could be elicited for /shu/ stimuli for that subject.

Table 4

Amplitudes and latencies in response to /sh/ and /shu/ stimuli for 8-9 year old children will mean and SD.

Parameters	Stimuli	Mean	SD
N1 P2 amplitude in μV	|shu|	6.03	1.04
	|sh|	4.58	2.36
N1 latency In msec	|shu|	93.36	9.75
	|sh|	101.4	9.01
N1 amplitude in μV	|shu|	-2.17	2.82
	|sh|	-0.19	3.08
P2 latency In msec	|shu|	166.8	9.52
	|sh|	154.8	24.28
P2 amplitude in μV	|shu|	3.83	3.25
	|sh|	4.39	3.99

In subjects who showed an LLR pattern, the response patterns are similar to those in adults, suggesting that the underlying neuropsychological mechanisms or functioning is quite similar. However, Wilcoxon signed rank test shows that there is no statistically significant difference ($P > 0.05$) between |shu| and |sh| stimuli across N1 P2 amplitude, N1 amplitude between stimuli, P2 latencies and amplitudes across stimuli. But N1 latency across the two stimuli is found to be significant ($P < 0.05$).

Table 5 shows the results of non-parametric Wilcoxon matched pair signed rank test administered for children in the age range of 8 to 9 years, to evaluate the significance of response across the two stimuli. And, Figure 4 displays a sample ACC response elicited from one of the subjects in 8 to 9 year old group.

Table 5

Results of Wilcoxon Signed rank test administered on 8-9 years old children

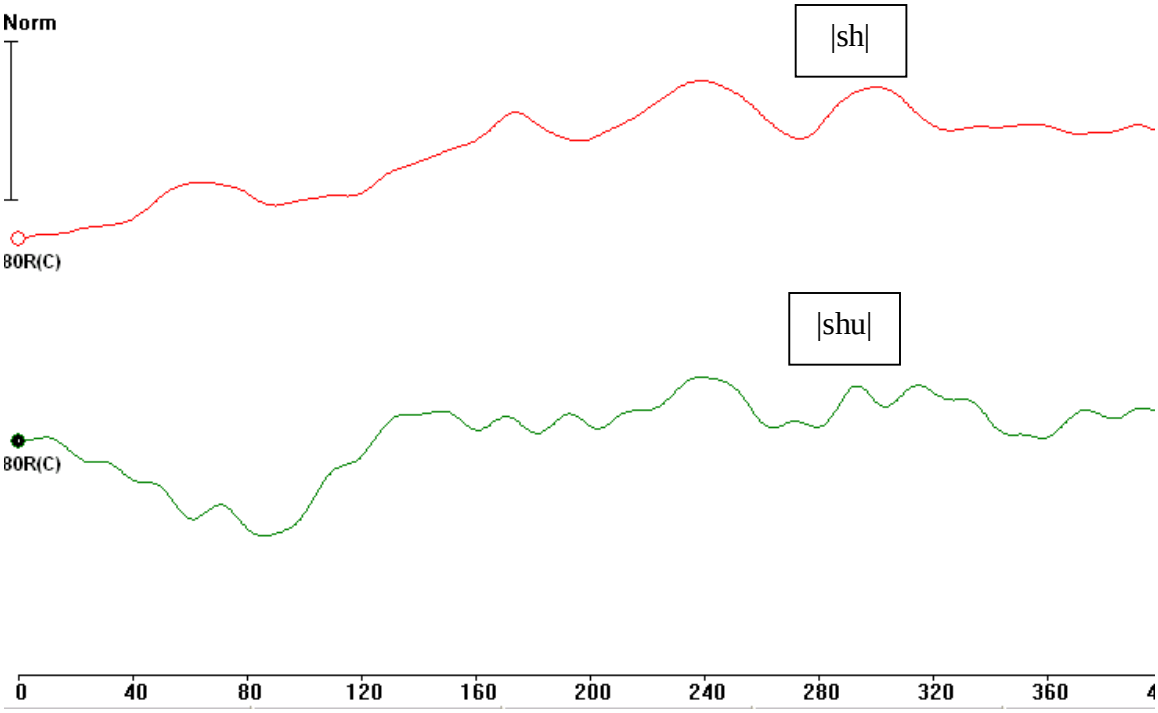
	N1P2 amplitude /shu/-/sh/	N1 latency /shu/-/sh/	N1 amplitude /sh/-/sh/	P2 latency /shu/-/sh/	P2 amplitude /shu/-/sh/
Z	-.674[a]	-2.023[b]	-.911[b]	-1.219[a]	-674[b]
Aysmp. Sig (2 tailed)	.500	.043*	.345	.223	.500

a. Based on positive ranks

b. Based on negative ranks

Figure 3

A sample waveform depicts the classical N1 P2 (or ACC) response that could be noted for /shu/ in comparison to /sh/ stimulus in Group II a.



The obtained results could be due to the fact that ALLR is not completely matured in children. These results are in consonance to those as reported in earlier studies. Cunningham, Zecker, Nicol and Kraus (2000), have stated that a maturational progression is seen in the appearance of N1 during school years. This statement is based on a measured increased in N1 presence from 45%, 50%, 55% and 60% of subjects in the age range of 5 to 7 years, 8 to 10 years, 11 to 12 years and 13 to 15 years respectively. They also reported that N2 was measured in 100% of the population. They have reported that the greatest degree of change for latency and amplitude occurred in the early school age years between 5 and 12 years and N1 latency appeared adult like by 13 to 15 years of

age. Ponton, Eggermont, Khosla, Kwong and Don (2002), evaluated the auditory system maturation and reported of three maturational groups: one group reaching maturity at 6 years of age and comprising of MLR components P1, Pb; P2 and T complex. A second group that was relatively fast to mature (50% per year) was represented by N2. A third group was characterized by a slower pattern of maturation with a rate of 11 to 17 % per year and included AEP peaks P1 and N1b.

To summarize the possible explanations for the enhanced latency of N1, probably, the N1 latency is just variable across the two stimuli, due to inherent differences in spectro-temporal configuration or due to restricted size of the sample. Or else, it is also possible that hierarchically as the cortical of generators mature over age atypical patterns of responses can be seen over the maturational course (Ceponiene, Rinne and Naatanen, 2002) and hence the results. Figure 5 shows the error bar diagram for latency and amplitude values of N1 and P2 peaks in 8 to 9 year old children.

(b) 9-10 year old children

Table 6

Amplitudes and latencies of N1 and P2 peaks and Z values of Wilcoxon singed rank test

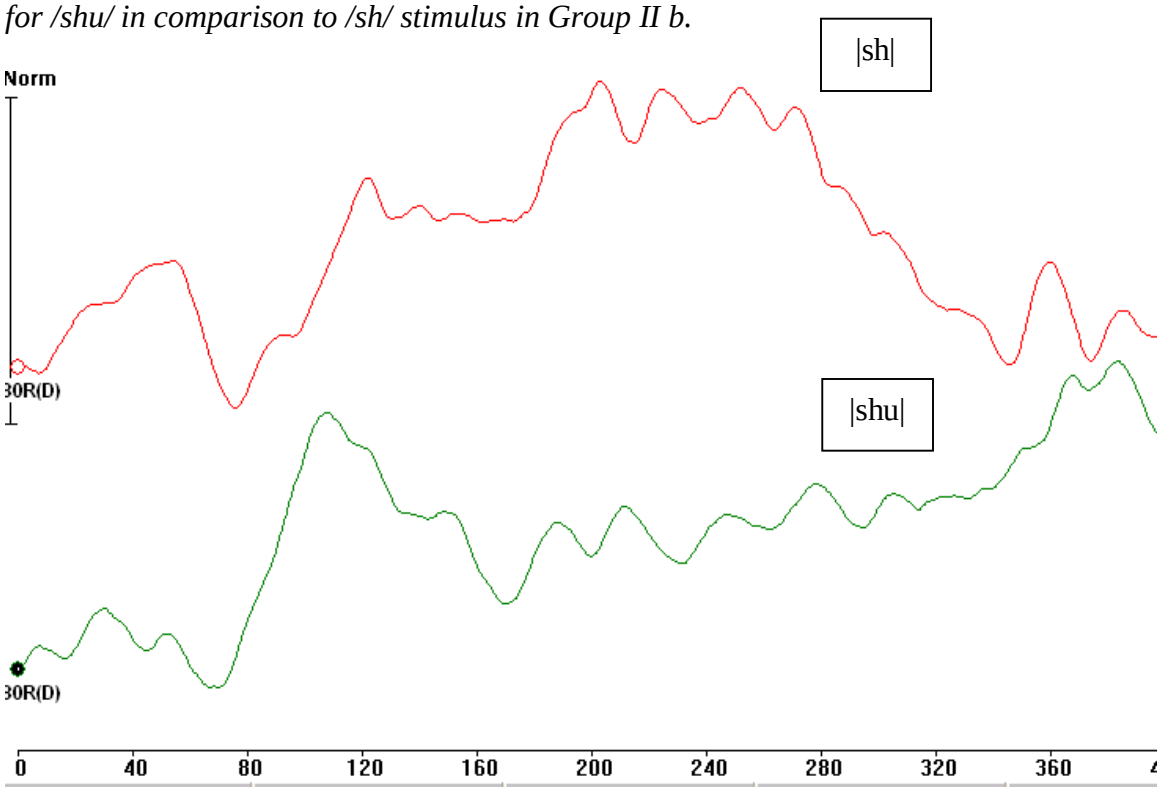
	Stimuli	Mean	SD	Z value	Sig (2 failed)
N1 P2 amplitude in μV	|shu| |sh|	5.27 3.23	2.46 2.26	-1.753[a]	.080
N1 latency in msec	|shu| |sh|	87.84 94.88	21.83 23.89	-0.944[b]	.345
N1 amplitude in μV	|shu| |sh|	-0.02 -0.25	1.78 1.62	-.674[b]	.500
P2 latency in msec	|shu| |sh|	136.64 135.84	29.07 40.66	-0.135[a]	.893
P2 amplitude in μV	|shu| |sh|	5.15 2.98	3.86 2.46	-1.753[a]	.080

Table 6 shows the mean and standard durations of N1 and P2 peaks and results of Wilcoxon signed rank test. The results reveal a decrease in absolute latency values of N1 and P2 peaks, when compared to those of 8-9 years but, it is more than those observed for adults. This once again clearly demonstrates the developmental maturational trend, as evident across age groups. The N1-P2 peaks progressively seems to mature from 8-9 years to 9-10 years, and in due course of time appears to approximate adult latency, amplitude and morphology characteristics.

As evidenced for the other groups, the N1P2 amplitude for /shu/ was comparatively more than that for /sh/ stimuli. This could once again be attributed to the stimulus characteristics, which incorporates a change in spectral envelopes. However, Wilcoxon matched pair sign rank test clearly shows that there is no statistical difference ($p > 0.05$) between the various N1 and P2 parameters (i.e. amplitude and latency values). Again, as in the previous age group, out of 10 subjects, ACC responses could be elicited only for 5 subjects with accuracy. Further, only a couple of subjects evidenced presence of N1P2 for /shu/ stimuli, while it was absent for /sh/ stimuli. This could be due to the fact that the peaks in children are composed of differentially weighed components and it shows the gradual onset of adult-like response morphology. In due course of time, perhaps the higher-level auditory system will mature and strengths of various anatomic generators will get optimized. So, the enhanced amplitudes could be hypothesized to be due to disproportionate firing of critical neurons during maturational course. Figure 6 shows a sample ACC response waveforms obtained for children in Group II b. across stimuli.

Figure 4

A sample waveform depicts the classical N1-P2 (or ACC) response that could be noted for /shu/ in comparison to /sh/ stimulus in Group II b.



Comparison across adults and children:

Thus, the results showed that there was an increase in N1-P2 amplitude for |shu| stimuli when compared to that of |sh| for all the groups. But the difference was statistically significant only in adults. It is possible that the difference was not statistically significant in children due to small sample size or due to the effect of maturational factors. Further analysis was carried out to check if there is significant change in the difference in N1-P2 complex for the two stimuli across age. The result of across group

comparison is shown in Table 7.

Table 7

Summarizes the N1P2 response amplitudes (normative values) across children and adults.

Groups	N1P2 response amplitude mean	SD
Adults	1.38	2.50
8-9 years	1.44	2.81
9-10 years	2.05	1.74

Table 7 gives the normative values for ACC responses in children and adults. From the visual inspection of Table 7, it can be observed that the ACC responses (or N1-P2 response differences) across stimuli are roughly the same for all the three age groups. But, the mean values are slightly higher for the children subset than that of adults, especially Group II b. (9 – 10 year olds). These results are slightly discrepant from previous published reports, like, the one as reported by Cunningham, Nicol, Zecker and Kraus (2000). They reported that absolute latencies of P1 and N2 peaks decrease progressively, while absolute latency of N1 peak increases with age (from 5 to 78 years). This discrepancy could be perhaps, due to smaller sample size of both the children sub-groups, although the groups are quite homogenous. Table 8 shows the results of non-parametric Kruskal Wallis test carried out for performing across group comparison for the difference in N1 P2 amplitudes (or ACC).

Table 8

Results of Kruskal Wallis test for significance across children and adults.

	Difference
Chi.square Of Asymp. Sig.	0.777 2 .678

From Table 8 and Figure 5, it is very clear that there is no statistically significant difference ($p > 0.05$) for the difference in N1-P2 amplitudes (or Acoustic change complex), across children and adults. Although there is genuine amplitude difference that is evident from the raw data across the three groups, but it is not to the extent of being significant. Therefore the ACC response across the three age groups does not seem to differ much. Perhaps that could be interpreted as, the perception of spectral change does not differ much or rather is mature (or reflected electrophysiologically) by 8-9 years of age. But, the major pre-requisite for the elicitation of this spectral change in children is the presence or rather maturation of LLR. Hence, the ACC is elicited in all three age groups, but with a lesser consistency in children.

The results of the present study, confirms that a peripheral mechanism is relaying the information, about acoustic change in an ongoing stimulus. The ability of the peripheral mechanism to respond to acoustic changes in one of the pre requisites for higher-level speech perception. The present findings thus suggest that Acoustic change complex (ACC); can be used as a clinical tool in the assessment of speech perception

ability in adults. But, the clinical applicability of ACC is limited by fact that a child should have attained LLR maturation as ACC could not be reliably and consistently elicited in younger age group. This could be due to the known immaturity of cortical obligatory potentials. Although, ACC seems to be a practical tool in the investigation of difficult-to-test population; a better insight could be obtained by evaluating this on a larger group of subjects.

CHAPTER 5

SUMMARY AND CONCLUSIONS

The N1-P2 complex is best known as an onset response (Hilyard & Picton, 1978; Naatanen, 1992; Naatanen & Picton, 1987; Onishi & Davis, 1968; Pantev, Eulitz, Hampton, Ross & Roberts, 1996). It has been shown that the N1P2 complex can be elicited by changes in an ongoing acoustic stimulus, and will result in an increase in the amplitude of the negative-positive complex and is termed as – Acoustic change complex (ACC), by Martin and Boothroyd (1999). In appearance and timing, the ACC is very similar to the conventional N1P2 complex.

Many electrophysiological measures such as speech evoked ABR, P300, MMN, CNV, have been researched to study speech perception. Each potential taps a specific aspect of speech sound processing in the higher level auditory system. Since, ACC has been elicited using a stimulus having a change in spectral envelope, it is believed that ACC has a potential utility as a tool for assessing the perception of spectral change (Ostroff, Martin & Boothroyd, 1998). It could, thus, possibly assess neurophysiological processing at cortical level and in combination with other tools like speech elicited ABR; constitute a comprehensive objective speech perception assessment battery in pre-phonologic children and difficult-to-test population.

There is dearth of literature regarding the administration and validation of this tool in children. A serious limitation that could influence recording the ACC in children would be the critical aspect of LLR (Late latency responses) maturation. Hence, the present study was designed with the following aims,

I. Establish normative data on ACC for |shu| and |sh| stimuli in pediatric population and in adults,

II. Investigate the effects of age on ACC.

Subjects included for the study were (Group I) 30 normal hearing adults (17 male, 13 female) and 20 normal hearing children (Group II). The adults were in the age range of 18 – 35 years. The children were further sub-divided into two groups:

a) Group II a. - 10 children (6 male, 4 female) between 8 to 9 years.

b) Group II b. - 10 children (7 male, 3 female) between 9 to 10 years.

The ACC was recorded with non-inverting electrode at vertex (Cz), inverting at nose-tip and common electrode on the forehead (Fpz). ACC was recorded by eliciting LLR for both |shu| and |sh| (naturally produced, truncated and scaled) stimuli individually, using IHS smart evoked potential system (version 2.39).

Results revealed that ACC could be recorded in all adult subjects. However, the results were slightly aberrant in children, and ACC was found to be recordable in only 50 % of the population. The statistical analysis was done using SPSS software, and the following conclusions were drawn from the analysis,

1. N1-P2 responses for both |shu| and |sh| stimuli are elicited in adults. There is a statistically significant difference in N1P2 amplitudes between the two stimuli, indicative of presence of ACC response, in adults.
2. In 8 to 9 year old and 9 to 10 year old children, LLR waveform could be elicited in only 50 % of the subject population. Although, there was a difference in N1-P2 amplitude across the stimuli indicating the presence of ACC, it was not statistically significant.

3. The difference in N1-P2 response amplitudes across stimuli is not significant across children and adults. Hence, ACC can be used as an electrophysiological tool for the perception of spectral change in adults and children if LLR is present.

The study has the following implications:

a) ACC could have immense clinical value in objectively assessing the candidature issue for pre-phonologic children planning to undergo implantation.
b) ACC could be a useful tool in terms of hearing aid selection and thus facilitate aural rehabilitation.
c) ACC could also evolve into a verification and validation procedure to assess post-amplification benefits, by objectively evaluating improvement in speech perception.

REFERENCES

Aaltonen, O., Niemi, P., Nyrke, T., & Tuhkanen, M. (1987). Event-related potentials and the perception of a phonetic continuum. *Biological psychology*, 24(3), 197-207. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

American National Standards Institute. (1996). Specification for audiometers. (S3.9 – 1996). New York: ANSI.

Beynon, A.J. Snik, .A.F. & Van der Broek, P. (2002). Evaluation of cochlear implant benefit with auditory cortical evoked potentials. *International Journal of Audiology*, 41(7), 429-435.

Boothroyd, A. (1971) Developmental factors in speech perception. *International Journal of Audiology*, 9, 30-38.

Boothroyd, A. (1991). Assessment of speech perception capacity in profoundly deaf children. *American Journal of Otology*, 12 (Suppl.), 67 – 72.

Boothroyd, A. (1997). Auditory development of the hearing child. *Scandinavian Audiology Supplement*, 46, 9-16.

Cacace, A.T., Dowman, R., & Wolpaw, J.R. (1988). T complex hemispheric asymmetries: Effects of stimulus intensity. *Hearing research*, 34(3), 225-232. Abstract retrieved February 20, 2005 from http://www.Entrezpubmed.com/.

Carhart, R. & Jerger, J. (1959). Some relations between normal hearing for pure tones and for speech. *Journal of Speech and Hearing research*, 2(2), 126-140.

Ceponiene, R., Lepisto, T., Alko, P., Aro, H., & Naatanen, .R. (2003). Event-related potential indices of auditory vowel processing in 3 year old children. *Clinical*

Neurophysiology, 114(4), 652-661. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Ceponiene, R., Rinne, T., & Naatanen, R. (2002). Maturation of cortical processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113(6), 870-872. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Connolly, J.F. (1993). The influence of stimulus intensity, contralateral masking and handedness on the temporal N1 and T complex components of the auditory N1 wave. *Electroencephalography and Clinical Neurophysiology*, 86(1), 58-68. Abstract retrieved February 20, 2005 from http://www.Entrezpubmed.com/.

Csepe, V., Karmos, G., & Molnar, M. (1987). Evoked potential correlates of stimulus deviance during wakefulness and sleep in cat– animal model of mismatch negativity. *Electroencephalography Clinical Neurophysiology*, 66(6), 571 – 578. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Cunningham, J., Nicol, T., Zecker, S., & Kraus, N. (2000). Speech evoked neurophysiologic responses in children with learning problems: development and behavioral correlates of perception. *Ear and Hearing*, 21(6), 554-568.

Davis, P.A. (1939). Effects of acoustic stimuli on the waking human brain. *Journal of Neurophysiology*, 2, 494-499. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Fischer, C., Blanc, A., Manguiere, F., & Courjon, J. (1981). Diagnostic value of brainstem auditory evoked potentials. *Revue de Neurologie*, 137, 229-240. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Furui, S. (1986). On the role of spectral transition for speech perception. *Journal of Acoustical Society of America*, 80(4), 1016-1025.

Geers, A.E., & Moog, J.S. (1994). Effectiveness of cochlear implants and tactile aids for deaf children: The Sensory aids study at Central Institute for the Deaf. *Volta Review*, 96, 1- 232.

Goodin, D.S., Squires, K.C., Henderson, B.H., & Starr, A. (1978). Age–related variations in evoked potentials to auditory stimuli in normal human subjects. *Electroencephalography and Clinical Neurophysiology*, 44(4), 447 –458. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Goto, H., Adachi, T., Utsounomiya, T., Nakano, H., & Chin, K. (1978). EEG evoked response, P300 and CNV during processing of sentence information: clinical application. *TIT Journal of Life Sciences*, 8(1-2), 55-58. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Gronen, P.A., Beynon, A.J, Snik, A.F., & Van der Broek, O. (2001). Speech-evoked cortical potentials and speech recognition in cochlear implant users. *Scandinavian Audiology*, 30(1), 31-40.

Groenen, P., Snik, A., & Van der Broek, P. (1996). On the clinical relevance of match negativity: results from subjects with normal hearing and cochlear implant users. *Audiology Neurotology*, 1(2), 112-124.

Hagoort, P., Brown, C.M., & Groothusen, J. (1993). The syntactic positive shift (SPS) as an ERP measure of syntactic processing. *Language and Cognitive Processes*, 8, 439-483.

Hagoort, P., & Brown, C.M. (2000). ERP effects of listening to speech compared to reading: The P600/SPS to syntactic violations in spoken sentences and rapid serial visual presentation. *Neuropsycholigia*, 38, 1531-1549.

Hall, J.W. (1994). *Handbook of Auditory Evoked Responses* Boston, Allyn and Bacon.

Hilyard, S.A., Mangun, G.R., Woldorff, M.G., & Luck, S. (1995). Neural systems mediating selective attention. In M. Gazzaniga (Ed.), *Cognitive neuroscience*. (pp. 665-681). New York: Plenium.

Hilyard, S.A. & Picton, T.W. (1978). ON and OFF components in the auditory evoked potential. *Perception and psychophysics*, 24, 391 – 398.

Jacobson, G.P., & Gans, D.P. (1981). The contingent negative variation an indicator of speech discrimination difficulty. *Journal of Speech and Hearing research*, 24(3), 345-350.

Johnstone, S.J., Barry, R.J., Anderson, J.W., & Coyle, S.F. (1996). Age–related changes in child and adolescent event-related potential component morphology, amplitude, and latency to standard and target stimuli in an auditory oddball task. *International Journal of Psychophysiology*, 24(3), 223 –238. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Josey, A. (1985). Auditory brainstem response in site of lesion testing. In J. Katz (ed.), *Handbook of Clinical Audiology*, Williams and Wilkins, Baltimore, MD., pp. 534–548.

Kaukoranta, E., Hari, R. & Loumasmaa, O.V. (1987). Responses of the human auditory cortex to vowel onset after fricative consonants. *Experimental Brain Research*,

69(1), 19-23. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Kaukoranta, E., Sams, M., Hari, R, Hamalainen, M., & Naatanaen, R. (1989). Reactions of human auditory cortex to a change in duration. *Hearing research*, 41(1), 15-21. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Khaladkar, A.A., Kartik, N., & Vanaja, C.S. (2005). Speech burst ABR: A bronze standard for perceptual deficits in sensorineural hearing loss. Scientific paper presented in XXXVII National conference of Indian Speech and Hearing Association, Indore.

Kileny, P.R., Boerst, A. & Zwolan, T. (1999). Cognitive evoked potentials to speech and tonal stimuli in children with implants. *Otolaryngology Head and Neck Surgery*, 117, (3 pt 1), 161-169.

King, C., McGee, T., Rubel, E.W., Nicol, T., & Kraus, N. (1995). Acoustic features and acoustic changes are represented by different central pathways. *Hearing research*, 85(1-2), 45-92.

King, C., Warrier, C.M., Hayes, E. & Kraus, N. (2002). Deficits in auditory brainstem pathway encoding of speech sounds in children with learning problems. *Neuroscience letters*, 319(2), 111- 115.

Kushnerenko, E., Ceponiene, R., Balan, P., Fellman, V., Huotilainen, M., & Naatanen, R. (2002). Maturation of the auditory event related potentials during the first year of life. *Neuroreport*, 13(1), 47-51. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Kutas, M., & Hilyard, S. (1980). Reading senseless sentences: Brain potentials reflect semantic incongruity. *Science,* 207, 203-205.

Laipply, E.M. (1990). *Chronological age effects on the audio/visual perception of phonologically significant speech feature contrasts*. Unpublished Master's thesis University of Florida, Tampa.

Maiste, D.C., Weins, A.S., Hunt, M.J., Scherg, M. & Picton, T.W. (1995). Event-related potentials and categorical perception of speech sounds. *Ear and Hearing,* 16(1), 68-90.

Martin, B.A., & Boothroyd, A. (1999). Cortical, auditory, event-related potentials in response to periodic and aperiodic stimuli with the same spectral envelope. *Ear and Hearing*, 20(1), 33 – 44.

Martin, B.A., & Boothroyd, A. (2000). Cortical, auditory, evoked potentials in response to changes in spectrum and amplitude. *Journal of Acoustical Society of America*, 107(4), 2155 – 2161.

Musiek, F.E. (1991). *Auditory evoked responses in site–of–lesion assessment.* In W.F. Rintelmann (Ed.), Hearing assessment, 2nd edition, Pro-Ed, Austin, TX, pp. 383-427.

Naatanen, R., Gaillard, A.W.K., & Mantysalo, S. (1978). Early selective attention effect on evoked potential reinterpreted. *Acta Psychologica*, 42, 313 – 329.

Naatanen, R. (1992). *Attention and Brain function.* Hills dale, New Jersey: LawrenceErlbaum Associates.

Naatanen, R. & Picton, T.W. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, 24, 375 – 425.

Neville, H.J., Nicol, J.L., Barss, A., Forster, K.I., & Garett, M.F. (1991). Syntactically based sentence processing classes: Evidence from event-related brain potentials. *Journal of Cognitive Neuroscience*, 3, 141-165.

Onishi, S. & Davis, H. (1968). Effect of duration and rise time of tone bursts on evoked V potentials. *Journal of Acoustical Society of America*, 44, 582-591.

Osterhout, L., & Holcomb, P.J. (1993). Event-related potentials and syntactic anomaly: Evidence of anomaly detection during the perception of continuous speech. *Language and Cognitive Processes*, 8, 413-437.

Ostroff, J.M., Martin, B.A., & Boothroyd, A. (1998). Cortical evolved responses to spectral change within a syllable. *Ear and Hearing*, 19, 290 – 297.

Ostroff, J.M. (1999). "*Parametric study of the acoustic change complex to synthetic vowel stimuli as a measure of peripheral auditory discrimination capacity*". Unpublished doctoral dissertation, Graduate Centre of The City University of New York.

Pantev, C., Eulitz, C., Hampton, S., Ross, B.G., & Roberts, L.E. (1996). The auditory evoked "off" response: Sources and comparison with the "on" and "sustained" responses. *Ear and Hearing*, 17, 255- 265.

Pantev, C., Luktenhoner, B., Hoke, M., & Lehnertz, K. (1986). Comparison between simultaneous recorded auditory-evoked magnetic fields and potentials elicited by

ipsilateral, contralateral, and binaural tone burst stimulation. *Audiology*, 25, 54-61.

Picton, T.W. (1995). The neurophysiological evaluation of auditory discrimination. *Ear and Hearing*. 16, 1-5.

Polich, J. (1989). P300 for a passive auditory paradigm. *Electroencephalography and Clinical Neurophysiology*, 74, 312-320. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Ponton, C., Eggermont, J.J., Khosla, D., Kwong, B., & Don, .M. (2002). Maturation of human central auditory system activity separating auditory evoked potentials by dipole source modeling. *Clinical Neurophysiology*, 113(3), 407-420 Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Ponton, C.W., Eggermont, J.J., Kwong, B., & Don, M. (2000). Maturation of the human control auditory system: evidence from multi channel evoked potentials. *Clinical Neurophysiology*, 111(2), 220-236. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Reddy, S., Kumar, U.A. & Vanaja, C.S. (2004). Characteristics of ABR evoked by speech bursts. Scientific paper presented at the XXXVI National Conference of Indian speech and Hearing Association, Mysore.

Rohrbaugh, J.W., & Gaillard, A.W.K. (1983). Sensory and motor aspects of contingent negative variation. In A.W.K. Gaillard & W. Ritter (Eds.), *Tutorials in event-related potential research: Endogenous components* (pp. 269 – 310). Amsterdam: North- Holland Publishing Company.

Salamy, A., Eggermont, J., & Eldredge, L. (1994). *Neurodevelopment and applications in evoked auditory potentials*. Boston, Allyn and Bacon.

Sharma, A., Kraus, N., McGee, T.J. & Nicol, T.G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant- vowel syllables. *Electroencephalography and Clinical Neurophysiology*, 104 (6) 540-545. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Sharma, A., Marsh, C.M., & Dorman, M.F. (2000). Relationship between N1 evoked potential morphology and the perception of voicing. *Journal of Acoustical Society of America*, 108(6), 3030 –3035.

Sharma, A., & Dorman, M.F. (1999). Cortical auditory evoked potential correlates of categorical perception of voice–onset time. *Journal of Acoustical Society of America*, 106 (2), 1078 – 1083.

Sharma, A. Kraus, N., McGee, T., Carell, T., & Nicol, T. (1993). Acoustic versus phonetic representation of speech as reflected by the mismatch negativity event-related potential. *Electroencephalography and Clinical Neurophysiology*, 88 (1), 64-71. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Shestakova, A., Huotilainen, M., Ceponiene, R. & Natanenen R. (2003). Event-related potentials associated with second language learning. *Clinical Neurophysiology*, 114(8), 1507-1512.

Steinschneider, M. Schroeder, C.E., Arezzo, J.C., Vaughan, H.G. Jr. (1995). Physiologic Correlates of the voice onset time boundary in primary auditory cortex (A1) of the awake monkey: temporal response patterns. *Brain Language*, 48(3), 326–340.

Sutton, S., Braren, M., Zubin, J., & John, E.R. (1965). Evoked potential correlates of stimulus uncertainty. *Science*, 150, 1187-1188. Abstract retrieved February 10, 2005 from http://www.Entrezpubmed com/.

Tremblay, K.L., Billings, C., & Rohila, N. (2004). Speech evoked cortical potentials: effects of age and stimulus presentation rate. *Journal of American Academy of Audiology*, 15(3), 226 –237.

Tremblay, K.L., Freisen, L., Martin, B.A., & Wright, R. (2003). Test- retest reliability of cortical evoked potentials using naturally produced speech sounds. *Ear and Hearing*, 24(3), 225 – 232.

Tyler, R.S. (1993). Speech Perception by Children. In R.S. Tyler (Ed.), *Cochlear implants audiological foundations* (pp.191-296). San Diego: Singular publishing Group.

Werner, L.A., & Rubel, E.W. (1992). Eds: *Developmental psychoacoustics.* Washington, D.C.: APA.

Wolpaw, J.R., & Penry, J.K. (1975). A temporal component of the auditory evoked response. *Electroencephalography and Clinical Neurophysiology*, 39, 609-620. Abstract retrieved February 20, 2005 from http://www.Entrezpubmed.com/.

Wolpaw, J.R., & Penry, J.K. (1978). Effects of ethanol, caffeine and placebo on the auditory evoked response. *Electroencephalography and Clinical Neurophysiology*, 44, 568-574. Abstract retrieved February 20, 2005 from http://www.Entrezpubmed.com/.