CORTICAL AUDITORY EVOKED RESPONSES TO COMPLEX SPEECH STIMULI IN AUDITORY NEUROPATHY SPECTRUM DISORDERS

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A Dissertation Submitted in Part Fulfilment of Final Year

Master of Science (Audiology)

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June – 2011

Certificate

This is to certify that this dissertation titled ***"Cortical evoked responses to complex speech stimuli in Auditory Neuropathy Spectrum Disorders"*** is a bonafide work of the student with Registration No: 09AUD032 submitted in part fulfilment for the degree of Master of Science (Audiology). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any diploma or degree.

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Certificate

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DECLARATION

This is to certify that this dissertation titled ***"Cortical evoked responses to complex speech stimuli in Auditory Neuropathy Spectrum Disorders"*** is the result of my own study under the guidance of **Mr. Vijay Kumar Narne Ph.D,** Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier to any other university for the award of any diploma or degree.

Mysore **Register No. 09AUD032**

June, 2011

This work is dedicated to

the God almighty

for showering me

with all HIS love

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Chapter 1

Introduction

Auditory Neuropathy Spectrum Disorder (ANSD) has been described as a hearing disorder characterized by abnormal auditory nerve functioning in presence of normal cochlear receptor hair cell activity (Starr, Picton, Sininger, Hood, & Berlin 1996). Hearing sensitivity in individuals with ANSD may range from normal hearing to profound hearing loss while approximately 60 to 70% of individuals have speech identification scores well below the identification scores estimated from their pure-tone thresholds (Zeng et al., 2005; Sininger & Oba, 2001). A series of studies have revealed that temporal processing is severely affected in these individuals (Zeng et al., 1999, 2001, 2005; Kraus et al., 2000). Objective measures of temporal resolution are hence required to correlate findings with behavioural measures for a better understanding of the auditory system in ANSD subjects.

Though Auditory Brainstem Responses (ABR) have been reported to be absent or grossly abnormal in cases with ANSD, cortical potentials have been reported in most of these cases indicating the preservation of some useful auditory capabilities (Starr, Picton, Sininger, Hood & Berlin, 1996; Kraus et al., 2000). The latency and amplitude of cortical potentials showed a significant correlation with open set speech perception abilities and the absence of cortical potentials indicates extremely poor speech perception abilities (Narne & Vanaja, 2008; Zeng et al., 2009; Vanaja & Manjula, 2004). Recent research has also supported the fact that electrophysiological measures like cortical potentials can be used for objective assessment of temporal processing in normal hearing, sensorineural hearing loss as well as in subjects with ANSD (Michalewski, Starr, Nguyen, Kong & Zeng, 2005;Michalewski, Starr, Zeng,

& Dimitrijevic, 2009). So, cortical potentials can be used to obtain important information regarding severity and management options for the ANSD population.

A cortical potential which can be utilized to understand the processing of the speech stimulus across a syllable is the Acoustic Change Complex (ACC). It is a complex similar to the P1-N1-P2 and is elicited by a change during an otherwise steady-state sound (Martin & Boothroyd, 1999). This complex representation was demonstrated in response to the transition from fricative to vowel in a naturally produced syllable by Ostroff & Martin, (1998). ACC is also reported for the detection of changes in formant transitions, amplitude change, changes of spectral envelope/ periodicity in normal hearing population (Ostroff & Martin, 98) and has been demonstrated in the sensorineural hearing loss population also (Martin, Tremblay & Korczak, 2008). This study aims to utilise the ACC complex as a tool to study the cortical representation of two consonant-vowel pairs: a fricative-vowel combination (su) and an affricate-vowel combination (chu). Since the stimuli used are natural, the consonant durations differ appropriately with /s/ having a longer duration and /ch/ having a shorter duration. This difference, along with differing envelope, fine structure and spectral aspects is bound to result in differing cortical representations. Further, an investigation into the interaction between the consonant and the vowel is attempted.

Need for the study

Speech perception in ANSD is significantly worse than what would be predicted from their pure tone average. So, understanding speech perception in ANSD population is of significant clinical and research interest. Electrophysiological investigations, though hampered by the absence of the brainstem responses, are

possible through cortical potentials. The studies done by Rance et al., (2004), Vanaja & Manjula (2004) and Dimitrijevic et al., (2010) have provided the evidence that individuals with ANSD who demonstrate cortical potentials show better speech perception scores and show benefit with aiding. The presence of ACC would indicate that the subjects could make use of the broad envelope change or the spectral (periodicity) change in detecting a transition from one phoneme to another within a syllable. Currently, there is no other objective tool which can reveal their ability to perceive envelope/spectral change. Hence, this is a preliminary attempt to examine whether the syllables /su/ and /chu/ elicit the ACC component in ANSD subjects.

Aim of the study

The aim of the study is to investigate the representation of complex speech syllable at the cortical level in ANSD and normal population.

Objectives of the study

- To determine the nature of pattern of activation produced by the cortical neurons in response to acoustic change contained in the syllables in individuals with Auditory Neuropathy Spectrum Disorder

- To investigate the relation between behavioral speech identification scores and the cortical potentials-Long Latency Responses (LLR) and the Acoustic Change Complex (ACC)

Chapter 2

Review of Literature

Auditory Neuropathy Spectrum Disorder (ANSD) has been described as a condition with abnormal retro outer hair cell functioning, that is, cochlear amplification function is normal, but neural transmission in the auditory pathways is disordered. It was described by Starr et al. 1991 in one subject and this was followed by a report on a series of subjects with similar symptoms (Starr, Picton, Sininger, Hood & Berlin, 1996). Since then, much research has gone into understanding the nature of this condition.

2.1 Clinical profile of patients with Auditory Neuropathy Spectrum Disorder

1. *Onset and Course*

Sininger and Oba (2001) studied a group of 59 individuals with auditory neuropathy and reported a mean age of onset of 9 years. 75 % of their patients were less than 10 years of age when the first symptom of auditory neuropathy was seen. Some subjects develop the ANSD condition in adolescence or early adulthood. The course is quite unpredictable and the condition may stay the same, resolve, fluctuate or worsen over time.

2. *Prevalence*

The estimates vary from roughly 1% (Foerst et al., 2006) to 10% in schools for the deaf (Berlin et al., 2000 ; Lee et al., 2001 ; Cheng et al., 2005) and between 10% in newborns (Sininger, 2002) and 40% in hearing-impaired NICU graduates (Rea & Gibson, 2003). Kumar and Jayram (2006) report a prevalence of around 0.53% among adults with sensory- neural hearing loss in India.

2.2 Pathophysiology

Based on their previous research (Starr et al., 1996), Starr and Sininger (2001) hypothesized the following explanation to describe the neurophysiological characteristics of individuals with ANSD. Figure 2.1 explains the different neurological manifestations and their results. If demyelination affects all the auditory nerve fibres to the same degree, then transmission through all fibres will be slowed down, and amplitude of the compound action potential will be unaffected despite slowing of conduction velocity (Figure 2.1, second column). On the other hand, if the extent of slowing varies from one fibre to the next, then the amplitude of action potential becomes small and smeared (Figure 2.1, third column). This smeared temporal representation of the acoustic stimulus may influence auditory perception that is dependent on temporal cues. If the number of the nerve fibres are reduced (fourth column), the synchrony is not affected, but resultant compound action potential is reduced in amplitude. The greatest effect is produced when the number of auditory nerve fibres is reduced and they are irregularly demyelinised (column 5).

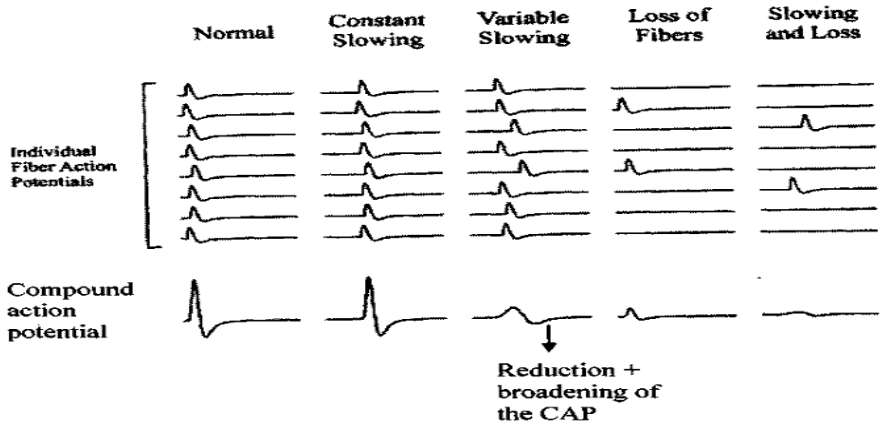

Figure 2.1. Action potential of individual fibers and resultant compound action potential. (From Auditory neuropathy, A new perspective on hearing disorders by Sininger and Starr , 2001).

2.3 Audiological Profile

The ANSD subject group is highly heterogeneous in nature. Correspondingly, their The Audiological profile is highly variable. Some trends are given below.

1. *Pure tone Audiogram*

Most of the patients present with bilateral hearing loss. However, Reports of unilateral ANSD are common (Konradsson, 1996; Podwall et al., 2002; Buchman et al., 2006) and accounted for upto 7.3% according to Berlin et al. (2010). They also reported that 77% of their 103 subjects had a symmetrical hearing loss while the rest 23% had an asymmetric presentation. The degree of hearing loss may range from hearing sensitivity within normal limits to profound hearing loss. However, it is difficult to determine the degree of hearing loss in persons with ANSD as they not only show inconsistent responses, but many also show reverse sloping or peaked audiograms. Starr et al. (2000), in a study of 67 patients with ANSD, reported flat audiogram in 41% , reverse sloping audiogram in 29% , an irregular saw-tooth pattern in 9%, a 'U' shaped audiogram in 5%, and a tent shaped audiogram with a peak usually at 2 kHz in 5% of the patients. Only 11 % had high frequency sloping which is typical of cochlear hearing loss.

2. *Speech Perception*

Speech perception is drastically affected in individuals with ANSD, particularly in noise. It has been noted that patients with ANSD have speech perception abilities that are out of proportion with their pure tone hearing loss (Li, Wang, Chen, & Liang, 2005; Starr, et al., 1996). Also, speech perception abilities of persons with ANSD is highly variable, with some patients performing at levels expected for cochlear hearing loss of the same degree, while some others show little or no measurable speech identification despite having adequate sound detection abilities. Furthermore, this discrepancy between sound detection and speech

identification appears to be related to suprathreshold distortion of temporal cues rather than audibility (Ranee, Mckay, & Grayden, 2004; Zeng, Oba, Garde, Sininger, & Starr, 1999; Zeng, Kong, Michalewski, & Starr, 2005).

Speech perception in noise is severely affected in individuals with ANSD (Shallop, 2002; Zeng & Liu, 2006). Even those with good speech recognition abilities in quiet have extreme difficulties in the presence of noise. Noise activates auditory neurons and reduces their responsiveness to other signals, a phenomenon known commonly as ‘‘the line-busy effect” (Derbyshire and Davis, 1935; Powers et al., 1995). In addition, noise can specifically interfere with neural synchrony independent of a change of responsiveness (Miller et al., 1987).

3. *Acoustic reflexes*

Abnormality in middle ear reflexes has been consistently noted in the literature. Most of the times, both ipsilateral and contralateral reflexes are absent when the affected ear is stimulated.

4. *Otoacoustic Emissions*

Normal functioning of the outer hair cells is correlated with robust oto-acoustic emissions in those with ANSD. However, in some individuals with ANSD, OAEs may disappear over time (Starr, Sininger & Pratt 2000).

5. *Cochlear Microphonics*

Cochlear Microphonics are typically robust and present for several milliseconds after the transient click (Berlin, 1999; Delantre et al., 1998; Duan & Wang, 2002; Starr, et al., 1996; Starr, et al., 2000; Santarelli & Arslan, 2002). Rance, Beer and Cone-Wesson reported the presence of cochlear microphonics in all their 33 ears with ANSD even though 17 ears failed to demonstrate TEOAEs. Similarly,

Delantre, Mansbach and Bozet (1999) reported preserved cochlear microphonics in two children with ANSD in whom OAEs disappeared over time.

6. *Auditory Brainstem Response*

Auditory brainstem responses are generally absent in persons with ANSD. However, if present, then they are severely abnormal. Starr et al. (2000) reported that 70 % of their patients did not show any component of ABR regardless of the level of the stimulus. 19 % of the remaining showed abnormal wave V, and in most of them, the peak was clearly defined though characterized by abnormal amplitude and latency. 6% did show wave III and V, but their amplitude, wave morphology and latency was abnormal.

2.4 Psychoacoustical findings

a) *Intensity Processing*

Rance et al. (2004) demonstrated that persons with ANSD show a slightly larger difference limen at low sensation levels than normals, but it approached normal values at high sensation levels similar to that seen in normals. Similar results were reported by Zeng, 99, 2001, 2005.

b) *Frequency Processing*

Rance et al. (2004) further demonstrated that frequency resolution in individuals with ANSD, measured using notched-noise technique was normal. Frequency discrimination ability of patients with ANSD is significantly poorer compared to that of normal hearing subjects, particularly at low frequencies (Rance et al., 2004; Starr et al., 1991; Starr et al., 1996; Zeng et al., 2005). These results can be explained by the affected phase locking ability which is important for low frequency perception. In general, these studies demonstrate that individuals with ANSD have

relatively intact intensity and frequency resolution, but demonstrate impaired temporal aspects of frequency discrimination.

c) *Temporal Processing*

Several investigators have explored the temporal processing abilities of individuals with ANSD. Zeng et al., (2005) evaluated several time-related functions like temporal integration, gap detection, temporal modulation detection, backward masking, forward masking and simultaneous masking in individuals with ANSD.

i) Temporal integration: They found improvement in thresholds with increase in signal duration in individuals with ANSD as is the case with normals. However, the slope of the integration function was slightly elevated in individuals with ANSD. Similar results were reported by Starr et al. (1991).

ii) Gap detection: Abnormal gap detection (identification of silent period embedded within a noise burst) has been reported in individuals with ANSD (Zeng et al., 2005; Zeng et al., 1999, 2001). Normal hearing individuals required a silent interval of around 50 ms to detect a gap at 5 dB SL. However, the detection threshold improved to 3 ms at higher sensation levels (30 to 40 dB SL). Individuals with ANSD performed similar to normal hearing subjects at low sensation levels, but unlike normals, required significantly larger gap to detect at higher sensation levels. This is probably due to the fat at high intensities; the excess masking caused may cause a smearing of sharp temporal changes in the internal neural representation.

iii) Temporal Modulation detection thresholds: Zeng et al. (1999) reported that individuals with ANSD showed high peak sensitivity of -8.7 dB compared to -19.9 dB in normal controls and lower cut off frequency of 17 Hz compared to 258.1 Hz in normal controls. Rance et al. (2004) also reported significant differences in

modulation detection thresholds between ANSD subjects who had good and poor speech perception scores.

iv) Temporal Masking: Kraus et al. (2000) reported exaggerated masking effect in one patient with ANSD who had near normal hearing thresholds. Temporal masking and simultaneous masking paradigms have shown that individuals with ANSD have difficult y in separating sounds that occur successively as well as in detecting signal in noise (Zeng et al., 2005). In forward masking, individuals with ANSD showed 60 % masking even when signal and masker were separated by as much as 100 ms while normal controls showed only 15 % masking at a signal delay of <20ms. In simultaneous masking condition, individuals with ANSD showed excessive masking of about 20 dB compared to the normal control group.

2.5 Cortical potentials in ANSD

The condition of ANSD results in absent or grossly distorted ABRs but the cortical potentials can still be recorded in many of these individuals. This is because the ABRs are typically action potentials and have fast time constants, typically around 1ms. So, synchronous firing of neurons is very essential for the action potentials to generate a far field potential which can be recorded at the vertex. The cortical potentials on the other hand, are *dendritic* potentials and have large time constants in tens of milliseconds. They are hence less dependent on synchrony (Starr et al.,1996). Also, the cortical neurons are greater in number and much nearer to the scalp. Hence, cortical potentials can still be recorded in most subjects with ANSD.

Kraus et al., (2000) presented a case of a 24year old woman with normal hearing thresholds, robust OAEs and 100% speech recognition scores in quiet. The performance, however drastically reduced in the presence of noise. Wave I was absent and waves III and V were present inconsistently and had a poor morphology. Cortical potentials like LLR and MMN showed good wave morphology, latency and amplitude. It was hence concluded that optimal auditory nerve and brainstem synchrony was not necessary in quiet, but was important in the presence of noise.

Rance et al., (2002) investigated the relationship between speech perception and cortical potentials in 18 children with ANSD. Results indicated that 50% of the children had open set speech recognition scores similar to those with SNHL and the other 50% had no measurable open set speech recognition. They found that children in the former group had recordable cortical potentials and derived significant benefit from amplification while the latter had absent responses and failed to get appreciable benefit from amplification. Manjula and Vanaja (2004) reported similar results in their adult subjects with ANSD.

Narne and Vanaja (2008) used click stimuli to evoke cortical potentials in ANSD. They divided their subjects into Good and Poor performers based on their speech recognition abilities. They noted that N1-P2 amplitude was significantly greater in the good performers when compared to the poor performers. They did not find a significant correlation between N100 latency and speech perception scores. However, they did find a correlation between N100 amplitude and speech perception scores. On the contrary, some studies indicate no correlation of amplitude with speech perception scores but report correlation with latency. For instance, Michalewski, Starr, Zeng, and Dimitrijevic (2009) assessed the effect of signal intensity and continuous noise on the N100 cortical potentials in ANSD subjects. Subjects were

tested with brief 100ms tones of kHz at various signal levels and in continuous noise. They reported that the N100 latency in quiet was delayed and the amplitude was reduced compared to the normal group. The extent of latency delay was related to psychoacoustic measures of gap detection and speech recognition scores, but not to audibility. Noise in normal hearing subjects was accompanied by N100 latency delays and amplitude reductions paralleling those found in ANSD tested in quiet. They hence speculate that ANSD subjects tested in quiet behave as if they were in a 'noisy' environment. They propose that the disrupted auditory nerve activity actually provides a 'background neural noise' which competes with the nerves that still respond somewhat synchronously.

Dimitrijevic, Starr, Bhatt, Michalewski, Zeng and Pratt (2010) did a study on the representation of frequency and intensity changes of continuous tones at the cortical level in 10 subjects with ANSD. They reported that ANSD subjects demonstrated N100 only for larger changes in frequency and intensity in comparison to normals. Further, N100 latency in these subjects was significantly delayed compared to normals, more so for 250 Hz than for 4000 Hz and more so for changes in intensity rather than frequency. The amplitudes were also significantly reduced in all, except for pre-synaptic dysfunction subjects in whom amplitudes were greater than controls. The authors attribute this to abnormal adaptation in the presynaptic dysfunction group.

From all these studies, it is clear that the cortical potentials in the ANSD population are different from both the normal hearing as well as the SNHL population. It is also evident that the cortical potentials could lead to a better understanding of the neurophysiological processes at the cortical level in the ANSD population. This study aims to probe deeper into the representation of complex speech

stimuli at the cortical level and the interaction among the phonemes within the speech syllable in normals as well as the ANSD population.

2.6 Cortical Potentials in Normals and Sensorineural Hearing Loss

2.6.1 *Cortical potentials in normal hearing subjects*

Auditory Long latency Responses (ALLR) can be elicited by stimuli like clicks, tone bursts ansd speech stimuli. This provides an objective measure of the neurophysiological processes that underlie individual's ability to perceive speech (Purdy, Katsch, Sharma, Dillon & Ching, 2001; Tremblay, Freisen, Martin & Wright, 2003). The ALLRs obtained from speech stimuli typically have a longer latency and more robust amplitudes than the responses obtained from the click stimulus (Chandra & Barman, 2009 ; Tremblay et al., 2003).

Ostroff, Martin & Boothroyd (1998) obtained the cortical potentials for /sei/ and its components /s/ and /ei/ in a group of 8 normal hearing adult subjects. They reported a negative to positive fluctuation following the initial P2, called the Acoustic Change Complex (ACC). The analysis of waveforms for /s/ and /ei/ led them to conclude that the waveform obtained for /sei/ could very well be obtained by the combination of waveforms its constituent phonemes. This concept has also received support from Burger, Hoppe, Lohscheller, Eysholdt and Dollinger (2009) who simulated a CV syllable with a toneburst combination designed to match the temporal changes in the speech stimulus. Martin and Boothroyd (1999) studied the N1-P2 change complex for a change in periodicity of the stimuli with the spectral envelope and RMS energy being the same. For this, they used a tonal complex which was

followed by a band of noise. They reported a clear change complex on transition from noise to tone and viceversa. Change complex was reported to be absent for the noise-only and tone-only condition.

Tremblay et al. (2003) obtained P1-N1-P2 responses from 7 normal hearing young adults in response to naturally produced speech tokens- /bi/, /pi/, /shi/ & /si/. The subjects were tested and retested within an eight day period. The results revealed that the P1-N1-P2 responses were reliably recorded using naturally produced speech sounds. The speech sounds, which represented different acoustic cues, evoked distinct neural patterns. They concluded that the responses reliable and valid enough that they can be used to study the neural processing of speech. They also suggested that the responses can be used to study the changes over time during various types of rehabilitation.

Agung, Purdy, McMohan and Newall (2006) recorded ALLRs for speech stimuli presented at a conversational level (65 dB SPL) via a loudspeaker. The stimuli used were /a,u,I,s,sh,m & ɔ/ which covered a wide range of the speech spectrum. They aimed to see whether the cortical response was different for each speech sound in terms of latency and amplitude. They report that Cortical Auditory Evoked Potentials (CAEPs) produced by speech sounds dominated by high-frequency energy were significantly different in amplitude from CAEPs produced by sounds dominated by lower-frequency energy. Significant effects of stimulus duration were also observed, with shorter duration stimuli producing larger amplitudes and earlier latencies than longer duration stimuli. They concluded that CAEPs can be reliably evoked by sounds that encompass the entire speech frequency range and that CAEP latencies and amplitudes may provide an objective indication that spectrally different speech sounds are encoded differently at the cortical level.

2.6.2 *Cortical potentials in SNHL*

Polen (1984) found that moderate to severe sensorineural hearing loss resulted in the prolongation of N1, P2 and P3 latencies and a reduction in N2 amplitude. In contrast, Wall et al reported no significant differences between normal and mild to moderate SNHL subjects for latency, but corroborated the reduction in amplitude of N1. Kraus, McGee, Carrell & Sharma (1995) reported results of two subjects with SNHL. One subject with poor discrimination who had no MMN while another with good discrimination was shown to have a present MMN.

Martin et al. (1997, 98) reported the effect of high pass masking on the LLR and MMN in normals and mild to moderate SNHL. They reported the following results: Decreased audibility due to noise masking resulted in decreased in amplitude and increase in latency of all cortical ERPs as well as poorer performance on behavioural indices of speech perception. N1 showed systematic changes in amplitude and latency as the cut off frequency is lowered and was present as long as the stimulus was audible. In contrast, MMN, N2 and P3 showed a marked change only when the cut off was low enough to affect discrimination of /ba/ vs /da/. They also reported that latency may be a more sensitive indicator than amplitude since even mild reductions in audibility resulted in significant changes in latency of N1,N2 and P3 while amplitude remained the same .

Oates, Kutzberg and Stappells (2002) recorded N1, MMN, N2 and P3 along with behavioural measures to speech sounds /ba/ and /da/ presented at 65 and 80dBppe SPL in normal hearing as well as in those with SNHL varying in degree (1k to 2 kHz) from mild to profound hearing loss. They again reported that that as long as the stimulus presented was 12 dB above the average threshold of 1000 and 2000 Hz,

the amplitude parameters remained stable. However, latency was prolonged even with mild threshold elevations. The amplitude changes which occurred with reduction in intensity was more for the later ERP peaks like N2 and P3 and behavioural discrimination measures than the initial responses like N1 and the MMN. They also reported an intriguing finding that the grand average waveform of those with moderate hearing loss had greater amplitudes for N1 and P3 than the normal hearing group at high presentation levels. It is not known if the finding holds good at the individual level.

As can be seen from the literature, studies have been done to assess representation of finer aspects of complex speech stimuli in both the normal as well as the sensorineural hearing loss population. Studies in ANSD subjects are hence needed for the better understanding of representation of speech at the cortical level in these individuals.

Chapter 3

Method

The method included the recording of cortical potentials in subjects with Auditory Neuropathy Spectrum Disorder and in normal hearing individuals to two natural consonant-vowel combinations and subjecting them into analysis.

3.1 Participants

The study consisted of two groups of participants: Group 1 consisted of the normal hearing subjects while Group 2 consisted of subjects with Auditory Neuropathy Spectrum Disorder (ANSD).

3.1.1 *Normal Hearing subjects (Group 1)*

This group included 11 participants consisting of 6 males and 5 females with a mean age of 22 yrs (18-26 yrs). The participants did not have any history or complaints of otological and neurological abnormalities. They all had thresholds within 15dB HL (ISO 389-2, 1994) over the frequency range of 250 Hz to 8000 Hz for air conduction stimuli and 250 Hz to 4000 Hz for bone conduction stimuli. Speech identification scores were above 90% in both ears and they demonstrated 'A'type tympanogram bilaterally with reflexes present at 500Hz, 1000Hz and 2000Hz within normal limits. They also demonstrated normal Auditory Brainstem Responses (ABR).

3.1.2 *ANSD subjects (Group 2)*

This group consisted of 10 participants who had been clinically diagnosed as having ANSD and were recruited from those who were evaluated at

All India Institute of Speech and Hearing, Mysore. Their ages ranged from 19-54 yrs, with a mean age of 36.5 yrs. Six of them were male and four were female. Table 3.1 gives the demographic and Audiological details of the ANSD subject group. The Speech identification scores were symmetrical across the two ears. The subjects did not have any otological abnormalities and the presence of tumor was ruled out by neurological evaluation. The subjects demonstrated otoacoustic emissions and/or cochlear microphonics and had absent acoustic reflexes. Auditory brainstem responses were absent in all the subjects.

Participant	**Age/gender**	**Hearing sensitivity**	**Speech Identification Scores**	**Audiometric pattern**
AN1	27y/M	Normal	70	Flat
AN2	28y/M	Moderate	36	Rising
AN3	19y/F	Normal	24	Flat
AN4	21y/M	Normal	36	Flat
AN5	35y/F	Mild	20	Flat
AN6	22y/F	Mild	68	Rising
AN7	28y/M	Mild	20	Rising
AN8	20/F	Normal	24	Flat
AN9	54/M	Moderate	80	Rising
AN10	21/M	Normal	44	Flat

Table 3.1 *Demographic and Audiological details of individuals with ANSD.*

The hearing loss in terms of pure tone thresholds ranged from nearly normal to moderate hearing loss. Open-set speech recognition scores in quiet ranged from 0% to 80% correct. The audiometric pattern was either flat or of the rising type.

3.2 Instrumentation

- A two channel Madsen OB922 clinical audiometer calibrated according to ANSI standards 1996, with Telephonics TDH39 earphones housed in MX/41 AR ear cushions and Radio ear B71 Bone vibrator for puretone audiometry
- A calibrated Grason Stadler Tympstar Immittance V26 instrument to assess middle ear function
- A ILO version 6 instrument for measuring otoacoustic emissions
- Intelligent Hearing Systems (IHS) Opti-amp evoked potential instrument with Smart EP version 3.94USBeZ software to elicit and measure evoked potentials

3.3 Procedure

The study was carried out in two phases: I. Stimulus preparation and II. Recording of evoked potentials

I. Stimulus Preparation

The stimuli consisted of CV syllables /su/ and /chu/. The stimuli were produced by an adult native speaker of Kannada and were recorded on to a PC at 16 bits and 44100Hz sampling frequency using Adobe Audition 1.5 software. The syllables /su/ and /chu/ had the durations of 350 ms and 180 ms respectively.

The two stimuli were further divided into their constituent consonant and vocalic parts. Figure 3.1 shows the waveforms of all the six stimuli.

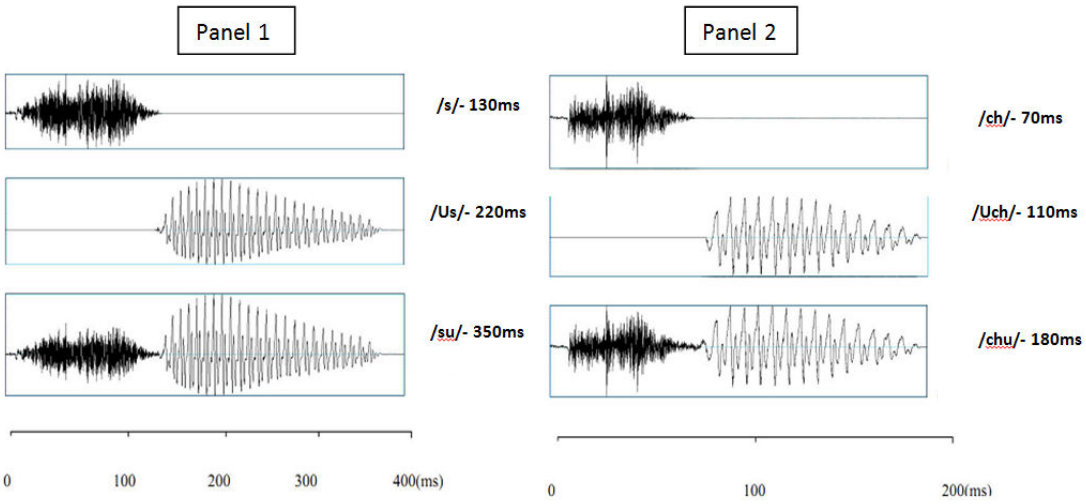

Figure 3: The figure displays the stimuli /su/ and /chu/ and their partials (constituent phonemes) Panel1: Waveforms of /su/ and its components- /s/ had a stimulus duration of 130ms while /u/$_{su}$ had a duration of 220ms; Panel2: Waveforms of /chu/ and its components- /ch/ had a stimulus duration of 70ms while /u/$_{chu}$ had a duration of 110ms

The beginning of the vowel was determined by the presence of formants and the course of the envelope of the stimulus similar to the procedure adopted by Diegser, Torsten & Ulrich, (2009). The vowel part was then zeroed to get the consonant part and the consonant part was zeroed to get the vowel part of the CV syllable. The vocalic parts from /su/ and /chu/ were denoted as /u/$_{su}$ and /u/$_{chu}$ respectively. Thus, we obtained 6 stimuli- /su/,/s/,/u/$_{su}$ and /chu/,/ch, /u/$_{chu}$. Note that the virtual duration of the vocalic and the consonant parts are the same as their CV counterparts (Portions of CV were only zeroed, not cut thus maintaining the duration).

The wave files of the stimuli were then converted into IHS-Stim files using the file conversion programme in the IHS software. The stimuli output from the transducers were then calibrated to 80dBSPL using a Bruel and Kjaer artificial ear and a Sound level meter.

II. Recording of evoked potentials

The patients were seated in an electrically and acoustically shielded room. A skin abrasive paste was used to clean the electrode sites and disc electrodes dipped in conduction paste were placed on the scalp and attached using a surgical tape. The subjects were relaxed and watched DVD movies played back without sound to avoid attention to the stimulus. They were asked to avoid excessive blinking.

For stimulus presentation and data recording, 4 channel Intelligent Hearing Systems (IHS) Smart EP version 3.95USBeZ was used. The stimulus were presented binaurally through ER-3A insert ear phones and calibrated to a presentation level of 80 dBSPL. The repetition rate was 0.8/sec. The ERPs were recorded from Cz, C3 and C4, referenced to the tip of the nose. Lower forehead served as the site for the ground electrode. Vertical eye movements were monitored with the electrodes placed over superior and inferior canthus of the right eye. The electrode impedances were maintained below 5kOhms with the relative impedance not greater than 2kOhms. The EEG signals were amplified 25000 times and filtered from 1 to 100Hz at 6dB/octave. The Ocular channel was amplified by only 5000 times and artifact rejection was set at 100μV similar to the procedure adopted by the previous investigators (Ostroff et al.,1998;Tremblay et al., 2002). The recording window consisted of 800ms post stimulus duration and a prestimulus baseline of 100ms. Offline, the waveforms were smoothened by digitally filtering from 1-30Hz at 12dB/octave.

The order of stimulus presentation was randomized to avoid any order effect. Each of the six stimuli were presented twice (2 runs), each run consisting of at least 200 sweeps. The two runs were then averaged and subjected to analysis. Total

duration of testing was approximately 1hr and 30 minutes. Breaks were provided to the subjects when necessary.

The grand averages of the waveforms were obtained for the normal population to aid in peak identification and measurement in the data from individual subjects. The waveform analysis was done only for those from Cz where the response amplitudes were the largest. The C3 and C4 channels were used for response verification. The averaged waveforms for the same stimulus were used to check for replication of waveforms and to aid in peak marking.

The latency and amplitude of P1, N1, P2 and the positivities and negativities of the ACC complex were measured. The amplitude measurements were based on the measurements from the 'corrected baseline' obtained by the mean of the amplitude values in the prestimulus latency region. The data was tabulated in terms of latency and amplitude for both the subject groups for both the stimuli.

Chapter 4

Results and Discussion

The aim of this investigation was to study the representation of complex speech stimulus at the cortical level. Cortical potentials were elicited by two stimuli /su/ and /chu/. The latencies and amplitudes of the peaks were calculated for both the subject groups for all the six stimuli and tabulated. To investigate the objectives of the study, the following statistical analysis were done.

- To find out if there was a significant difference in latency and amplitude between the controls and the ANSD subjects in terms of latencies and amplitudes of the peaks, Mann-Whitney U test was done. This was done separately for the two stimuli.
- To find out if there was a significant correlation between latency and amplitude parameters of the cortical potentials and the Speech Identification (SI) scores in the ANSD group, Pearson product moment correlation test was done separately for both the stimuli.

4.1 Cortical potentials in normal hearing subjects

The normal hearing subjects had robust cortical potentials for all the stimuli. The nomenclature used to name the peaks has varied throughout the literature (Ostroff, Martin & Boothroyd, 1998; Tremblay, Billings, Friesen, 2006). In this study, we utilized the simple nomenclature of naming the first positivity as P1, first negativity as N1 etc. The Acoustic change complex corresponds to region of N2' and P3' for the 'whole' stimuli like /su/ and /chu/ (Ostroff, Martin & Boothroyd, 1998) to differentiate it from the usual negativity following P2 seen for partials has been

simply named N2. The results will be discussed based on the stimuli used for the eliciting the cortical potentials: /su/ and /chu/.

4.1.1 */su/ stimulus*

The /su/ stimulus elicited a change complex in all the 11 normal hearing subjects. The grand average for the /su/ and its components are shown in the Figure 4.1 and Table 4.1 displays the mean and standard deviations of latency and amplitude values for /su/. Similar to the present study, previous investigators reported that ACC was present in almost all of their normal hearing subjects ( ). Ostroff, Martin & Boothroyd (1998) employed /sei/ as their stimulus and reported longer latencies and lesser amplitude values than those noticed in the present study. In their study, the onset of the diphthong /ei/ followed a 150ms fricative /s/ and they reported that the change complex was obtained at a latency of around 250ms while the change is reported at around 230ms in this study. Although the absolute latencies in the present study differ from those obtained in other studies, the relative latency i.e the latency with respect to the onset of the change in the stimulus remains similar.

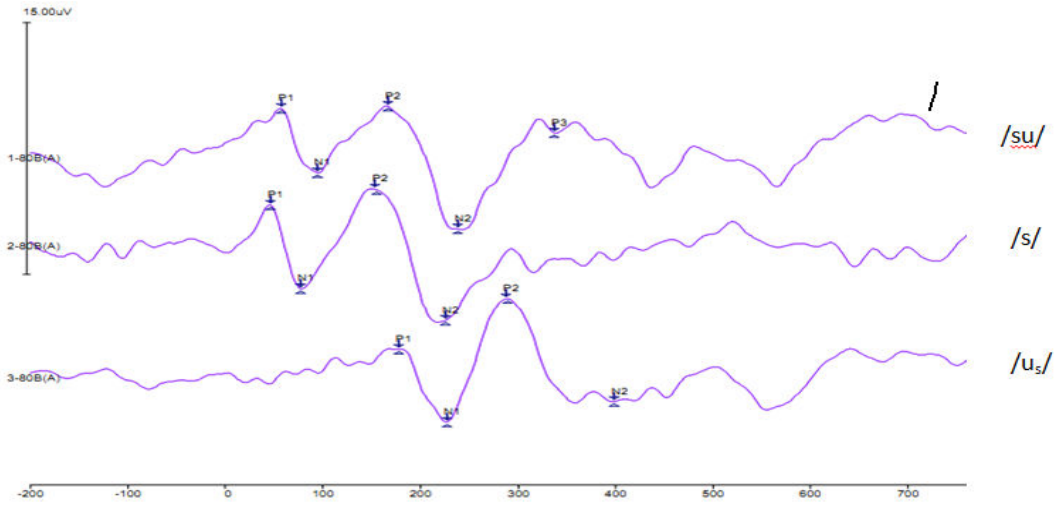

Figure 4.1: *Grand averaged waveforms for /su/ and its components /s/ and /u$_s$/ in normal hearing subjects*

Table 4.1

Mean and standard deviations of latency and amplitude parameters fornormal hearing subjects.

Wave component for /su/	**Mean latency (ms)**	**SD for latency (ms)**	**Mean amplitude (µV)**	**SD for amplitude (µV)**
P1	49.1(6.5)	6.5	2.4	1.2
N1	89.9	5.1	-1.0	1.4
P2	155.3	13.2	2.9	1.0
N2'	236.4	6.1	-4.0	1.2
P3'	317.8	16.5	1.5	1.4

The N2' and P3' components for the /su/ stimulus (first waveform) evidence the presence of the acoustic change complex. The waveforms of the partials /s/ and /u_s/ (second and third waveforms respectively) help us understand as to how the complex waveform for the whole stimulus /su/ was formed. Notice that the silence of 130 ms corresponding to the consonant duration in the /u_s/ stimulus is also very well represented as a continuum of the prestimulus baseline. The combination of the waveforms for /s/ and /u/ result in the waveform similar to that obtained from /su/.

4.1.2 */chu/ stimulus*

The change complex for /chu/ was present in all the 11 normal hearing subjects. Figure 4.2 displays the grand average of /chu/ (first waveform), /ch/ (second) and /u_{ch}/ (third) respectively and Table 4.2 displays the mean and standard deviations

of latency and amplitude parameters for /chu/. Most of the previous studies in literature have employed stimuli where the onset of change occurs after a relatively long duration (>100ms) after the onset of the first component of the stimulus. For instance, Martin & Boothroyd (1999) employed a duration of 400ms for a change from noise to tone. In this study, in /chu/ the change starts from 70ms and hence, the change complex results in latencies earlier than those reported by previous investigators.

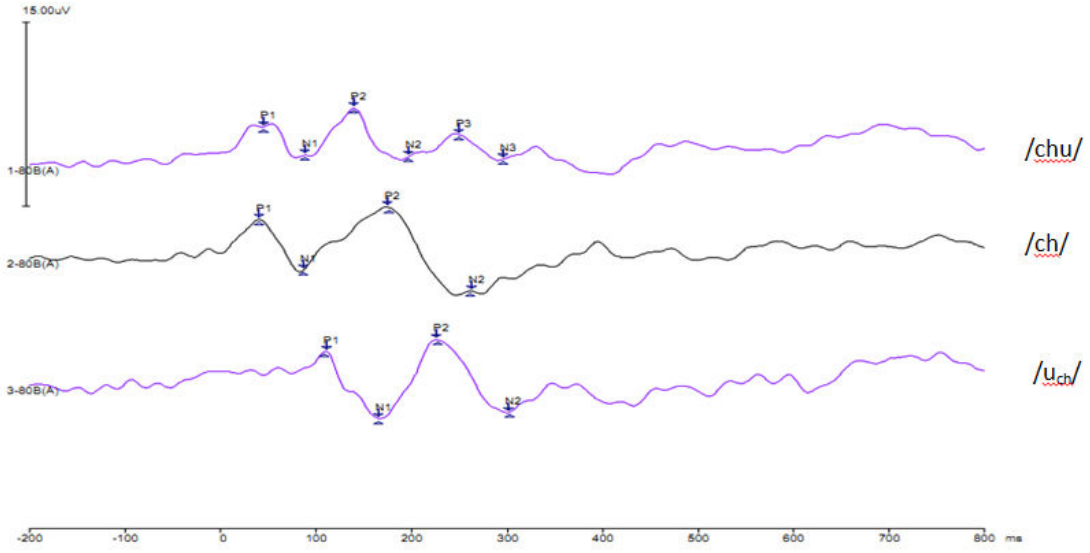

Figure 4.2. Grand averaged waveforms for /chu/ and its components /ch/ and /u_{ch}/ in normal hearing subjects

The waveform for /chu/ is much more complex in terms of interaction of the waveforms of the stimulus partials /ch/ and /u_{ch}/. Again, the addition of the waveforms for the partials reveals the way the complex waveform for the 'whole' stimulus is formed. The N1 for /ch/ partially combines with the P1 for /u_{ch}/ and positive P2 of /ch/ merges with negative N1 of /u_{ch}/ causing a narrower P2 in /chu/. Finally, the N2 of /ch/ and P2 of /u_{ch}/ combine to produce a much smaller P3' for /chu/.

Table 4.2.

Mean and standard deviations of latency and amplitude parameters for /chu/ in normal hearing subjects.

Wave component for /chu/	**Mean latency (ms)**	**SD for latency (ms)**	**Mean amplitude (µV)**	**SD for amplitude (µV)**
P1	49.3	8.8	2.3	1.0
N1	86.6	8.4	-1.9	1.8
P2	137.1	5.7	2.8	1.3
N2'	195.4	9.6	-2.5	1.7
P3'	246.4	14.2	0.9	1.5

4.1.3 *Comparison between /su/ and /chu/ in normal hearing subjects*

The latencies for /chu/ were much earlier than /su/ due to the earlier vowel onset in /chu/ (70ms) than /su/ (130ms). The responses from both the stimuli had comparable amplitude values. Looking at the morphology of the waveform in /su/, sometimes the second P1 corresponding to the vowel onset was visible which was never the case with /chu/. The waveform for /chu/ is much more complex because the P1 of the second component of the stimulus /u_{ch}/ interacts with the N1 of the first stimulus /ch/ rather than with the P2 of the first stimulus as it happens in /su/. These differences in the cortical responses suggest that the stimuli yield different information. /su/ gives us an idea of the ability of the auditory system to resolve change over a longer duration while /chu/ yields information regarding the ability of

the system to resolve over a shorter duration. The difference in latency and amplitude parameters between /su and /chu/ were not statistically compared since the latency differences were quite apparent.

4.2 Cortical Potentials in ANSD subjects

The cortical potentials were highly variable across subjects indicating heterogeneity of the ANSD subject group. Similar to the previous section on normal hearing subjects, the results are discussed based on the stimulus used to elicit the cortical potentials.

4.2.1 */su/ stimulus*

Table 4.3 gives the mean and standard deviations of latency and amplitude parameters for /su/ in the ANSD subjects. Only 4 out of 10 (40%) of the subjects with ANSD had an acoustic change complex present. Further, 5 of the subjects lacked a replicable P1.

Table 4.3

Mean and standard deviation of latency and amplitude for /su/ in ANSD subjects

Wave component for /chu/	**Mean latency (ms)**	**SD for latency (ms)**	**Mean amplitude (µV)**	**SD for amplitude (µV)**
P1	70.1	22.6	2.3	0.7
N1	110	23.0	-0.6	-1.4
P2	195.1	19.7	2.7	2.8
N2'	290.7	31.7	-3.5	-2
P3'	353.6	19.2	1.8	1.2

The absence of P1 may not be clinically significant since the P1 is often reported to be of a small amplitude or even absent in adult subjects (Davis & Zerlin, 1996; Jerger & Jerger, 1970). The latencies and amplitudes of other peaks are in consonance with those reported other studies in the ANSD population (Dimitrijevic et al, 2010; Michalewski, Starr, Zeng & Dimitrijevic, 2009). The figure 4.3 shows the cortical potentials for the subjects with the ACC.

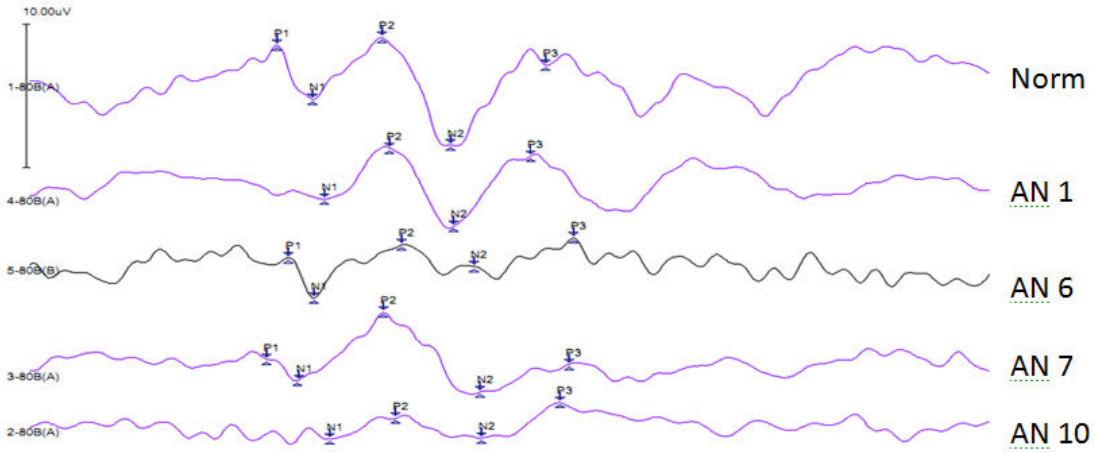

Figure 4.3. The cortical potentials for /su/ in ANSD subjects with ACC. Normal grand average waveform is also given for comparison.

From figure 4.3, a few things can be readily observed. Two of the subjects (AN 1 and AN 10) did not have a replicable P1 component. The most prominent component is the central negativity of N2'. Interestingly, those who demonstrated the change complex had better speech identification scores (AN1:76%, AN6:68%, AN7: 80% and AN10: 44%) than those who did not have an ACC (Refer Table 3.1). Thus, the speech identification scores correlated very well with the presence of the change complex. This is in consonance with the previous studies like Dimitrijevic et al (2010)

who report that the subjects who demonstrate the presence of cortical potentials to frequency and intensity change had better speech perception scores than those who did not.

Six subjects: AN2, AN3, AN4, AN5, AN8 and AN9 demonstrated the initial LLR, but not the following ACC component. These subjects typically had poor SI scores in quiet ranging from 36% to 12% (Refer Table 3.1). The latency and amplitude values for these subjects were widely distributed and extremely heterogeneous as also noted in the literature (Dimitrijevic et al, 2010; Narne & Vanaja, 2009; Starr et al, 1996). Though the precise reason for this is not known, following are the probable reasons. First, a demyelinatory condition may induce a conduction block causing an increase in latency (Starr et al, 1996). Secondly, Dimitrijevic et al (2010) reported that the latencies were prolonged in ANSD subjects with postsynaptic site of dysfunction than those with presynaptic type site of dysfunction. Thirdly, conditions like axonal neuropathy may cause a decrease in amplitude while preserving the latency within normal limits. Figure 4.4 displays the waveforms of one such subject AN3.

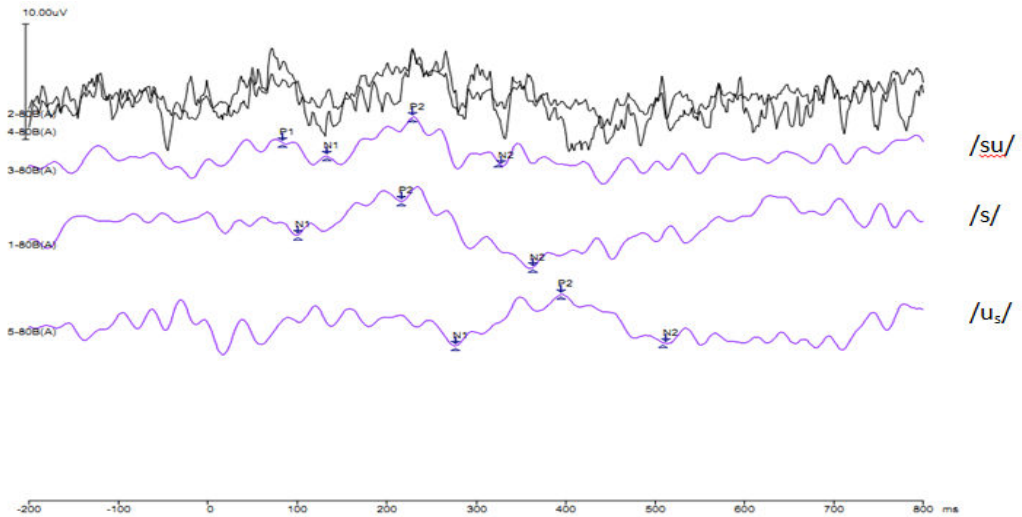

Figure 4.4. Cortical potentials for /su/, /s/ and /u$_s$/ in AN3

For the subjects who did not demonstrate ACC, long latency responses (LLR) were indeed present for the individual components /s/ and /u_s/. But, in the response to the whole stimulus /su/, the change complex was absent. This may be because the subjects could not resolve the two phonemes as being different due to their reduced temporal resolution (Zeng et al, 2005). This effect may also be enhanced by increased forward masking reported in these subjects (Zeng et al, 2005, Kraus et al, 2000). Also, note the broadening of peaks, particularly the P2 component which might be attributed to dys-synchronised neural discharges which can add to result in broader responses (Starr et al, 1996).

Correlational analysis was used to examine if the the latency and amplitude parameters had any correlation with the speech identification scores. The Pearson product moment correlation showed no correlation ($p>0.05$) with any of the latency and amplitude parameters. This is in opposition with previous studies who report a significant correlation of speech identification scores with N1 latency (Michalewski et al, 2009) and N1 amplitude (Narne & Vanaja, 2008). However, this study used the speech stimulus as against the tones and clicks used in the previous studies. The results are in consonance with the results from Chandra & Barman (2009) who used speech stimuli and reported no significant correlation between the parameters of cortical potentials with speech identification scores. This result may also be attributed to the heterogeneity of the sample under study as well as the numerous other variables like stimulus and recording parameters, subject state etc. However, the results do suggest that the presence of the ACC is a positive indicator for better speech perception abilities, ie the ability of the subjects to resolve the syllable into its components at the cortical level as reflected by the far-field cortical potentials is correlated with better speech perception abilities.

4.2.1.2 *Comparison with the control group*

Mann-Whitney U test for latency measures revealed a significantly prolonged latency for N1 and P2 ($p<0.05$). This is in agreement with the previous studies like Michalewski et al 2009 who reported that N1 was particularly sensitive and was delayed in ANSD subjects. The prolongation may be attributed to decreased neural synchrony which may lead broader peaks and cause reductions in amplitude. Further, demyelination leads to reduced conduction velocity and repetitive stimulation of demyelinated fibres may lead to excitation delay, further reduction in the velocity of the action potential and intermittent/total block in their propagation (Raminsky & Sears, 1972).

Mann-Whitney test for amplitude measures revealed a no significant difference in amplitude parameters for both /su/ /. This is in agreement with Chandra & Barman (2009) who reported absence of a significant difference across amplitude measures. It must be noted that the mean amplitude of N1 for /su/ / were greater in the control group than the ANSD subjects. However, they did not reach statistical significance due to large standard deviations.

4.2.2 */chu/ stimulus*

Table 4.3 gives the mean and standard deviations of latencies and amplitudes for /chu/. Only one (AN 7) out of 10 subjects (10%) demonstrated the presence of the ACC. Also, two of the subjects did not have a replicable P1. Figure 4.5 displays the ACC for control group and AN7 who demonstrated the ACC for /chu/. As can be seen from the figure 4.5, the latency and the amplitude values lie within normal limits. The peaks however are much broader, particularly the P3 component in the response to /chu/ and the P2 component in response to /ch/. Similar

to the results for /su/, this subject demonstrated the highest speech perception scores in the whole group (80%) demonstrating that the presence of the ACC is a positive predictor of speech identification scores. This correlates with the results of previous studies like Dimitrijevic et al (2010) who reported that speech perception scores were significantly correlated with the presence of cortical potentials to changes in frequency and intensity.

Table 4.4

Mean and standard deviations of latency and amplitude parameters for /chu/ in ANSD subjects.

Wave component for /chu/	**Mean latency (ms)**	**SD for latency (ms)**	**Mean amplitude (µV)**	**SD for amplitude (µV)**
P1	63.5	19.3	0.8	0.4
N1	99	20.4	-1.4	1.2
P2	192.5	33.2	2.0	1.0
N2	290.8	60.1	-1.4	1.0
P3	-	-	-	-

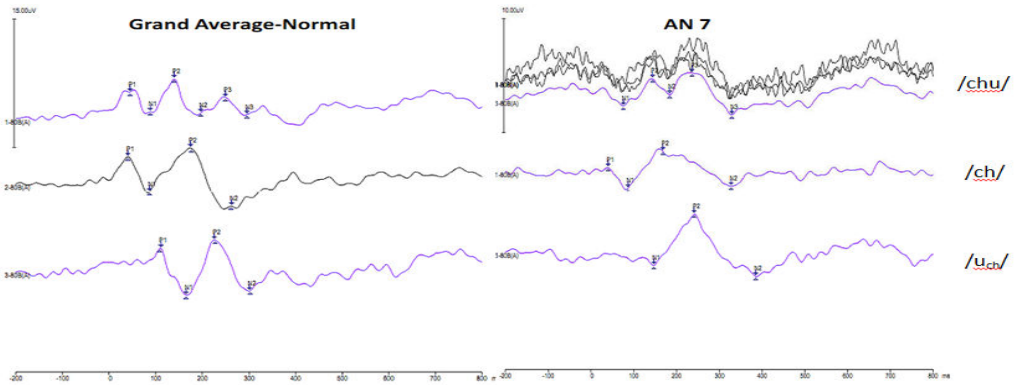

Figure 4.5. Cortical potentials for /chu/ in AN7. Grand average for control group is given to the left for comparison.

The rest of the nine subjects with ANSD did not reveal the presence of ACC. Figure 4.6 displays the waveforms for one such subject AN 9. Similar to the response observed with /su/, the change complex was not present for the whole stimulus /chu/ even though responses are present to its constituent phonemes /ch/ and /u_{ch}/. Again, this may be attributed to decreased temporal resolution and increased forward masking effect observed in subjects with ANSD (Zeng et al, 2005; Kraus et al, 2000).

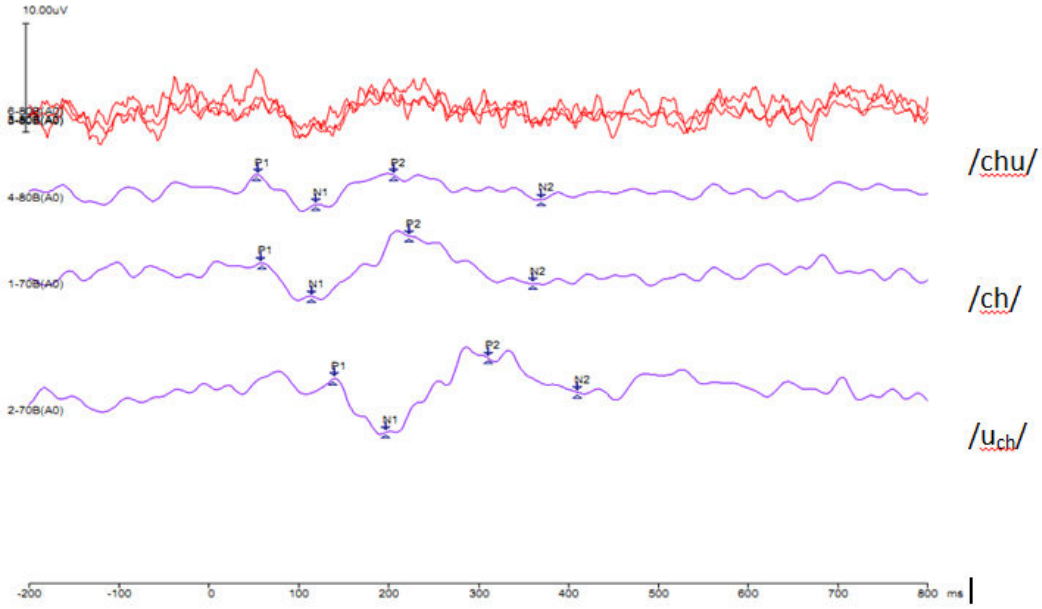

Figure 4.6. Cortical potentials for /chu/, /ch/ and /u_{ch}/ for the subject AN 9

Correlation analysis was done to examine if latency and amplitude parameters had any correlation with the speech identification scores. The Pearson product moment correlation showed no correlation ($p>0.05$) with any of the latency and amplitude parameters. This may be attributed to the heterogeneity of the sample under study as well as the numerous other variables like stimulus and recording parameters, subject state etc. There is a lack of consensus in literature regarding the correlation of LLR parameters with speech identification scores (Narne & Vanaja, 2008;

Michalewski et al, 2009; Chandra & Barman, 2009). The presence of ACC however, remained a positive indicator for good speech identification scores.

4.2.2.2 *Comparison with the control group*

Mann-Whitney U test for latency measures revealed a significantly prolonged latency for N1 and P2 ($p<0.05$) for /chu/. This is in agreement with the previous studies like Michalewski et al 2009 who also reported a significant correlation of N1 latency with psychoacoustical measures like gap detection. Similar prolongations in latency are seen in normals at reduced intensities (Michalewski et al, 2009) and in noise (Chandra & Barman, 2009) both of which are known to cause a reduction in the synchrony of neural discharges.

Mann-Whitney test revealed a no significant difference in amplitude parameters for /chu/ which is in agreement with previous studies (Chandra & Barman, 2009). Some subjects with ANSD did have decreased amplitudes while some had normal amplitudes. Decrease in amplitudes may be explained by reduction of synchrony which leads to less constructive addition of discharges and by the decrease in the neural population seen in axonal neuropathy (Strarr et al, 1996). The magnitude of reduction in amplitude in either of pathophysiology depends upon the severity of the condition. Further investigation correlating cortical potentials with neurological findings need to be carried out to confirm this.

4.3 Comparison of the Acoustic change complex of /su/ and /chu/ in ANSD group

The fact that only one of the subjects out of ten (10%) had an ACC for /chu/ while four of the subjects (40%) had an ACC for /su/ must be considered. /su/ had a slow fricative lasting for a duration of 130ms before the vowel started. Even though

the peaks in the ANSD group were delayed and broader than the control group, the LLRs for the partials could still stay sufficiently separated leading to the detection of a change component. This however was not the case with /chu/. Since the consonant had a duration of only 70ms, broadening of peaks led to the overlap of the two LLRs for the partials resulting in a single visible LLR. This is readily evident on studying the difference in the mean latencies of N1 for /s/ &/u_s/ vs the difference in the mean latencies of N1 for /ch/ & /u_{ch}/. It is 133ms for /su/ components and 72ms for /chu/ components. The larger separation for /su/ hence resulted in a clearer ACC. This effect was also found in normals by Diegser, Wohlberedt & Hoppe (2009) when the consonant duration very small (47ms). It is also possible that forward masking effect (Kraus et al, 2000) may be more in /chu/ than /su/ since the second component is introduced after a very short duration after the first component. Be it as it may, increased difficulties in shorter duration component processing is reflected in these results which correlates well with the previous results on speech perception (Zeng & Liu, 2006; Zeng, Oba, Sininger, Garde & Starr, 1999).

Chapter 5

Summary and Conclusions

Auditory Neuropathy Spectrum Disorder (ANSD) has since been studied extensively using behavioural measures and severe processing deficits in the temporal domain have been reported (Zeng et al, 1999, 2001, 2005; Kraus et al, 2000). Previous research has indicated that electrophysiological measures like cortical potentials can be used for objective assessment of temporal processing (Michalewski, Starr, Nguyen, Kong& Zeng, 2005; Michalewski, Starr, Zeng, & Dimitrijevic, 2009). This study was a preliminary investigation into the use of cortical potentials to understand the representation of complex speech stimuli at the cortical level in ANSD population.

The study investigated the nature of cortical potentials for two consonant-vowel combinations /su/ and /chu/ and their partials /s/, /u_s/ and /ch/, /u_{ch}/. 11 normal hearing (6 males and 5 females) and 10 ANSD subjects (6 males and 4 females) were considered for the study. The potentials recorded from the vertex were peak marked and the potentials recorded from C3 and C4 were used for the confirmation of the peaks at the vertex. The latency and amplitude values were tabulated for the stimuli across both the subject groups.

The data was first examined for the presence and absence of the acoustic change component. Mann-Whitney U test was used to examine if the latency and amplitude measures were significantly different across the two subject groups for both the stimuli /su/ and /chu/. Pearson product moment correlation test was done to

examine is speech perception was significantly correlated with any of the latency and amplitude parameters for the responses to /su/ and /chu/. The following were the findings-

- 4/10 subjects (40%) had an acoustic change component for /su/ and 1/10 (10%) had an acoustic change component for /chu/. This indicated that ANSD subjects have more problems perceiving changes across a shorter time span (Stops, Affricates) than stimuli which change across longer time spans (fricative, vowel)
- The presence of ACC was always associated with good speech identification scores. So, presence of ACC was found to be a positive predictor for good speech perception abilities.
- The latency and amplitude values in the ANSD group were characterised by significant heterogeneity consistent with variations in type and site of lesion, severity of the disorder, onset of the disorder etc and could range from being within normal limits to being indicative of abnormality. Accordingly, correlation was not found for these parameters with speech perception.

This preliminary study thus revealed that the some of the ANSD subjects do have the ability to demonstrate the Acoustic Change Complex to the consonant-vowel combinations and that its presence is associated with good speech perception abilities. Significant heterogeneity was observed in latency measures with responses ranging from normal to very prolonged. Similarly, the amplitude measures ranged from being within normal limits to a much diminished value. The results of this study support the feasibility of utilizing cortical potentials in ANSD subjects to examine the responses to more complex stimuli like the CVC, VCV combinations and to understand the interaction of one phoneme over the other when they are combined into a syllable in

terms of possible forward and reverse masking effects. Also, research into the hearing aid benefit in persons with and without the acoustic change complex, the change complex with and without amplification and the cortical potentials to complex stimuli with cochlear implants will yield rich information regarding the neurophysiological processes underlying speech perception in normal hearing as well as in individuals with various disorders of the auditory system.

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