Cortical Auditory Evoked Potentials to Complex Speech Stimuli in Auditory Neuropathy Spectrum Disorders

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Abstract

The present study examined cortical auditory evoked potentials (CAEPs) for complex CV syllables in individuals with auditory neuropathy spectrum disorders (ANSD). CAEPs were recorded for /su/ and /chu/ (350ms and 180ms respectively) and their constituents (/s/, /u$_s$/, /ch/, /u$_{ch}$/) in 10 subjects with ANSD and 11 subjects with normal hearing. All normal subjects demonstrated an acoustic change complex (ACC) for both /su/ and /chu/. On the other hand, four out of 10 subjects with ANSD demonstrated an ACC for /su/ and one out of 10 subjects demonstrated an ACC for /chu/. In addition, those who had ACC absent demonstrated the CAEPs for individual constituents of the syllable but the whole stimulus elicited only a single response rather than a change complex probably indicating reduced temporal resolution and forward masking effects. Further, latencies and amplitudes were highly heterogeneous in the ANSD population and ranged from being within normal limits to being highly deviant. The study supports the feasibility of using the cortical potentials to study the cortical representation of complex speech stimuli in ANSD subjects.

Key words: *Cortical, acoustic change complex, Auditory Neuropathy Spectrum Disorder*

Introduction

Auditory Neuropathy Spectrum Disorder (ANSD) is 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, Kong, Michalewski, & Starr, 2005; Sininger & Oba, 2001). A series of psycho-acoustical studies have revealed that temporal processing is severely affected in these individuals (Zeng, Kong, Michalewski, & Starr, 2005; Kraus, Bradlow, Cheatham, Cunningham, King & Koch, 2000). Though the behavioural performance is known, the physiological encoding of the stimulus in ANSD subjects is not clear. Objective tools like electrophysiological responses from the auditory system can be used to better understand the representation of speech stimulus in central auditory nervous system.

Though Auditory Brainstem Response (ABR) and Frequency Following Responses (FFR) have been reported to be absent or grossly abnormal in cases with ANSD, cortical potentials were reported to be present in majority of the cases indicating the preservation of some useful auditory capabilities (Starr, et al., 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., 1999; Vanaja & Manjula, 2004).

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 and has been demonstrated in the sensori-neural hearing loss population also (Martin, Tremblay & Korczak, 2008).

The current study aims to utilise the ACC as a tool to study the cortical representation of two consonant-vowel pairs: a fricative-vowel combination (su) and an affricate-vowel combination (chu). 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.

Method

Participants

Normal Hearing participants (Group 1): This group included 11 participants consisting of 6 males and 5 females with a mean age of 22 years (18-26 years).

The participants did not have any history or complaints of otological and neurological abnormalities. They all had thresholds within 15 dBHL 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 500 Hz, 1000 Hz and 2000 Hz within normal limits. They also demonstrated normal auditory brainstem responses (ABR).

ANSD participants (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 to 54 years, with a mean age of 36.5 years. Six of them were male and four were female. Table 1 gives the demographic and Audiological details of the ANSD subject group. The speech identification scores were symmetrical between 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 oto-acoustic emissions and/or cochlear microphonics and had absent acoustic reflexes. ABR was absent in all the subjects. 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.

Procedure

I. Stimulus preparation: The stimuli consisted of CV syllables /su/ and /chu/. The stimuli were produced by an adult native speaker of Kannada (a Dravidian language spoken in southern part of India) and were recorded on to a PC at 16 bits and 44100 Hz 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. 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 & Hoppe, (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. In total there were 6 stimuli- /su/,/s/,$/u/_{su}$ and /chu/,/ch, $/u/_{chu}$. Figure 1 shows the waveforms of all the six stimuli. The wave files of the stimuli were converted into Intelligent Hearing System (IHS)-Stim files using the file conversion programme in the IHS software. The stimuli output from the transducers were then calibrated to 80 dBSPL using a Larsen and Davis artificial ear and a Sound level meter.

II. Recording of evoked potentials: The patients were seated comfortably in a reclining chair 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 without sound to avoid attention to the stimulus. They were asked to avoid excessive blinking.

For stimulus presentation and data recording, 4 channel IHS Smart EP version 3.95 USBeZ was used. The stimuli were presented binaurally through ER-3A insert ear phones and calibrated to a presentation level of 80 dBSPL. Each of the

Table 1: Demographic and Audiological details of individuals with ANSD

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

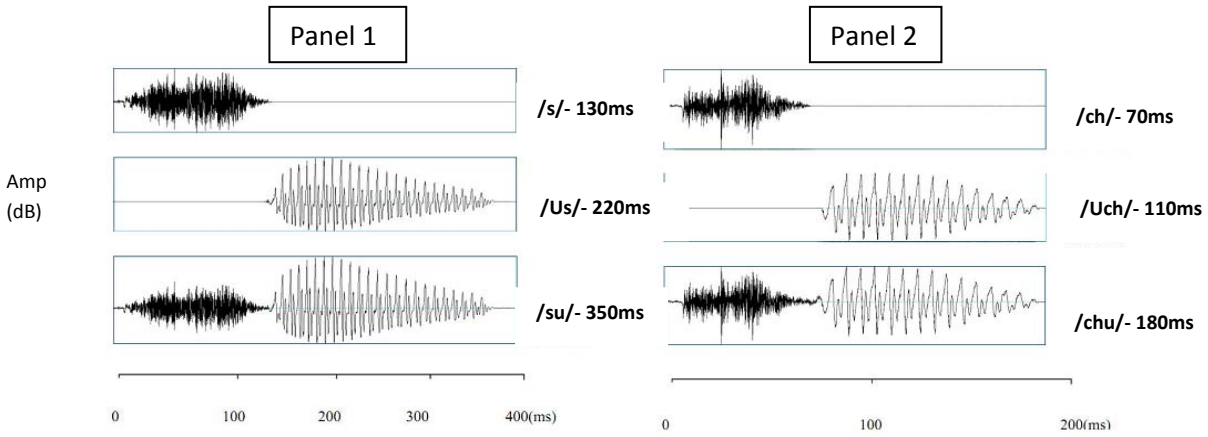

Figure 1: The figure displays the stimuli /su/ and /chu/ and their partials (constituent phonemes) Panel 1: Waveforms of /su/ and its components- /s/ had a stimulus duration of 130ms while /u/$_{su}$ had a duration of 220ms; Panel 2: Waveforms of /chu/ and its components- /ch/ had a stimulus duration of 70ms while /u/$_{chu}$ had a duration of 110ms.

six stimuli were presented twice (2 runs), each run consisting of at least 200 sweeps at a rate of 0.8/sec. The order of stimulus presentation was randomized to avoid any order effect.

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 positive electrode placed over superior canthus and inverting electrode on the inferior canthus of the right eye. The electrode impedances were maintained below 5 kOhms with the relative impedance not greater than 2 kOhms. The EEG signals were amplified 25000 times and filtered from 1 to 100 Hz at 6 dB/octave. The Ocular channel was amplified by only 5000 times and artifact rejection was set at 100 µV similar to the procedure adopted by the previous investigators (Ostroff, et al., 1998; Tremblay, Souza & Piskosz, 2002). The recording window consisted of 800 ms post-stimulus duration and a pre-stimulus baseline of 100 ms. Offline, the waveforms were smoothened by digitally filtering from 1-30 Hz at 12 dB/octave. Total duration of testing was approximately 1 hour 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. 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 N2' and P3' (Ostroff, et al., 1998). The waveform analysis was done only for potentials obtained 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 was calculated from the 'corrected baseline' which was obtained by the averaging the pre-stimulus amplitude values. The data was tabulated in terms of latency and amplitude for both the subject groups for both the stimuli.

Results and Discussion

Cortical potentials in normal hearing subjects

The normal hearing subjects had robust cortical potentials for all the stimuli. The results will be discussed based on the stimuli used for the eliciting the cortical potentials: /su/ and /chu/.

/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 2 and Table 2 displays the mean and standard deviations of latency and amplitude values for /su/. Ostroff, et al., (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 150 ms fricative /s/ and the change complex was obtained at a latency of around 250 ms while the change complex obtained in this study was around 230ms. Although the absolute latencies in the present study differ from those obtained in other studies, the

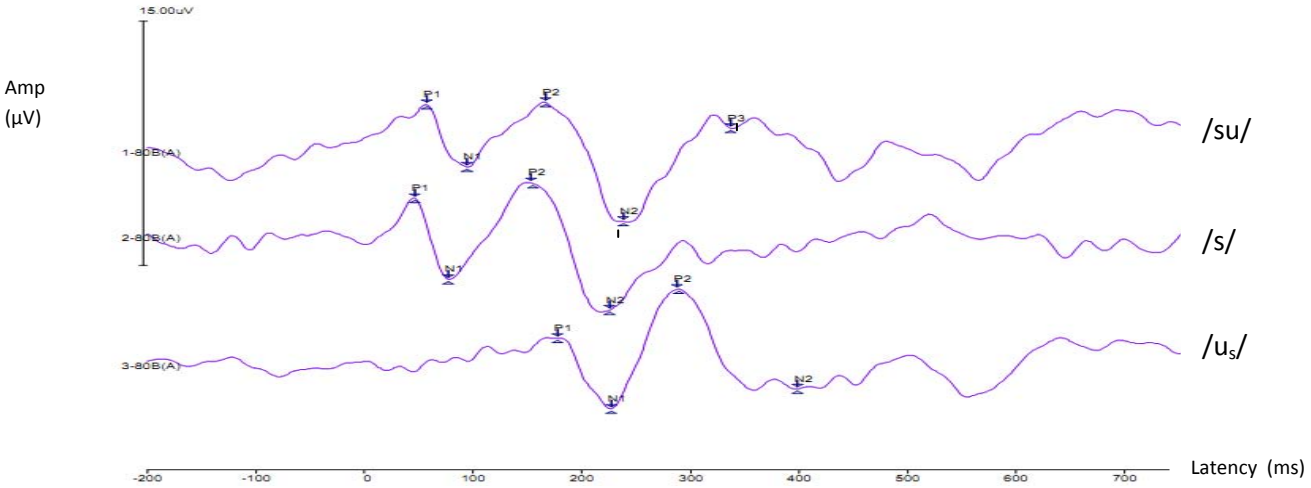

Figure 2: Grand averaged waveforms for /su/ and its components /s/ and /u_s/ in normal hearing subjects.

Table 2: Mean and standard deviations of latency and amplitude parameters for /su/ in normal hearing subjects

Wave component for /su/	Mean latency (ms)	SD for latency	Mean amplitude (μV)	SD for amplitude
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

relative latency i.e the latency with respect to the onset of the change in the stimulus remain similar. The initial P1-N1-P2 complex was also found to be similar to what was reported by the above authors.

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 pre-stimulus baseline. The combination of the waveforms for /s/ and /u/ result in the waveform similar to that obtained from /su/.

/chu/ stimulus: The change complex for /chu/ was present in all the 11 normal hearing subjects. Figure 3 displays the grand average of /chu/ (first waveform), /ch/ (second) and /u_{ch}/ (third) respectively and Table 3 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 (>100 ms) after the onset of the first component of the stimulus. For instance, Martin & Boothroyd (1999) employed a duration of 400 ms 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.

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/.

Comparison between /su/ and /chu/ in normal hearing participants: The latencies for /chu/ were much earlier than /su/ due to the earlier vowel onset in /chu/ (70 ms) than /su/ (130 ms). 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 not the case with /chu/. The

Table 3: 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	Mean amplitude (μV)	SD for amplitude
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

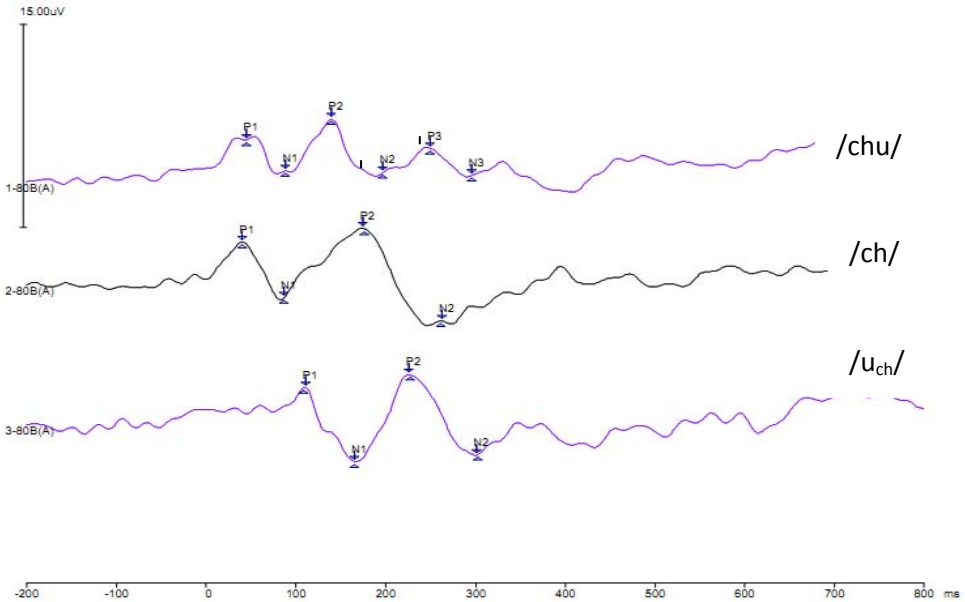

Figure 3: Grand averaged waveforms for /chu/ and its components /ch/ and /u_{ch}/ in normal hearing subjects.

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 two 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.

Cortical Potentials in ANSD participants

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.

/su/ stimulus: The ACC complex for /su/ was present in only 4 out of 10 (40%) of the subjects with ANSD. Table 4 gives the mean and standard deviations of latency and amplitude parameters for /su/ in the ANSD subjects. The values for N2’ and P3’ is only for the four subjects with ACC while the rest of the values are based upon values from all the subjects. The mean latencies and amplitudes of the P1-N1-P2 complex are in consonance with the results from the previous studies (Narne & Vanaja, 2008; Chandra & Barman, 2009). The figure 4 shows the cortical potentials for the subjects with the ACC.

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 1). Similarly, Dimitrijevic, Michalewski, Zeng, Pratt and Starr (2008, 2009) recorded cortical potentials to frequency and intensity changes in between continous tone and reported that those with good speech perception scores had greater amplitudes and better latencies. Also, Rance, Cone-Wesson, Wunderlich, and Dowell (2002) reported a good correlation between the presence of cortical potentials and speech identification scores in children.

Table 4: Mean and standard deviation of latency and amplitude for /su/ in ANSD subjects

Wave component for /su/	Mean latency (ms)	SD for latency	Mean amplitude (µV)	SD for amplitude
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

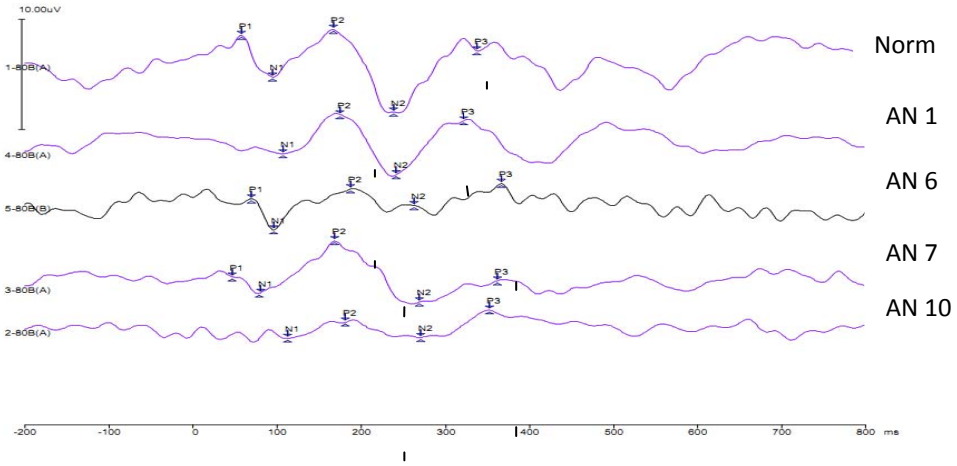

Figure 4: The cortical potentials for /su/ in ANSD subjects with ACC. Normal grand average waveform is also given for comparison.

Six subjects (AN2, AN3, AN4, AN5, AN8 & AN9) did not demonstrate an ACC complex for /chu/. These subjects typically had poor SI scores in quiet ranging from 36% to 12% (Refer Table 1). The latency and amplitude values for these subjects were widely distributed and extremely heterogeneous as has been noted in the literature (Dimitrijevic, Starr, Bhatt, Michalewski, Zeng, & Pratt, 2011; 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., (2011) reported that the latencies were prolonged in ANSD subjects with post-synaptic site of dysfunction than those with pre-synaptic site of dysfunction. Thirdly, conditions like axonal neuropathy may cause a decrease in amplitude while preserving the latency within normal limits. Figure 5 displays the waveforms of one such subject 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 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, Starr, Zeng, & Dimitrijevic, 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

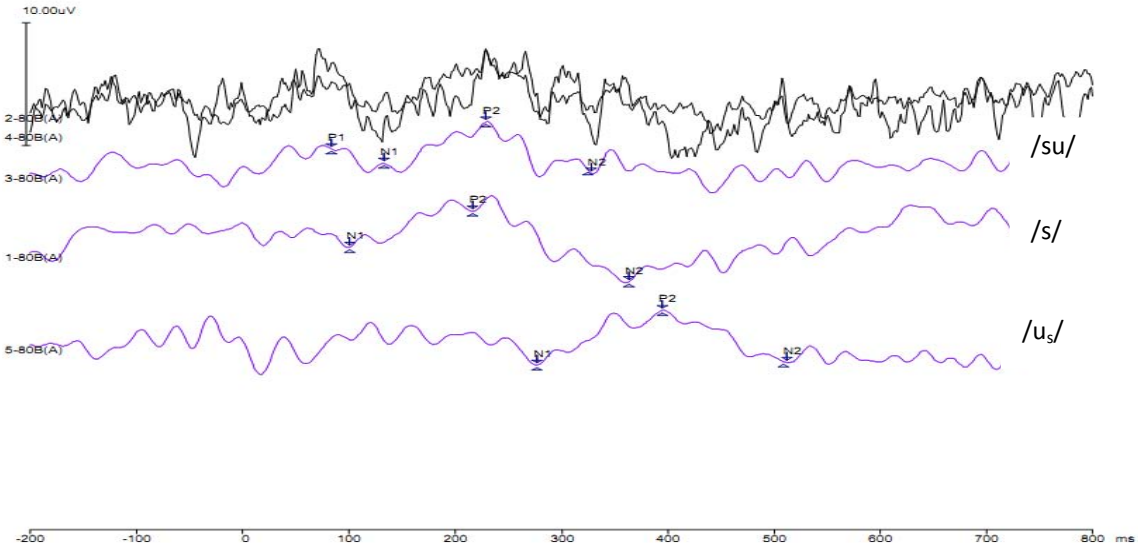

Figure 5: Cortical potentials for /su/, /s/ and /u$_s$/ in AN3.Raw waveforms are also shown for /su/ to demonstrate replication.

Table 5: Mean and standard deviations of latency and amplitude parameters for /chu/ in ANSD subjects

Wave component for /chu/	Mean latency (ms)	SD for latency	Mean amplitude (μV)	SD for amplitude
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	-	-	-	-

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.

Comparison with the control group for /su/: 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 /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.

/chu/ stimulus: Table 5 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 6 displays the ACC for control group and AN7 who demonstrated the ACC for /chu/. As can be seen from the Figure 6, 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., (2008, 2009) who reported that speech perception scores were significantly correlated with the presence of cortical potentials to changes in frequency and intensity.

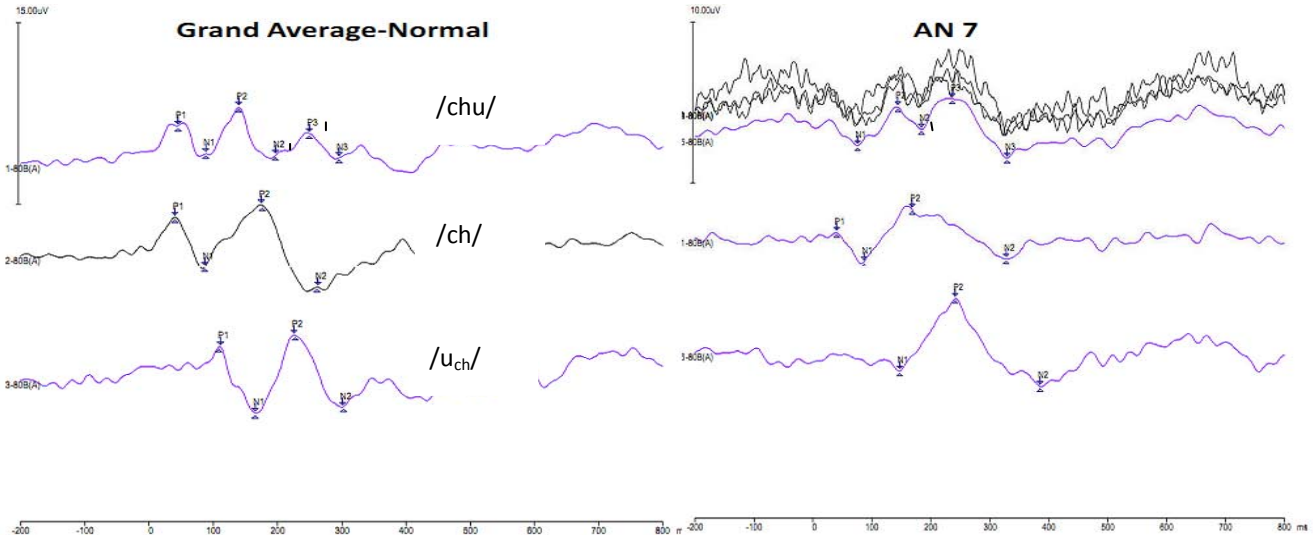

Figure 6: Cortical potentials for /chu/ in AN7. Grand average for control group is given to the left for comparison. Raw waveforms are also shown for /chu/ for subject AN7.

The rest of the nine subjects with ANSD did not reveal the presence of ACC. Figure 7 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).

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.

Comparison with the control group for /chu/: 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.

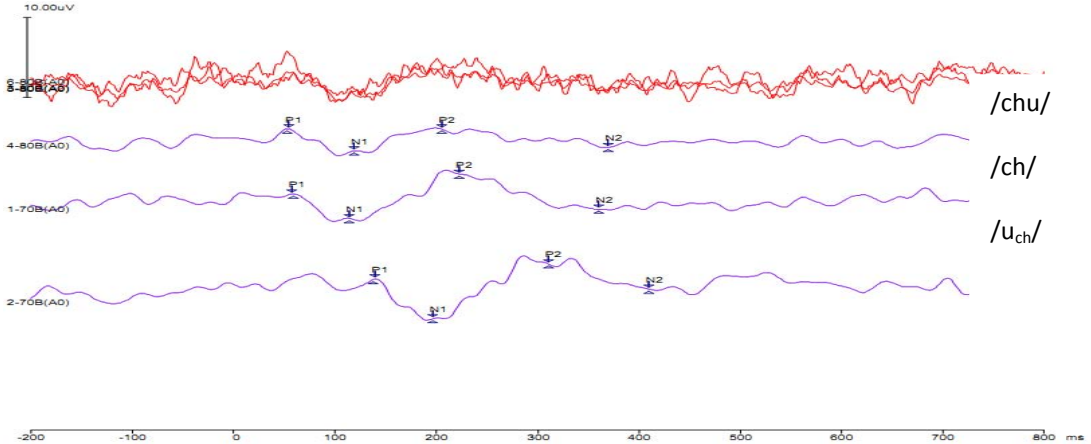

Figure 7: Cortical potentials for /chu/, /ch/ and /u_{ch}/ for the subject AN 9. Raw waveforms are also shown for /chuu/ to demonstrate replication.

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 (Starr, 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.

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 130 ms 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 70 ms, 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 133 ms for /su/ components and 72 ms for /chu/ components. The larger separation for /su/ hence resulted in a clearer ACC. This effect was also found in normals by Burger, Hoppe, Lohscheller, Eysholdt, and Dollinger (2009) when the consonant duration very small (47 ms). 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, et al., 1999).

Conclusions

This preliminary study thus revealed that the some of the participants with ANSD do have the ability to demonstrate the change complex to the consonant-vowel combinations and that its presence is associated with good speech perception abilities. The change complex was more readily observed when the two phonemes in the syllable are farther apart than when they are nearer indicating temporal resolution difficulties in the ANSD participants. The results of this study support the feasibility of utilizing cortical potentials in ANSD participants 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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