AIDED ACOUSTIC CHANGE COMPLEX IN INDIVIDUALS WITH COCHLEAR HEARING LOSS

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A Dissertation Submitted in Part Fulfilment of Final Year

Master of Science (Audiology)

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ALL INDIA INSTITUTE OF SPEECH AND HEARING

MANASAGANGOTHRI, MYSORE – 570 006

MAY, 2012.

Dedicated to

My Parents,

Brother,

Achachan and

My Guide

CERTIFICATE

This is to certify that this dissertation entitled **"Aided Acoustic Change Complex in Individuals with Cochlear Hearing Loss"** is the bonafide work submitted in part fulfillment for the Degree of Master of Science (Audiology) of the student (Registration No.: 10AUD015). 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 other Diploma or Degree.

Mysore

May, 2012

Dr. S. R. Savithri
Director
All India Institute of Speech and Hearing,
Manasagangothri, Mysore –570006.

CERTIFICATE

This is to certify that this dissertation entitled **"Aided Acoustic Change Complex in Individuals with Cochlear Hearing Loss"** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in other University for the award of any Diploma or Degree.

Mysore

May, 2012

Mr. Sreeraj K.
Guide
Lecturer in Audiology
All India Institute of Speech and Hearing,
Manasagangothri, Mysore- 570006

DECLARATION

This is to certify that this Master's dissertation entitled **"Aided Acoustic Change Complex in Individuals with Cochlear Hearing Loss"** is the result of my own study under the guidance of Mr. Sreeraj K, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in other University for the award of any Diploma or Degree.

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Chapter 1

INTRODUCTION

Sensorineural hearing loss (SNHL) is probably the most common form of hearing loss and these type of hearing loss not only lead to elevation of threshold for detection of sound but, also the affects the way in which sound is perceived. The perception of speech in individual with SNHL is also dependent the configuration of hearing loss (Coughlin, Kewely-Port & Humes, 1998; Dubino, Dirks & Schafer, 1987; Sher & Owens, 1974). In SNHL different audiometric patterns like flat, raising, sloping etc. are evident, were the most common configuration is sloping type (Pittman & Stelmachowicz, 2003). One of the most common management options for this type of permanent hearing loss are hearing aids. Hearing aids can compensate sensorineural hearing loss by amplifying sound. Despite adequate amplification of sound by hearing aid, the person with cochlear hearing loss continues to report unclear and distorted speech. The effectiveness of hearing aid in person with SNHL also depends on the ability of the central auditory system to represent and integrate the spectral and temporal information delivered by the hearing aid, other than hearing aid related factors.

The individuals with SNHL avoid the use of hearing aids or adjust to the listening situations without the use of hearing aids. This kind of avoidance and adjustments for a longer period of time can lead to auditory deprivation (Silman, Gelfand, & Silverman, 1984). Hence, there is interest in examining the neural representation (by hearing aid) of speech cues and amplified speech cues in people

with hearing loss. It will be still more interesting, if it is possible to get knowledge about the different durations of hearing loss and its effect on neural representation of speech cues and amplified speech cues (by hearing aid) in people with hearing loss.

The P1-N1-P2 complex is first tool for assessing the neural representation of sound in population with and without hearing loss. The P1-N1-P2 complex is an auditory evoked potential that is characterized by a positive peak (P1), followed by a negative peak with a latency of about 100 milliseconds after stimulus onset (N1), followed by a positive peak called P2. These peaks reflect neural activity generated by multiple sources in the thalamic-cortical segment of the central auditory system. Multiple overlapping P1-N1-P2 responses can be seen in response to naturally produced speech stimuli like syllables, words etc. If we consider multiple overlapping responses to a syllable, the first P1-N1-P2 complex reflects the onset of the consonant whereas the second P1-N1-P2 response reflects the consonant vowel (CV) transition. These complex waveform patterns were shown to reflect acoustic changes, from silence to sound (onset of consonant) and the CV transition (from consonant to vowel) (Ostroff, Martin& Boothroyd, 1998). Martin and Boothroyd in 1999, termed this cortical evoked response as 'Acoustic Change Complex' (ACC). ACC and other cortical potentials like mismatch negativity (MMN), P300 give information on the auditory discrimination ability of individual. But, to elicit MMN and P300, individual's co-operation is required whereas for ACC, the same is not mandatory. Moreover, the average amplitude of ACC is 2.5 time larger compare to MMN in normal hearing individuals (Martin & Boothroyd, 1999). These advantages of ACC over other potentials in normal hearing individuals gives more interest to probe in to study effect of hearing impairment and amplification on cortical representation of acoustic changes within speech sounds. It is still more imperative to study effect of

sloping sensorineural hearing loss (SNHL) on ACC, reason behind is that individual with sloping SNHL has more problem with perception of speech because the higher frequency speech sounds contributes primarily to the speech intelligibility. Hence, the high frequency speech sounds like fricatives within a syllable can be used to elicit ACC. Very few researchers gave more interest to probe more in to it (Tremblay, Billings, Friesen & Souza, 2006, Tremblay, Friesen, Martin & Wright, 2003).

Tremblay et al., (2006), found that /shee/ and /si/ each elicit distinct ACC responses. The first negative peak, signaling the onset of the consonant, was not significantly different for the /shee/ and /si/ stimuli. However, the second P1-N1-P2 complex (N345 and P413), presumably reflecting the CV transition, occurred significantly earlier when evoked by the /shee/ stimulus than when evoked by /see/. Onset of the vowel in /shee/ was 30 milliseconds earlier than the onset of the vowel portion in /see/. This 30-millisecond difference appears to correspond to the 30-millisecond latency difference between the negative peak N345 elicited by the /shee/ stimulus and the negative peak N375 elicited by the /si/ stimulus.

Tremblay et al., (2003), examined ACC patterns in normal-hearing young adults, evoked by two different speech stimuli, /shee/ and /si/. These particular stimuli were chosen because they share similar acoustic features and are frequently confused by listeners with hearing loss. /shee/ and /si/ are similar in that they are fricatives and different in that (1) /shee/ and /si/ differ by place of articulation, (2) the fricative portion of /shee/ contains lower spectral energy than the fricative portion of /si/, and (3) the fricative portion of /shee/ is shorter in duration than the fricative portion of /si/. Karthik and Vanaja (2005), demonstrated that ACC is an electrophysiological index of speech discrimination in adult and children. The study showed that ACC could be

recorded in all adult subjects and there was a significant difference between N1P2 amplitudes between two stimuli indicative presence of ACC response.

All these research evidences suggest that ACC can be recorded successfully in adult normal hearing individuals. So, there is a need to know how successfully the aided ACC can be recorded in person with cochlear hearing loss and to correlate the findings to various degree and duration of hearing loss. In a study by Tremblay, Kalstein, Billings and Souza, 2006, recorded ACC in adult hearing aid users with mild to severe degree of sloping sensorineural hearing loss for two consonant-vowel (CV) syllables (/shee/ and /si/). The result of the study showed that /shee/ and /see/ elicited different waveforms in terms of latency and amplitude. This finding indicated different neural detection of CV transitions (indicated by the presence of a P1-N1-P2 response) for /shee/ and /si/.The latency of the second N1 in the evoked cortical neural response coincided in time with the onset of the vowel in the /shee/ and /si/ syllable. This finding is in accordance with the study done by Tremblay, Billings, Friesen, and Souza in 2006. To critically evaluate, the study demonstrated the coding of acoustic changes in the subjects with mild to severe sloping sensorineural hearing loss in aided condition but did not studied the effect of mild to severe degree of SNHL on ACC. Moreover, duration of sloping SNHL along with the degree of hearing loss could also have impact on unaided and aided ACC.

Interestingly, the further studies in the literature also failed to make an attempt to study effect of degree and duration of sloping SNHL on unaided and aided ACC. This leads to the initiation of the current study.

Need for the Study

Firstly**,** the persons with sloping SNHL have problems in perceiving acoustic changes within speech sounds (mainly the syllables having high frequency consonant and low frequency vowel combination). The ability to perceive these acoustic changes is very important for normal perception of speech. How well the acoustic changes within the speech stimuli are coded in the cortical level can be studied electro physiologically using ACC. So, there is a need to study the ACC in individual with varying degree of sloping hearing loss to see cortical representation of acoustic changes within speech sounds.

Secondly, the amount of benefit the hearing aid provides varies greatly among individual with sloping sensorineural hearing loss. The factors contributing could be that the hearing aid is incapable to process spectral and temporal information properly or could be the inability of the peripheral and central auditory system in processing acoustic changes within speech sounds provided through hearing aid. Hence, there is also a need to study the neural representation of (cortical representation) acoustic changes within speech sounds through a hearing aid in individual with sloping SNHL across the various durations of losses.

Thirdly, the degree and duration of hearing loss could be the factors that affect the cortical representation of acoustical changes within speech sounds in individual with sloping SNHL. Hence, there is a need to compare the effect of different degrees across various duration of hearing loss with and without the use of amplification, and study the difference in cortical representation of speech.

Aim of the study

The aim of the study was to investigate the effect of degree and duration of sloping sensori-neural hearing loss and digitally amplified speech on Acoustic Change Complex.

Objectives of the study

1) To compare the unaided & aided ACC separately between minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss.
2) To compare between less than or greater than two years of duration of hearing loss in minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss separately.
3) To compare the unaided and aided ACC within each subgroup to see the effectiveness of amplification:
    a. Minimal to moderate with duration of hearing loss less than two years (subgroup A).
    b. Minimal to moderate with duration of hearing loss greater than two years (subgroup B).
    c. Moderate to severe with duration of hearing loss less than two years (subgroup C).
    d. Moderate to severe with duration of hearing loss greater than two years (subgroup D).

Chapter 2

REVIEW OF LITERATURE

Sensorineural hearing loss is one of the most commonly seen types of hearing loss. It can be either of cochlear origin or neural origin. Cochlear hearing loss can manifest itself in various configurations such as rising, flat or sloping of which sloping is the one we usually come across (Pittman & Stelmachowicz, 2003). The management options for individuals with cochlear hearing loss are amplification devices which basically try to compensate the hearing loss by amplifying the signal. Unfortunately, for most of the individuals with sloping cochlear hearing loss, these amplification devices will do nothing much to lessen their discomfort and frustration in their listening environment.

The spectral and temporal cues serve as the most important elements in speech perception since they aid in differentiating speech sounds from one another. Some of the individuals with sloping cochlear hearing loss are able to utilize these cues to some extent and compensate their listening difficulty in their daily life listening situations without the help of amplification devices. But, avoidance of use hearing aids for a longer duration can lead to auditory deprivation leading to increase in degree of hearing loss (Silman, Gelfand & Silverman, 1984). This addition to the prevailing hearing loss significantly affects perception of speech and communication needs in the daily listening situations. After which the individual would turn towards the amplification devices for assistance but in such a case, the increase in hearing loss and the amount of auditory deprivation might also play a role in the benefit gained from hearing aids.

The reasons for lack of benefit from hearing aids can be attributed to hearing aid itself and/or individual's peripheral and central auditory systems which might be unable to process the spectral and temporal cues adequately. Hence, the current study was carried out to assess the cortical neural representation of on-going changes in speech sounds with and without amplification in naive adult hearing aids users.

The review of literature of the current study can be divided into three broad categories;

1) Speech evoked cortical potentials in individuals with cochlear hearing loss

2) Aided speech evoked cortical potentials in cochlear hearing loss

3) Speech evoked potentials and auditory deprivation

All the above mentioned categories throw light on the neural representation of speech sounds and the on-going changes in them with and without amplification in individuals with normal hearing and individuals with cochlear hearing loss.

2.1 Speech evoked cortical potentials in individuals with cochlear hearing loss

Auditory Long Latency Response (ALLR), Acoustic Change Complex (ACC), Mismatch negativity (MMN) and P300 form a small part of the broad range of cortical potentials accessible to human experimentation. These aforementioned evoked potentials provide an insight into the neural mechanism underlying speech processing.

ALLR is the first reported evoked potential in the literature (Geiseler, Frishkopf, & Rosenblith, 1958; Davis, 1939), which was used to estimate the hearing sensitivity (Appleby, Mc Dermick & Scott, 1963; Beagley & Kellong, 1969; Cody & Brickford, 1965; Davis, 1966; Davis, Hirsch & Shelnott, 1967; Perl, Galambos, &

Glorig, 1953; Mendel, Hosick & Windman, 1975), before Auditory Brainstem Response (ABR) came into picture. But after the discovery of ABR, these potentials lost scope in hearing sensitivity assessment as ABR is not affected by attention, sleep and sedation unlike these potentials, and is highly replicable in individual subjects (Stapells, Picton & Durrieux-smith, 1994; Stapells, Gravel & Martin, 1995). So, even today ABR is mainly used for assessing hearing sensitivity in infants, young children and adults regardless of whether they are awake or asleep. Hence, the interest in the utility of ALLR shifted to the assessment of suprathreshold auditory skills such as speech perception.

Very few studies have investigated the effect of cochlear hearing loss on speech evoked cortical potentials. One of the earlier studies was by Polen (1984), who compared the group of individuals with moderate to severe SNHL with a group of listeners with normal hearing sensitivity. Speech sounds /v/ and /ʃ/ served as the stimuli to evoke the ALLR. The result of the study clearly indicated prolongation of the N1, P2, N2, & P3 latencies and reduction of P2 and N2 amplitudes in individuals with hearing loss. But, Polen did not have a homogenous hearing impaired group and the presentation levels used were considerably lower and closer to the hearing thresholds.

Later in 1991, Wall, Dallebout, Davidson and Fox gave an idea about the effects of mild to moderate SNHL on ALLR. The study was conducted on five subjects using the stimuli /be/- /de/ and /be/ - /pe/ voicing contrasts. The stimuli were presented at 40dBSL for clinical and normal hearing control group. The results showed that N1 amplitudes were significantly smaller in the clinical group compared to control group. The authors reasoned this out as some frequency information reaching the auditory cortical structures would be lost and this loss of ‘information

load would be reflected in N1 amplitude. This study could overcome the pitfalls in the Polen's study.

Oates, Katzberg and Stapells (2002), investigated the effect of mild to moderate (hearing thresholds ranged from 25 to 49 dBHL) and severe to profound (75 to 120 dBHL) sensorineural hearing loss (SNHL) on cortical ERPs N1, MMN, N2, and P3 and their associated behavior measures (d' and reaction time) to speech sounds /ba/ and /da/ presented at 65 to 80 dBpeSPL. They found that ERP amplitude and behavior discrimination /d'/ scores were lower for listeners with SNHL than listeners with normal hearing. Results of this study indicated that latency measures were more sensitive indicator of early effect of decreased audibility than a response strength measure (amplitude, d', or percentage correct measures). The latency and amplitude response changes that occurred with SNHL for later ERP peaks (N2/P3) and behavior discrimination measures (d' and reaction time) were significantly greater in comparison with N1/MMN. This indicates SNHL has greater impact on higher level cortical processing. The authors concluded that the feasibility of MMN as a diagnostic tool for hearing impaired population is limited in comparison with other cortical potentials like N1, N2 & P300 due its high variability and lower detection rate.

Kortzak, Kurzbag and Stapells (2005), also demonstrated prolongation in N1 latency elicited by /ba-da/ pair in 14 adults with hearing loss which ranged from moderate to profound as compared to 20 normal hearing participants.

Next, a series of simulated studies followed, which focused on the effect of different configurations of hearing loss on evoked potentials. The study by Martin, Signal, Katzberg, and Stapells (1997), investigated the effects of decreased audibility brought about by high-pass noise masker on ALLR. The stimuli used were speech sounds /ba/ and /da/presented at 65 and 80 dBSPL. They recorded ALLR in quiet and

in the high pass filtered noise at different cut off frequencies of 4 kHz, 2 kHz, 1 kHz, 500 Hz and 250 Hz presented at intensity sufficient to completely mask the response to the 65-dB SPL speech sounds. The result of the study indicated that as the cut off frequency of the high-pass masker was lowered, N1 and N2 latencies increased and amplitudes decreased. The latency of N1 was longer for the 65-dB SPL speech sounds than that for 80-dB SPL and to /ba/ than to /da/ even in the quiet condition. N1 amplitude reduction was gradual from 4 KHz to 2 KHz cut off frequency and further decrease in cut off frequency lead to steep drop in amplitude. The amplitude of N1 was higher in quiet compared to all high pass masking noise conditions. N2 latencies increased and amplitude decreased with decrease in high-pass noise cut off frequency. The N1 reflected audibility in the stimulus whereas N2 reflected discriminability. The reason for reduction in amplitude and prolongation of latency were more for N2 compared N1 is that audibility of any portion of the stimulus such as only the vowel or fundamental frequency is sufficient to elicit N1 whereas, mid to high frequency content of the stimulus is essential to elicit N2. Since, high pass masking noise (Simulating high frequency hearing loss) was used which is assumed to reduce the mid to high frequency content of the stimulus.

Whiting, Martin, and Stapells (1998), aimed to find out whether mild to moderate degree of hearing loss will have an effect on speech evoked cortical potentials. They simulated mild to moderate degree of hearing loss in normal hearing individuals by using broad band noise (BBN). BBN elevated thresholds for low frequencies around mild degree and high frequencies around moderate degree. They found out that even mild to moderate reduction in audibility can lead to significant prolongation of N1 and N2 latencies. Compared to N2 the prolongation for N1 was lesser. The reason for this finding given by the authors is same as their own previous

study is that audibility of any portion of the stimulus such as only the vowel or fundamental frequency is sufficient to elicit N1 whereas, mid to high frequency content of the stimulus is essential to elicit N2. Since, broad band masking noise was used which is assumed to reduce the mid to high frequency content of the stimulus (depends on the cut-off frequency used in the study N2 latency was prolonged to a greater extent compared to that of N1).

Martin, Kurtzberg and Stapells in 1999, went one step forward and studied both the behavioral measures (d' & reaction time) and N1 evoked potential elicited by speech sounds /ba/ and /da/ presented at 65 dB SPL, in the presence of high pass masking noise. The testing was done in quiet and in presence of high pass noise with cut off frequencies of 4 kHz, 2 kHz, 1 kHz, 500 Hz and 250 Hz. The results showed reduction in behavioral d' scores and increase in reaction time as the noise cut off frequency lowered below 2 KHz. The quoted reason for this finding was that the frequency region containing the second formant transition for the speech sounds was masked by the high pass masking noise at cut off frequencies below 2 KHz, hence the poor scores. Progressively increasing the cut off frequency of high pass noise progressively increased the N1 latency. The N1 latency was significantly lower in quiet compared to high pass noise conditions. The amplitude of N1 decreased as the high pass noise cut off frequency was lowered. The N1 amplitude and latency was higher for /ba/ than that for /da/. The explanation given for the higher N1 amplitude was that, since /ba/ is of lower frequency content than /da/ and it is known that N1 is larger in response to low frequency tones than for high frequency (Picton, Woods & Proulex, 1978), N1 amplitude was higher for /ba/. Whereas, the reason for latency prolongation was the larger travelling delay for low frequency /ba/ stimulus compared to high frequency /da/ stimulus.

Though simulated hearing loss studies come handy with many advantages, they are not without cons. It is important to determine whether sensorineural hearing loss produces similar response changes in cortical ERPs as simulated hearing loss. There are several factors that could result in differences between these two groups. To name a few, differences in frequency, intensity, and/or temporal processing abilities; age of onset of hearing loss; duration of hearing loss and the effects of central masking in the simulated hearing loss group. The effects of peripheral hearing loss on the response properties, synchrony, and organization of cortical neurons may also affect the ERPs in ways different from those found in the simulations of hearing loss.

In summary, the aforementioned studies convey that to elicit N1, audibility is important and it is less frequency dependent where as to elicit N2, mid to high frequency content of the stimulus is important which deals with discrimination. The difference evidenced between simulated studies and studies dealing with sensorineural hearing loss is that the latency prolongation is more in individuals with actual hearing loss compared to simulated hearing loss.

One more evoked potential which helps in assessing the speech perception ability in individuals with hearing loss is the Mismatch Negativity (MMN). It is evident from MMN studies that MMN can be elicited in individuals with cochlear hearing loss (Oates, Kurtzberg & Stapells, 2002; Korczak, Kurtzberg & Stapells, 2005), but, it depends on their behavioral discrimination ability. One such study was conducted by Kraus, McGee, Carrel, and Sharma in 1995 wherein, they studied MMN in two adult listeners with sloping sensorineural loss. Both participants had similar configuration of audiogram (mild low frequency hearing loss sloping to moderate to severe loss for the high frequencies). They elicited MMN to a /da/-/ga/ contrast stimuli. One participant had poor behavioral discrimination of the contrast whereas;

the other had good behavioral discrimination of the contrast. The result of the study showed that the first participant with poor behavioral discrimination had no MMN. But, the second participant with good behavioral discrimination had a clear MMN.

However, there are certain limitations to the use of MMN in studying the speech perception ability. The amplitude of MMN is inferior in relation to background EEG activity (Picton, 1995). In addition, to increase the signal to noise ratio, a greater amount of response averaging is required which is not practically feasible (Martin & Boothroyd, 1999). There is, however, another similar evoked potential which comes handy in assessing the speech perception ability, i.e., the P300. P300 is found to be more sensitive to sensorineural hearing loss compared to other potentials (Wall et al., 1991; Oates et al., 2002; Kortzak, Kurzbag & Stapells, 2005). But to elicit P300, individual's co-operation is required, whereas for another evoked potential, namely the Acoustic Change Complex (ACC), the same is not mandatory. Also, P300 potential gives us an idea about the voluntary discrimination ability between any two stimuli whereas ACC throws light on the cortical neural coding of acoustical changes within a syllable. ACC amplitude is three times bigger than MMN and easier to elicit. These advantages of ACC over other potentials gave more interest to the researchers to probe more into it.

The time varying changes within a signal evoke cortical auditory evoked potentials (CAEP) consisting of complex multiple overlapping P1-N1-P2-N2 complexes. These overlapping P1-N1-P2-N2 complexes reflect the acoustic changes across the entire stimulus (Ostroff et. al, 1998). This collection of overlapping potentials has been named as Acoustic Change Complex by Martin and Boothroyd (1999). Further, its test retest reliability was questioned by a few researchers.

Tremblay, Friesen, Martin, and Wright (2003), examined the test retest reliability of ACC in seven young adult native English speakers with normal hearing sensitivity elicited by naturally produced speech stimuli /bi/, /pi/, /ʃi/ and /si/. Tests and retests were conducted within an 8 day period. The result of the study indicated that ACC can be reliably recorded by naturally produced speech sounds. The voiced stimulus /bi/ evoked responses which were different from /pi/. The negative (N130) and positive peaks (P217) amplitude were larger in response to /bi/ stimulus. But, there was no significant latency difference between /bi/ and /pi/. The waveform patterns evoked by /ʃi/ and /si/ were also different from each other. The potentials N345, P413 and N519 appeared earlier in latency in response to /ʃi/. The reason being given for this finding was that the stimulus /ʃi/ had earlier onset of vocalic portion because of lesser fricative duration in comparison to /si/ stimulus.

Another study was carried out by Martin and Boothroyd (in press), which examined the effects of sensorineural hearing loss on ACC as well as the relationship between behavioral measures of speech discrimination and ACC. ACC was elicited by nine stimuli containing a range of second formant frequency changes in 10 adults with sensorineural hearing loss. The stimuli ranged from a 1200 Hz acoustic change at stimulus midpoint (perceived as /ui/) to a stimulus containing no acoustic change at midpoint (perceived as a long /u/). Participants were asked to press one response button if they perceived no change at stimulus midpoint, and another if they perceived an acoustic change at stimulus midpoint. Stimuli were presented at the participant's highest comfortable listening level. Results showed that ACC was clearly elicited in individuals with moderate sensorineural hearing loss. It was noticed that the amplitude decreased and latency increased as the amount of second formant frequency change decreased. It was also found out that ACC thresholds showed good

agreement with behavioral thresholds. These results are essentially consistent with those obtained by Ostroff (1998) in a similar study involving listeners with normal hearing, except that ACC and behavioral thresholds were elevated in the current study, as would be expected for listeners with hearing loss.

2.2 Aided speech evoked cortical potentials in cochlear hearing loss

To date, only a few studies have examined the effects of amplification on cortical ERPs in response to speech stimuli. Early studies were typically individual case studies or studies with a smaller number of participants and showed somewhat conflicting results. For example, Rapine and Grazianni (1967), found that the majority (5 of 8) of 5 to 24-month-old subjects with severe to profound sensorineural hearing loss showed an improvement in ERP thresholds of at least 20 dB in the aided, compared to the unaided condition. However, two of the infants showed no change in their cortical ERP thresholds between the aided and unaided conditions.

Stapells and Kurtzberg (1991), compared cortical ERPs with speech stimuli in the aided versus unaided conditions in four children with moderate to profound sensorineural hearing loss. Three of them showed clear obligatory P1 responses, which were followed by a prominent negativity (N2) only in the aided condition. In contrast, one child with a progressive hearing loss initially demonstrated a large obligatory response in the aided condition that later disappeared when she could no longer behaviorally respond to the sound.

Kraus and McGee (1994), held a study on two adults with sensorineural hearing loss. One individual had good behavioral discrimination of the /ta/-/da/ contrast while using a hearing aid and showed a present MMN and P3, whereas the other participant had poor behavioral discrimination of the contrast, even with a hearing aid, and had an absent MMN but a present P3. One possible explanation for the conflicting results

in these earlier studies may have been the functional status of the hearing aids at the time of testing.

In the above mentioned studies, hearing aid functioning was not evaluated by electroacoustic measurements or real ear measurements. Studies are now emerging in which the status of the hearing aid before testing are being objectively evaluated to more systematically appraise the combined effects of sensorineural hearing loss and hearing aids in adults and children.

Kortzak, Kurzbag & Stapells, 2005 examined cortical ERPs (N1, MMN, N2 & P3) to a /ba/-/da/ contrast presented at 65 and 80 dB peSPL in a relatively large group of listeners with either moderate or severe-profound sensorineural hearing loss with and without their hearing aids, and a control group of normal hearing adults. ERPs were recorded in both active (deviant stimulus response) and passive (stimuli ignored) conditions. This study was unique, in that hearing aid function was checked with both electroacoustic and real-ear measurement techniques before testing. Results indicated that the use of hearing aids improved the detestability of all the cortical ERPs particularly for individuals with severe-profound hearing loss. Secondly, the mean amplitudes and latencies of all cortical ERPs as well as the behavioral indices of discrimination were improved in the aided conditions compared with the unaided conditions. This was especially true for the lower stimulus intensity. Finally, even though the majority of hearing-impaired subjects showed increased amplitudes, decreased latencies, and improved waveform morphology in the aided conditions, the amount of response change was quite variable across individual participants. Some individuals showed large improvements; whereas, others showed little or no improvements with amplification which might be because sensori-neural hearing loss involves the factors other than reduced audibility.

Korczak, et al., 2005, compared the aided results of the hearing-impaired individuals with the normal control participants. Results of these analyses showed that even with adequately functioning hearing aids, most subjects had prolonged ERP latencies and reaction times (RTs) along with reduced amplitudes relative to the normal hearing group, especially at the lower stimulus intensity. This study demonstrated that, although the use of hearing aids resulted in improved response measures, individuals with hearing loss process speech in a less effective manner (reflected by increased latencies and decreased amplitudes) and with less accuracy (reflected by poorer behavioral discrimination performance) compared with their normally hearing counterparts, even while wearing their hearing aids.

Cortical ERPs to aided speech stimuli have also been successfully recorded in a small group of school-aged children with sensorineural hearing loss (Stapells, 2002).

Preliminary yet promising data from a large group of infants and children with sensorineural hearing loss indicate that aided, obligatory cortical ERPs can be successfully recorded to conversational-level speech stimuli, in individuals with mild to severe losses (Purdy, Kelly &Thorne, 2003). Obligatory cortical responses were recorded to several speech stimuli presented at a normal conversational level (65 dB SPL) in infants and young children with normal hearing as well as those with sensorineural hearing loss who listened with their personal hearing aids. Similar to results shown in adults, the detestability of the aided response to 65 dB SPL speech stimuli was quite high for the mild/ moderate and severely impaired groups (100% and 80%, respectively) and declined to 50% for the profoundly impaired group. For several children in the mild to severe groups, the aided obligatory response seemed to be sensitive to changes in the frequency content of the speech stimuli, thus suggesting

this response may serve as a useful tool in assisting audiologists' to fine-tune hearing aids.

Collectively, these preliminary findings suggest that cortical ERPs might be a useful clinical tool for assessing hearing aid benefit in the younger hearing-impaired population and may be of assistance to audiologists in initially fitting and adjusting these instruments. Later on, the studies focused on aided ACC.

The first study on aided ACC was done by Tremblay, Kalstien, Cuttis, Billing and Souza (2006). The aims of the study were to determine the test retest reliability of amplified natural speech (/si/ and /ʃi/) evoked ACC, effect of amplification on ACC and whether different amplified speech sounds evoke different ACC or not. To study the effect of amplification regardless of hearing loss they took seven normal hearing English speaking adults in the age range of 21 to 31 years as participants of the study. They recorded unaided and aided ACC in these subjects for /si/ and /ʃi/ stimuli. The result of the study indicated that speech evoked ACC can be reliably recorded in both aided and unaided conditions; hearing aids that provide mild high frequency gain only subtly enhance peak amplitudes relative to unaided recordings. The acoustic changes within the stimuli (/si/ and /ʃi/) were neurally represented clearly with and without wearing hearing aids as authors were able to elicit unaided and aided ACC without any significant latency difference.

In the above mentioned study aided ACC was done in normal hearing individuals not in individuals with hearing loss. The reason behind this was that the authors wanted to demonstrate only the effect of hearing aid on ACC and wanted to remove the factor of hearing loss because both the factors such as hearing aid functioning and hearing loss can have a combined effect on the aided ACC recorded in individual with sensorineural hearing loss.

Tremblay, Kalstein, Billings and Souza (2006), did another study on aided ACC to know more about the combined effect of amplification and hearing loss on the cortical neural representation of CV transitions. Stimuli used were /si/ and /ʃi/. Seven subjects aged 50 to 76 years with bilateral mild to severe sensorineural hearing loss were considered for the study. They found that the group mean waveforms from all electrode sites demonstrated distinct response to /si/and /ʃi/ stimuli. The second negative peak in the ACC waveform appeared at a latency of 331.86msec for /ʃi/ and 361.43msec for /si/. So, there was a significant difference between the responses of the two stimuli. The second finding of the study was that the amplification did not significantly alter the onset of /s/ and /ʃ/, because they did not find any latency or amplitude difference in the first negative peak obtained for /si/and /ʃi/ stimuli. To conclude, this study demonstrated that acoustic changes within a syllable (fricative + vowel) are normally represented in the auditory cortex in subject with bilateral mild to severe sloping sensorineural hearing loss. To critically evaluate, this study did not demonstrate the coding of acoustic changes in the subjects with mild to severe sloping sensorineural hearing loss in aided condition. This leads to the initiation of the current study.

2.3 Speech evoked cortical potentials and auditory deprivation.

Arlinger, Gatehouse, Bentler, Bryne & Cox (1996), defined auditory deprivation as a "systematic decrease over time in auditory performance associated with the reduced availability of acoustic information."

Silman, Gelfand, and Silverman (1984), reported that adults with bilateral sensorineural hearing loss, who use a single hearing aid, exhibit a phenomenon in the unaided ear that they termed as “late-onset auditory deprivation.” Late-onset auditory deprivation may also be seen in both the ears in individuals with bilateral

sensorineural hearing loss who don't use amplification. This can be studied objectively through the use of cortical evoked potentials. CAEP can reflect the changes in neural activity associated with auditory deprivation and auditory stimulation (auditory training).

The first attempt to study CAEP and auditory deprivation was done by Ponton (2001), who assessed CNS activity by measuring long-latency auditory evoked potentials (AEPs) recorded from teens and adults with late-onset (post-childhood) profound unilateral deafness. Compared to monaurally simulated normal-hearing subjects, the AEPs recorded from central electrode sites located over auditory cortical areas showed significant increases in inter-hemispheric waveform cross-correlation coefficients, and in inter-hemispheric AEP peak amplitude correlations. These increases provide evidence of substantial changes from the normal pattern of asymmetrical (contralateral Ipsilateral amplitude) and asynchronous (contralateral earlier than Ipsilateral) central auditory system activation in the normal hearing population to a much more symmetrical and synchronous activation in the unilaterally deaf. These cross-sectional analyses of AEP data recorded from the unilaterally deaf also suggest that the changes in cortical activity occur gradually and continue for at least 2 years after the onset of hearing loss. Analyses of peak amplitude correlations suggest that the increased inter-hemispheric symmetry may be a consequence of changes in the generators producing the N1 potential. These experience-related changes in central auditory system activity following late-onset profound unilateral deafness thus provide evidence of the presence and the time course of auditory system plasticity in the adult brain.

However, there is a dearth of studies which exactly target the effect of long duration of onset unilateral or bilateral sloping hearing loss on CAEP.

Chapter 3

METHOD

The purpose of study was to investigate the effect of degree and duration of sloping sensori-neural hearing loss and digitally amplified speech on Acoustic Change Complex. The specific objectives formulated were to compare the unaided & aided ACC separately (1) between minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss, (2) between less than or greater than two years of duration of hearing loss in minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss separately, (3) between unaided and aided ACC within each subgroup.

3.1 Participants

Total of 19 individuals with unilateral or bilateral sloping sensorineural hearing loss who are the naïve hearing aid users were selected as participants of the study. The participants were in the age range of 19 -55 years (17 males and two females with a mean age range of 41.31 years) and the hearing loss was post lingual in onset. A total of 21 ears with sloping sensorineural hearing loss were selected for the recording of aided and the unaided ACC who has less than or greater than two years of duration hearing loss. The slope of the audiogram was defined based on the occurrence of the thresholds at equal or successively higher levels from 250 to 8000 Hz and the difference between thresholds at 250 and 8000 Hz was always >20 dB (Pittman & Stelmachowicz, 2003). The degree of the slope of audiogram was calculated based on PTA_1 (average of the pure tone thresholds at 500Hz, 1KHz, 2KHz) and PTA_2 (average of the pure tone thresholds at 1KHz, 2KHz, 4KHz). All 27 ears were divided in to two groups, group one had 10 ears with minimal to moderate

sloping cochlear hearing loss and group two had 11 ears with moderate to severe sloping sensorineural hearing loss. The number of ears in the group I and group II were categorized in to four subgroups based on duration of hearing loss; minimal to moderate sloping sensorineural hearing loss (SNHL) with duration of hearing loss less than two years (subgroup A), minimal to moderate sloping SNHL with duration of hearing loss greater than two years (subgroup B), moderate to severe sloping SNHL with duration of hearing loss less than two years (subgroup C), moderate to severe sloping SNHL with duration of hearing loss greater than two years (subgroup D) as Shown in the Table 3.1 below.

Table 3.1: Number of ears selected in each group based on the durations of hearing loss

SI.No	***Degree of Sloping cochlear hearing loss.***	***Duration of hearing loss (Number of ears)***	
		<2 years	***>2 years***
1.	Minimal to moderate	6	4
2.	Moderate to severe	4	7

Participant selection criteria

Group I

Ten ears in the Group I were selected based on the following audiological findings:

i) Pure tone Audiometry**:** Pure tone audiometry results shows minimal to moderate sloping sensorineural hearing loss. The air bone gap of less than 10 dBHL from 1 kHz to 4 kHz and lesser than 20 dBHL for 250 HZ and 500 Hz. The range between PTA_1 (average of the pure tone thresholds at

500 Hz, 1 kHz, 2 kHz) and PTA_2 (average of the pure tone thresholds at 1 kHz, 2 kHz, 4 kHz) were within 16 to 55dBHL.

ii) Acoustic Immittance: Normal middle ear functioning as indicated by normal otoscopic and immittance finding. Typanometric finding of A Type Tympanogram with ear canal volume less than 2cc, tympanometric peak pressure within +/- 100 da pa and static compliance less than 1.6cc. Acoustic reflexometric findings of bilateral or unilateral presence of Ipsilateral and contralateral acoustic reflexes for pure tone frequencies 500Hz, 1 kHz, 2 kHz and for broad band noise.

iii) Speech identification scores: Speech identification scores more than 70% in the test ear.

Group II

12 ears in the group II were selected based on based on following audiological findings:

i) Pure tone Audiometry**:** Pure tone audiometry results shows moderate to severe sloping sensorineural hearing loss. The Air bone gap of less than 10 dBHL from 1 KHz to 4 KHz and 20 dBHL for 250HZ, 500Hz respectively. The range between PTA_1 (average of the pure tone thresholds at 500Hz, 1 KHz, 2 KHz) and PTA_2 (average of the pure tone thresholds at 1 KHz, 2 KHz, 4 KHz) were within 56 dBHL to 90 dBHL.

ii) Acoustic Immittance: Normal middle ear functioning indicative normal otoscopic and Immittance finding. Typanometric finding of A Type Tympanogram with ear canal volume less than 2cc, tympanometric peak pressure within +/- 100 da pa and static compliance less than 1.6cc.

Acoustic reflexometric findings of bilateral or unilateral presence of Ipsilateral and contralateral acoustic reflexes for pure tone frequencies 500Hz, 1 kHz, and for broad band noise.

iii) Speech identification scores: Speech identification scores more than 55% in the test ear.

3.2 Test environment

All tests were carried out in an acoustically treated audiometric room (ANSI S3.1 1991).

3.3 Instrumentation

Audiometer

A calibrated double channel audiometer (GSI- 61 version) with TDH – 39 housed in MX – 41/ AR ear cushions was used to measure air conduction thresholds and speech identification scores of the selected participants. The bone conduction thresholds were obtained using Radio ear B -71 bone vibrator.

Immittance meter

A calibrated Immittance meter, GSI-TS version-2 was used for the measurement of middle ear functioning.

Hearing aid

A four channel digital behind the ear (BTE) hearing aid.

Personal computer and Hi – pro

NOAH 3 and hearing aid specific software installed on a personal computer was used to program the hearing aid

.

Hearing aid analyzer and real ear measurement system

Fonix FP 7000 was used for electro acoustic measurement of the hearing aid in order to confirm whether the hearing aid is working according to manufacturer's specification and also to conduct real ear measurements to verify the gain of the hearing aid at ear canal level. Electoacoustic measurement of the selected hearing aid was done in such a way that the hearing aid was connected to the 2cc coupler of the hearing aid analyzer by using BTE adapter. The substitution method was used. The sound chamber was leveled and hearing aid was placed for testing in the test chamber. That is, the hearing aid was connected to a 2cc coupler and the output was collected through a test microphone for analysis. The amount of gain provided by the hearing aid in 2 cc coupler for an input of 65 dB SPL across 250Hz to 6500Hz was verified.

Evoked potential system

A two channel Bio-logic Navigator Pro version-7.0.0 was used for the recording of aided and unaided ACC. The uploaded stimulus in the Bio-logic Navigator Pro software for recording aided and unaided ACC was directed to the free field loud speaker calibrated at 45^{o} azimuth at a distance of one meter from the participant in the test room. The trigger was activated synchronously with the stimulus onset to record ACC.

Loudspeaker and Trigger

A calibrated dB Technologies 160 free field speaker, with a frequency response range of 50 Hz to 20000 Hz and maximum sound pressure level of 99dB SPL was used to present the /si/ stimulus for recording ACC in aided and unaided condition. Bio-logic Natus trigger was connected to Bio-logic Navigator Pro evoked potential system to get the synchronous presentation of stimulus to record ACC.

3.4 Stimulus generation

Aided and unaided ACC was recorded for naturally produced speech stimuli /si/. The stimulus was recorded from three different adult male individual with normal hearing sensitivity, recorded at sampling rate of 44000/sec. The overall duration of the three stimuli were kept equal (250.8ms) using adobe audition version 1.5 software. After recording of the three stimuli, goodness of fit test was run for all three stimuli in ten individual with normal hearing sensitivity. The most preferred /si/ stimulus was considered for acquiring ACC. The rationale behind to select /si/ as a stimulus to elicit ACC was that in /si/ (CV syllable); the initial phoneme (s) is high in frequency and low in intensity. This may be difficult for an individual with sloping cochlear hearing loss to perceive. The acoustic features of stimulus /si/ and the wave form of it is shown in Table 3.2 and Fig 3.1 respectively.

Table 3.2: acoustic features of stimulus /si/

Acoustic features	***Values***
Overall duration	250.8 msec
Fricative duration	127 msec
Frequency characteristics of fricative (Max frequency)	4445.61 Hz

Fricative to vowel transition	
F1	24msec
F2	18 msec
F3	19 msec
Vowel duration	123.8
Frequency characteristics of vowel (Hz)	
F1	322.2
F2	2594.01
F3	2978.24

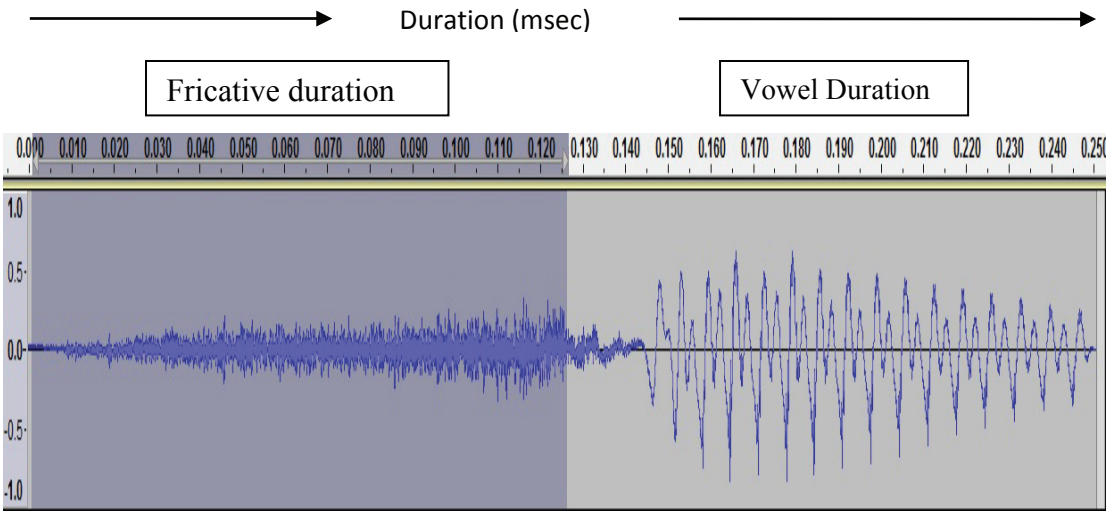


Fig 3.1: Wave form of stimulus /si/.

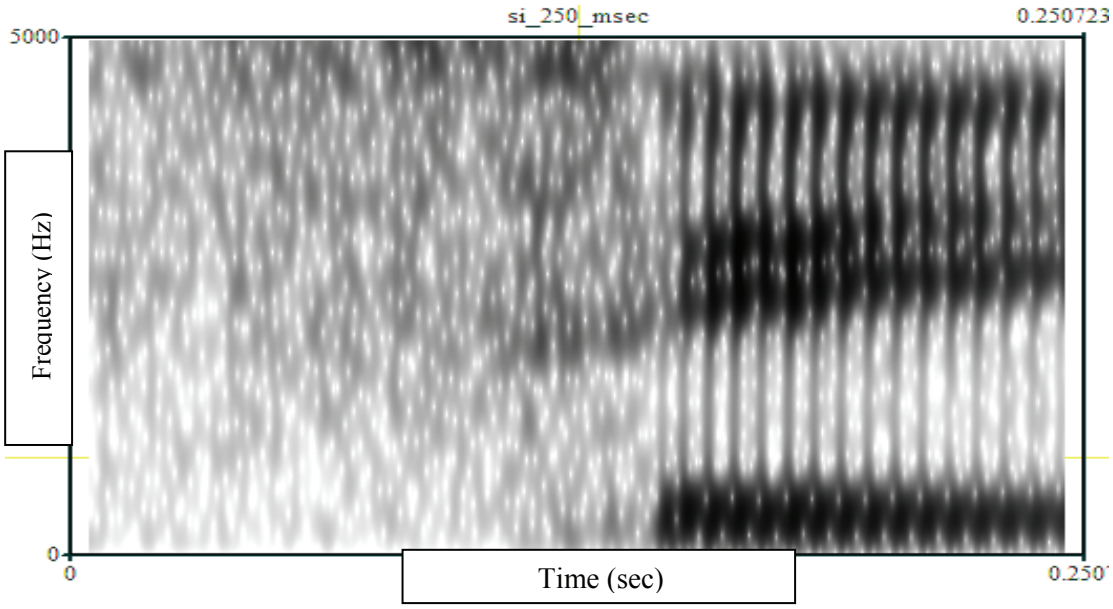


Fig 3.2: Spectrogram of stimulus /si/.

3.5 Procedure

Procedure involved four phases:

1) Selection of participants
2) Selection of hearing aid
3) Hearing aid fitting
4) Acquiring aided and unaided Acoustic change complex

Phase 1: Selection of participants

The following audiological tests were carried out for participant selection in to group I and group II.

i) Case history: A detailed case history was taken for each participant to make sure that the participants have no symptoms of retro-cochlear pathology.
ii) Pure tone Audiometry: Modified Hugston and Westlake method by Carhart and Jerger (1959), was used to measure the air conduction thresholds for octave frequencies from 250 Hz to 8 kHz and bone conduction thresholds for octave frequencies from 250 Hz 8 KHz. PTA_1 (average of the pure tone thresholds at 500Hz, 1 KHz, 2 KHz) and PTA_2 (average of the pure tone thresholds at 1 KHz, 2 KHz, 4 KHz) were calculated to define the degree and configuration of hearing loss for group I and group II.
iii) Speech audiometry: Speech identification score was obtained using the speech identification test material given by Yathiraj and Vijayalakshmi (2005), at a level of 40 dB SL (Re: SRT). 25 words were presented in the live mode (male speaker) at a level of 40 dBHL above PTA1. Scoring was done in such a way that each correct responses (repeating back correctly) got a score of 4%.

iv) Immittance evaluation: Tympanometry was done using probe frequency of 226 Hz (Brooks, 1968; Holte, Margolis & Cavanaugh, 1991) at 85 dB SPL. Ipsilateral and contralateral reflexes were obtained from test ear at 500 Hz, 1 KHz, and 2 KHz and for broad band noise. The audiological profile of each participant and mean threshold for group at each frequency is given in the Table 3.2 & 3.4 below.

Table 3.3: Audiological profile of each participant selected for the study

Sl. No.	***Duration of hearing loss (Years)***	***PTA_1 (dBHL)***		***PTA_2(dBHL)***		***SIS (%)***	
		R	***L***	***R***	***L***	***R***	***L***
1.	0.75	38.3	71.6*	51.60	73.66	96	58*
2.	1.00	10.0	20.0	15.00	31.66	96	100
3.	0.60	38.3	11.6	61.60*	13.33	84*	100
4.	0.60	35.0	33.3	51.60	58.66	72	80
5.	1.90	61.6	63.6	71.60	73.33	92	92
6.	1.10	46.6	48.3	56.60	56.66	84	80
7.	1.50	60.0	50.0	75.00	65.00	76	80
8.	1.00	50.0*	61.6*	11.60	8.333	80*	100
9.	2.00#	33.3	30.0	50.00	41.60	92	92
10.	4.00	25.0	20.0	41.30	35.00	72	76
11.	25.0	98.3	61.6	108.6	83.33	12*	56
12.	2.50	65.0	56.6	76.66	63.33	64	72
13.	4.00	30.0	70.0*	58.33	80.00*	76	48*
14.	5.00	28.3	95.0*	41.33	98.66*	96	24*
15.	11.00	18.3	51.6	30.00	66.66*	92	88*
16.	10.0	63.3	63.3	73.33	73.33	88	92
17.	7.0	46.6	48.3	56.66	56.66	84	80
18.	3.50	23.3	25.0	46.66	45.00	100	100
19.	12.0	91.6*	63.3	105	71.66	52*	72

Note: PTA_1 = Pure tone average of 500Hz, 1000Hz, and 2 KHz. PTA_2 = Pure tone average of 1 KHz, 2 KHz and 4 KHz. R = Right ear, L = Left ear. SIS = Speech identification score, * indicates masked thresholds/speech identification score.
# indicates that subject is included in group 2 (duration of loss >2 yrs).

Table 3.4 Mean thresholds at each audiometric frequency for each group

Degree of hearing loss	***Duration of hearing loss (years)***	***Mean threshold (dBHL)***					
		250 Hz	***500Hz***	***1 kHz***	***2 kHz***	***4 kHz***	***8 kHz***
Minimal to moderate	*< 2*	18.33	22.55	37.55	48.33	60.00	64.16
	> 2	18.00	22.00	35.00	45.00	68.00	89.00
Moderate to severe	*< 2*	25.00	42.55	52.55	70.00	75.00	73.75
	>2	42.88	50.77	60.00	70.00	80.71	91.42

Phase 2 - Selection of hearing aid

Four channel digital behind the ear hearing aid was selected to record aided ACC. The fitting range of the hearing aid covered severe to profound hearing loss. According to the manufacturer's specification, in 2cc coupler the frequency response of the hearing aid extended from 100 Hz to 6200Hz. Peak full on gain was 70dB and high frequency average full on gain was 62dB. Attack time was 10ms and release time was 51ms. The total harmonic distortion was less than 3% at 500 Hz, 2% at 800 Hz and 1% at 1600 Hz, whereas, the equivalent input noise was 18 dBSPL. The measured electroacoustic characteristics of the hearing aid were according to manufacturer's specification. The measured electroacoustic characteristics of hearing aid are shown in Table 3.3 below.

Table 3.5: Shows the electroacoustic characteristics and measured value of the hearing aid used for recording aided ACC

***Electroacoustic* characteristics**	***Measured value***
OSPL 90	132.7
Peak full on gain (dB)	69.3
Frequency response (Hz)	200 to 5830
High frequency full on gain (dB)	60.3
Equivalent input noise (dB)	15.3

Total harmonic distortion (%) at 500Hz	2.8
800Hz	1.0
1600Hz	1
Hearing aid delay (ms)	3.3
Attack time and release time (ms)	9 & 51

Phase 3 - Hearing aid fitting

After the selection of the hearing aid hearing aid fitting was done to the participants test ear. Regular ear mold that is attached to the BTE adapter was used to ensure that the squealing was absent and that is snugly fit in to the participants test ear without creating discomfort. The following two approaches were made prior recording of ACC - i) programming of hearing aid ii) Real ear measurement of hearing aids.

Programming of hearing aid

The selected hearing aid was connected to the personal computer and Noah fitting software by using HiPro and the hearing instrument interface for programming. The Bass Boost facility of the hearing aid was turned off, and the hearing thresholds were fed on to Noah fitting software and fitting module. The hearing aid was set in omni-directional mode with enabled compression circuits, where compression ratio in the NAL-NL1 default setting was used and volume control was disabled. NAL-NL1 fitting formula was used to prescribe the gain for hearing aid and hearing aid was programmed to match the target gain at acclimatization level two.

Real ear measurement

The participants were seated in front of the free field speaker of phoenix FP 7000 equipment. The speaker of equipment was placed at 45 degree azimuth and 1 meter distance from the participant. Then the sound field was equalized.

The audiogram of the test ear was plotted and fitting formula of NAL NL1, stimulus of digi-speech at 65 dBSPL was selected in the phoenix FP 7000 software. The probe tube of the microphone was inserted by means of premeasured length, 25 to 30 mm past tragal notch of the test ear. Then, the hearing aid was fitted to the test ear using regular ear mold attached with a with a BTE adapter. The real gain was verified to match the NAL-NL1 target. The gain of the hearing aid was increased to match the target whenever the real ear SPL was not matching the target gain curve.

Phase 4 - *Acquisition of aided and unaided Acoustic change complex*

Once the gain of the hearing aid in the ear canal is verified through real ear measurement, ACC was acquired in aided and unaided condition in each participant. The participants were seated comfortably in an armed chair and the fitted hearing aid was switched to 'on' position. The electrode site was cleaned with skin preparation gel. Disc type gold coated electrodes was placed with the help of conduction gel at the test ear mastoid (M1/M2), upper forehead (Fpz), and vertex (Cz). The impedance was less than 5 KΩ and inter-electrode impedance within 2 KΩ. Caution was taken about the chance of closer proximity or contact of electrode and hearing aid microphone. The hearing aid was positioned to the periphery of the pinna and made sure that the hearing aid is not in closer proximity or contact of electrode and hearing aid microphone. For recording the ACC in aided conditions, the stimulus /si/ was presented through free field speaker, positioned at one meter distance at 45 ^{o} azimuth.

The stimulus was presented at level of 65dB SPL and ACC was recorded for checking replicability of waveform. The non-test ear was blocked with ear mold impression material to avoid its participation (whenever required). The subjects were asked ignore the stimulus and watch a close captioned video while recording ACC.

After the acquisition of aided ACC the hearing aid was removed from participant's ear and unaided ACC was recorded. The non-test ear remained blocked with ear mold impression material to avoid its participation (whenever required). Below represented is the set up used for recording aided and unaided ACC and stimulus recording parameters used for recording, it is represented in Fig 3.2 and Table 3.6 respectively.

Set up used for recording unaided ACC	Set up used for recording aided ACC

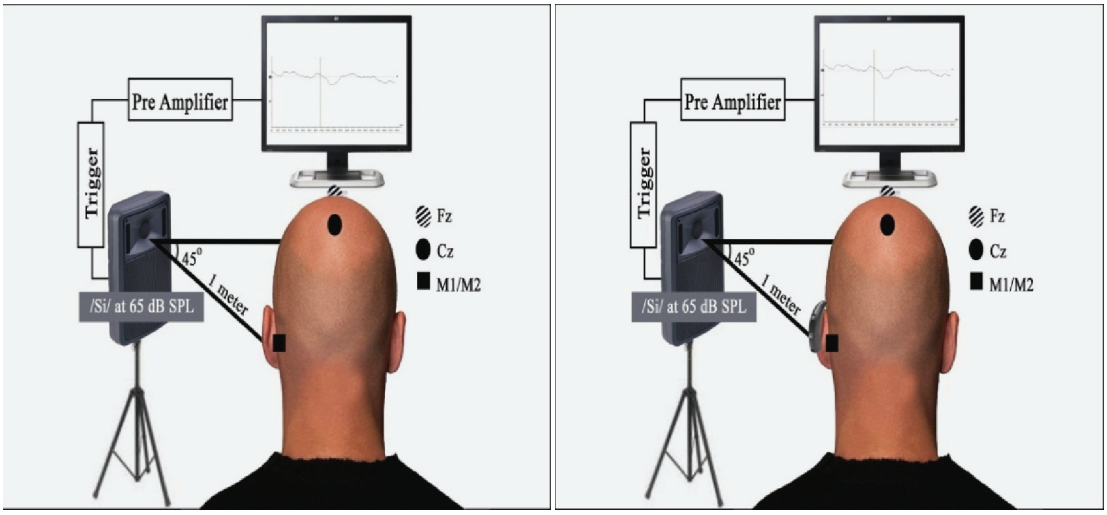


Figure 3.2: Shows the Set up used for recording aided and unaided ACC in participants with sloping SNHL.

Table 3.6: Shows the Stimulus parameters and Acquisition parameters for recording ACC

Stimulus parameters		***Acquisition parameters***	
Stimuli	/Si/	Mode of stimulation	Ipsi
Duration of stimuli	250.8	Electrode montage	Cz(+ve),

			M1/M2(-ve) of test ear and ground at Fz
Intensity	65dBSPL	Filter setting	0.1-30 Hz.
Polarity	Alternating	Analysis window	535 msec.
Transducer	Loudspeaker	No. of channels	Single
Mode of presentation	Free Field	Amplification	25,000
		Repetition rate	1.1 per sec
		Number of sweeps	150
		No. of repetitions	2

3.6 Analysis of waveforms

Analyses of unaided and aided ACC waveforms were done for all participants. The peak identification and morphology rating were done by two experienced audiologist in wave form analysis. The peaks which are marked by both audiologists were considered for analysis. When an audiologist was marked a peak, but, not the other, the peak was not considered for analysis. The peaks of ACC were marked as P1, N1, P2, N2, $P1^{1}$, $N1^{1}$, $P2^{1}$, and $N2^{1}$. Amplitude, latency, and morphology were the three measures considered for waveform analysis. The amplitude of the each peak was defined as the largest positive or negative deflection depending on whether it's a negative or positive peak in the response window. The latencies of the peaks were calculated by taking the center or midpoint when wave form contained double peak of equal amplitude. Latency was measure at the center of larger peak when peaks were not equal in amplitude (Ostroff, Martin & Boothroyd, 1998). The data point values representing N1, P2, $N1^{1}$ and $P2^{1}$ were tabulated for statistical analysis.

Chapter 4

RESULTS AND DISCUSSION

The aim of the present study was to investigate the effect of degree and duration of sloping sensorineural hearing loss and digitally amplified speech on Acoustic Change Complex. The specific objectives were to compare the unaided & aided ACC separately (1) between minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss, (2) between less than or greater than two years of duration of hearing loss in minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss separately and finally, (3) the last objective was to do a comparison of the unaided and aided ACC within each subgroup. To do this, three independent variables; subgroup, unaided, and aided condition were taken and their influence on the dependent variables; parameters of ACC (latency and amplitude) was studied. N1, P2, $N1^1$, $P2^1$ were the target parameters considered for statistical analysis. The statistical analysis was carried out using Statistical Package for Social sciences (SPSS) (version 16).

The following statistical tests were carried out in the present study.

i. Descriptive statistics (mean and standard deviation) were calculated for each subgroup; minimal to moderate sloping sensorineural hearing loss (SNHL) with duration of hearing loss less than two years (subgroup A), minimal to moderate sloping SNHL with duration of hearing loss greater than two years (subgroup B), moderate to severe sloping SNHL with duration of hearing loss less than two years (subgroup C), moderate to severe sloping SNHL with duration of hearing loss greater than two years (subgroup D).

ii. Mann Whitney test was administered to compare the unaided & aided ACC separately, between minimal to moderate & moderate to severe degrees of sloping sensorineural hearing loss, with duration of hearing loss less than and greater than two years.

iii. Mann Whitney test was administered to compare between less than or greater than two years of duration of hearing loss in minimal to moderate & moderate to severe degree of sloping sensorineural hearing loss separately.

iv. Wilcoxon signed rank test was done for comparing unaided and aided ACC within each subgroup.

Results of the present study are discussed under the following headings:

i. *Results of the descriptive statistics for each subgroup (A, B, C, and D)*
ii. *Effect of different degree of sloping SNHL on unaided and aided ACC.*
iii. *Effect of duration of hearing loss along with degree on unaided and aided ACC.*
iv. *Comparison of aided and unaided ACC within each subgroup*.

4.1 Results of the descriptive statistics for each subgroup (A, B, C, and D)

Results of the descriptive statistics for each subgroup (A, B, C, and D) are given in table 4.1, 4.2, 4.3, and 4.4 respectively. The effect of different degree and duration of hearing loss on latency and amplitude of peaks of ACC in unaided and aided condition is shown in graph 4.1 and 4.2 respectively. The grand mean waveform of each subgroup is shown in fig 4.1, 4.2, 4.3 and 4.4.

Table 4.1: Mean and standard deviation of ACC peak latency & amplitude in subgroup A (minimal to moderate sloping SNHL with duration of less than two years)

Peaks	***N***	***Mean***	***Std. Deviation***
LaencytN1(unaided)	2	126.61	9.744
Latency of N1(aided)	5	128.77	14.174
Latency of P2(unaided)	3	174.01	15.002
Latency of P2(aided)	6	184.30	14.213
Latency of $N1^{I}$ (unaided)	6	261.93	16.512
Latency of $N1^{I}$(aided)	6	255.04	7.960
Latency $P2^{I}$ (unaided)	6	321.93	10.42
Latency of $P2^{I}$ (aided)	6	321.40	10.92
Amplitude of N1(unaided)	2	-0.8600	1.103
Amplitude of N1(aided)	5	-0.3600	1.077
Amplitude of P2 (unaided)	3	0.4433	.5054
Amplitude of P2(aided)	6	1.0650	.8534
Amplitude of $N1^{I}$ (unaided)	6	-0.5450	.7416
Amplitude of $N1^{I}$ (aided)	6	-02.918	1.600
Amplitude of $P2^{I}$ (unaided)	6	1.0920	1.368
Amplitude of $P2^{I}$ (aided)	6	1.5630	.6954

Table 4.2: Mean and standard deviation of ACC peak latency and amplitude in subgroup B (minimal to moderate sloping SNHL with duration of greater than two years)

Peaks	***N***	***Mean***	***Std. Deviation***
LaencytN1(unaided)	0		
Latency of N1(aided)	3	136.03	10.83
Latency of P2(unaided)	2	203.00	4.412
Latency of P2(aided)	4	203.26	13.22
Latency of $N1^{I}$ (unaided)	3	260.94	6.692
Latency of $N1^{I}$(aided)	4	262.07	8.384
Latency $P2^{I}$ (unaided)	3	320.17	8.850
Latency of $P2^{I}$ (aided)	4	327.91	7.746

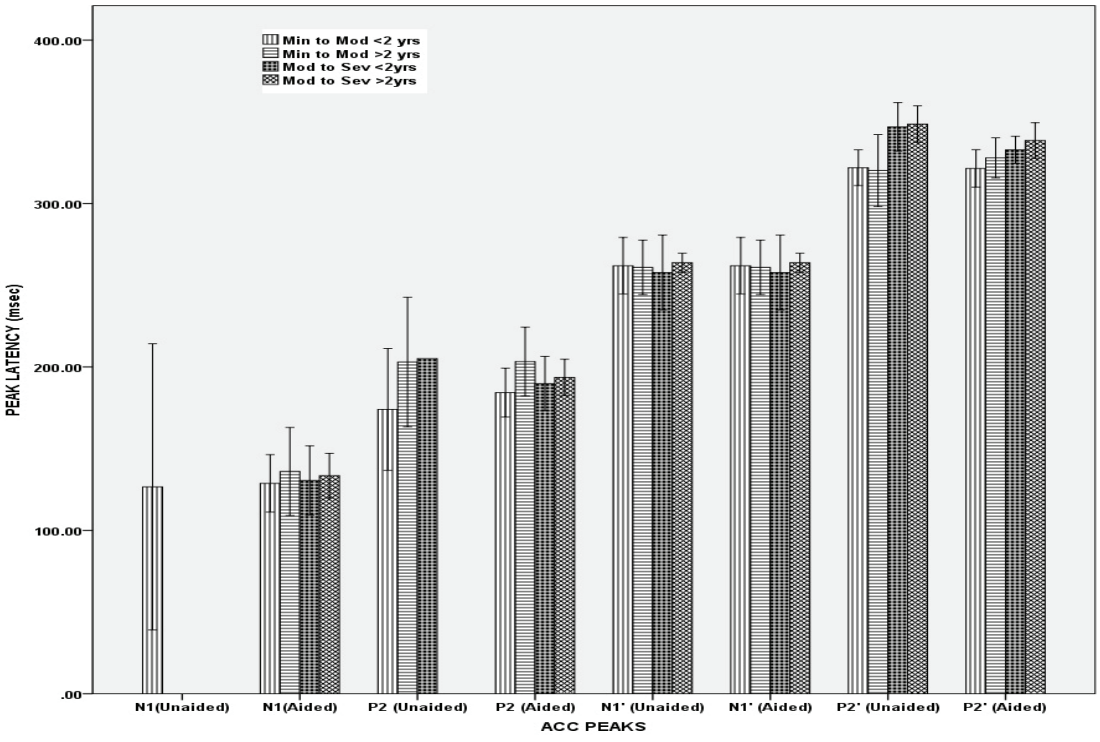
Amplitude of N1(unaided)	0		
Amplitude of N1(aided)	3	-0.040	1.133
Amplitude of P2 (unaided)	2	2.6700	.3111
Amplitude of P2(aided)	4	2.4820	1.255
Amplitude of $N1^1$ (unaided)	3	-03.90	1.624
Amplitude of $N1^1$(aided)	4	-02.94	1.133
Amplitude of $P2^1$ (unaided)	3	3.040	.5894
Amplitude of $P2^1$ (aided)	4	1.830	1.368

Table 4.3: Mean and standard deviation of ACC peaks in subgroup C (moderate to severe sloping SNHL with duration of less than two years)

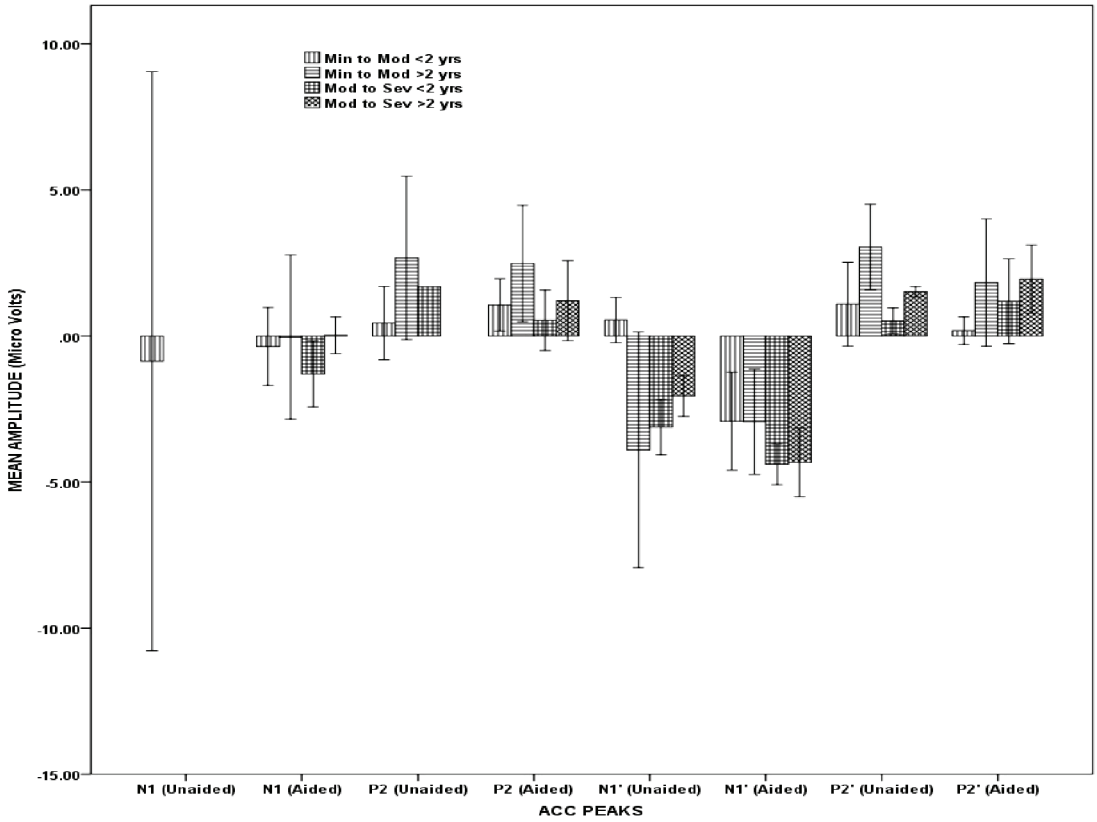
Peaks	***N***	***Mean***	***Std. Deviation***
LaencytN1(unaided)	0		
Latency of N1(aided)	4	130.55	13.328
Latency of P2(unaided)	0	.	.
Latency of P2(aided)	3	189.81	6.691
Latency of $N1^1$ (unaided)	4	257.73	14.402
Latency of $N1^1$(aided)	4	253.35	9.802
Latency $P2^1$ (unaided)	4	346.92	9.325
Latency of $P2^1$ (aided)	4	332.86	5.196
Amplitude of N1(unaided)	0		
Amplitude of N1(aided)	4	-1.297	.7083
Amplitude of P2 (unaided)	1	1.6900	.
Amplitude of P2(aided)	3	.5367	.4148
Amplitude of $N1^1$ (unaided)	4	-3.117	.5990
Amplitude of $N1^1$(aided)	4	-4.392	.44131
Amplitude of $P2^1$ (unaided)	4	.5175	.2777
Amplitude of $P2^1$ (aided)	4	1.192	.9152

Table 4.4: Mean and standard deviation of ACC peaks in subgroup C (moderate to severe sloping SNHL with duration of greater than two years)

Peaks	***N***	***Mean***	***Std. Deviation***
LaencytN1(unaided)	0		
Latency of N1(aided)	6	133.49	13.010
Latency of P2(unaided)	0		
Latency of P2(aided)	6	193.59	10.586
Latency of N1^{1} (unaided)	7	263.79	6.3100
Latency of N1^{1}(aided)	7	252.78	2.8260
Latency P2^{1} (unaided)	3	348.60	4.4857
Latency of P2^{1} (aided)	7	338.53	11.747
Amplitude of N1(unaided)	0		
Amplitude of N1(aided)	6	.02333	.59762
Amplitude of P2 (unaided)	0		
Amplitude of P2(aided)	6	1.2083	1.3014
Amplitude of N1^{1} (unaided)	7	-2.0543	.75934
Amplitude of N1^{1}(aided)	7	-4.3300	1.2689
Amplitude of P2^{1} (unaided)	3	1.5200	.07000
Amplitude of P2^{1} (aided)	7	1.9514	1.2585



Graph 4.1: The effect of different degree and duration of hearing loss on latency of each ACC peaks in unaided and aided condition.



Graph 4.2: The effect of different degree and duration of hearing loss on amplitude of each peak of ACC in unaided and aided condition.

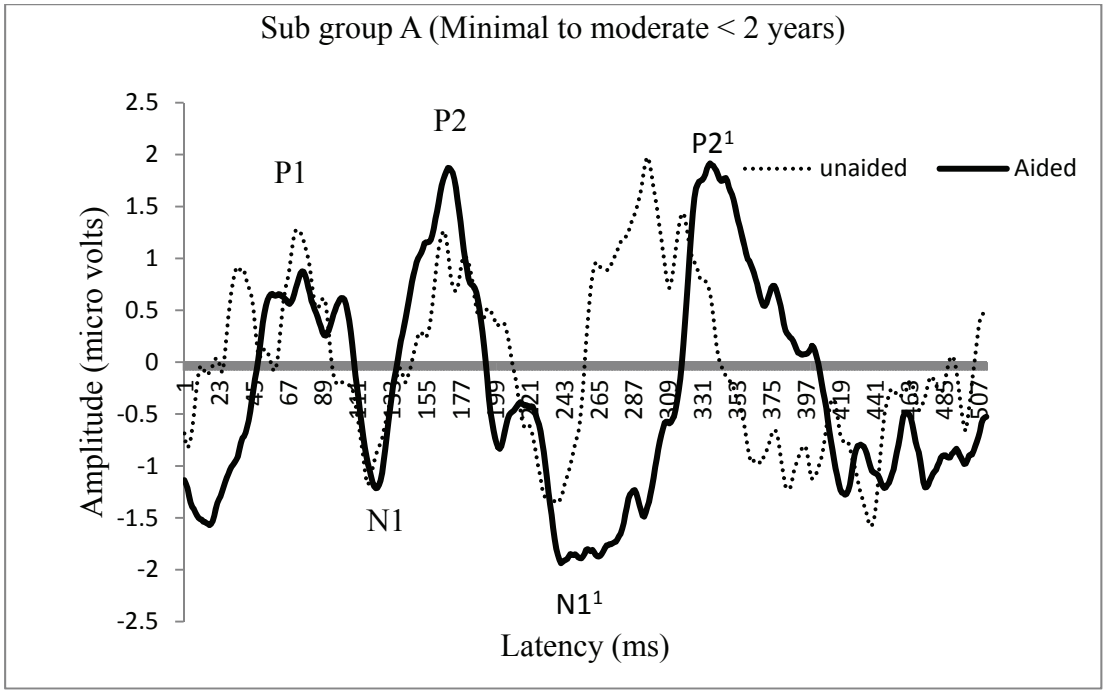


Fig 4.1.The Grand mean ACC waveform of subgroup A (minimal to moderate sloping SNHL with duration of hearing loss less than two years).

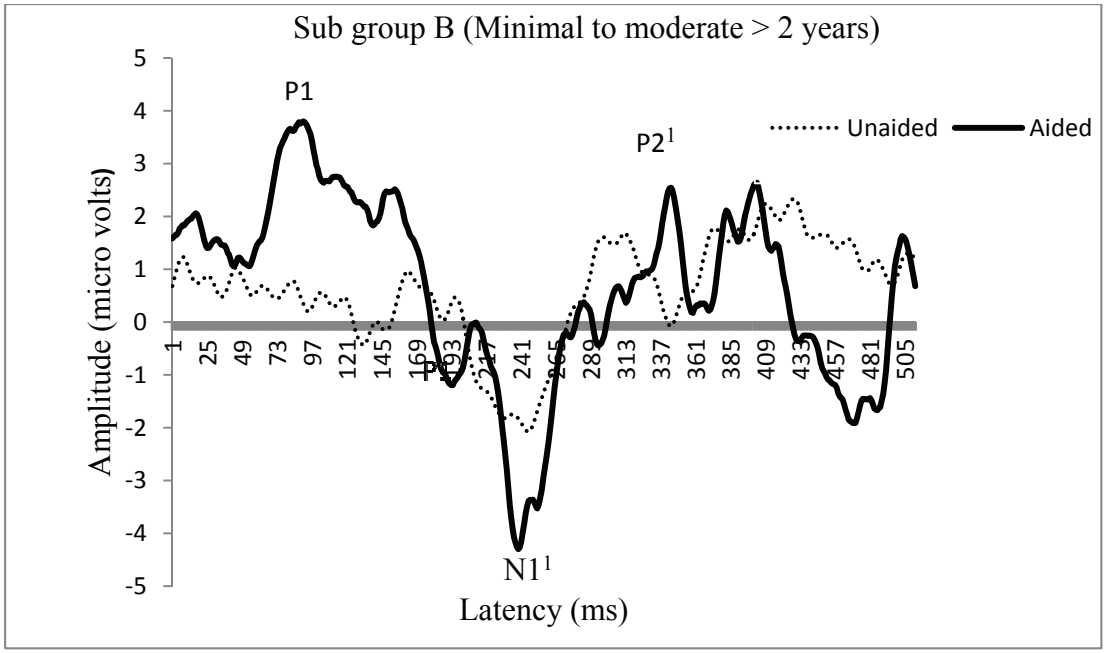


Fig 4.2.The Grand mean ACC waveform of subgroup B (minimal to moderate sloping SNHL with duration of hearing loss greater than two years).

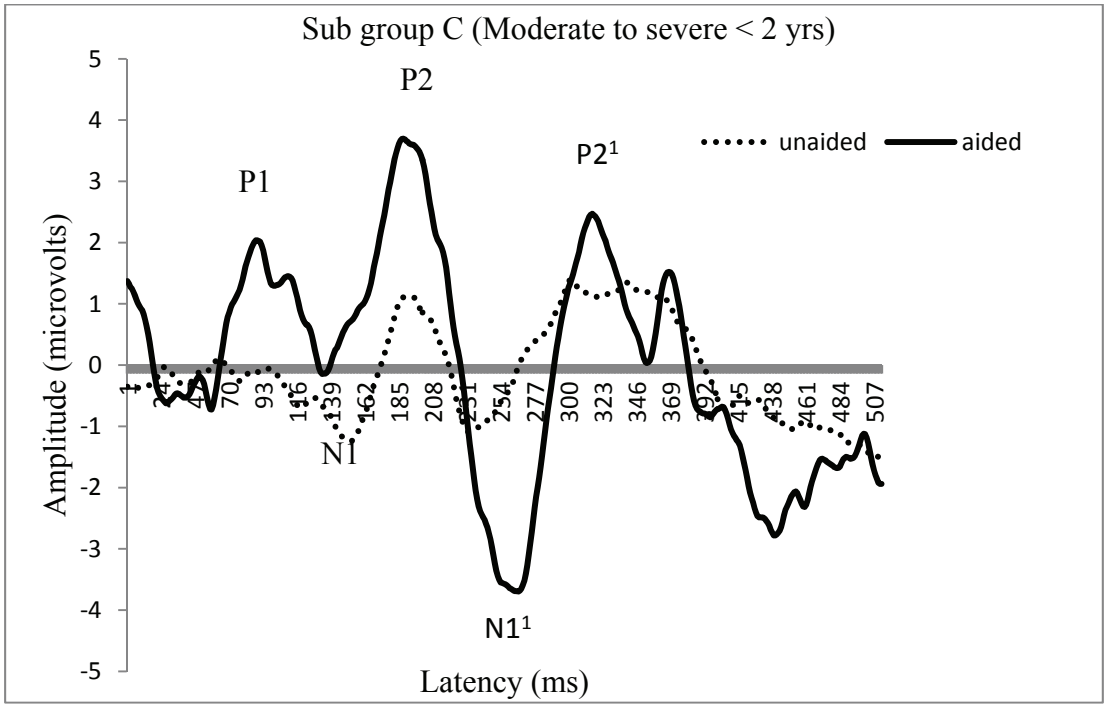


Fig 4.3.The Grand mean ACC waveform of subgroup C (moderate to severe sloping SNHL with duration of hearing loss less than two years).

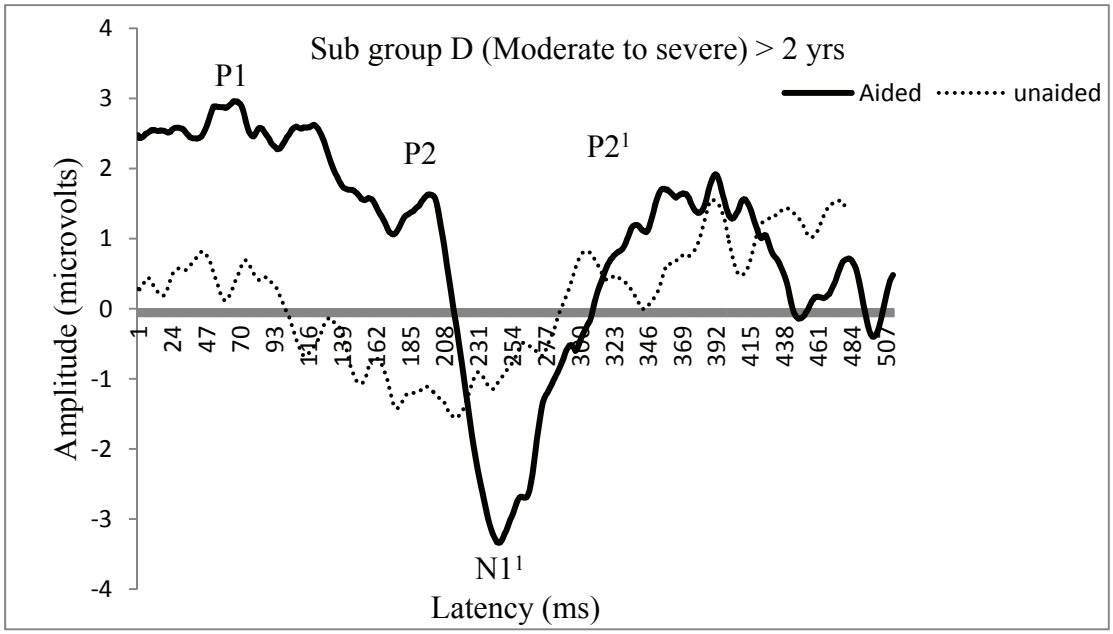


Fig 4.4: The Grand mean ACC waveform of subgroup D (moderate to severe sloping SNHL with duration of hearing loss greater than two years).

4.2 Effect of different degree of sloping SNHL on unaided and aided ACC.

To study the effect of different degree of sloping SNHL on unaided and aided ACC, Mann Whitney test was done; to do a comparison of group A vs. C (minimal to moderate sloping SNHL with duration of less than two years vs. moderate to severe sloping SNHL with duration of less than two years) and B vs. D (minimal to moderate sloping SNHL with duration of greater than two years vs. moderate to severe sloping SNHL with duration of greater than two years) in unaided and aided condition. The results on the effect of different degree of sloping SNHL on unaided and aided ACC are discussed under two headings:

i. Effect of minimal to moderate vs. moderate to severe sloping SNHL with duration of hearing loss less than two years on unaided and aided ACC.

ii. Effect of minimal to moderate vs. moderate to severe sloping SNHL with duration of hearing loss greater than two years on unaided and aided ACC.

4.2.1 Effect of minimal to moderate vs. moderate to severe sloping SNHL with duration of hearing loss less than two years on unaided and aided ACC

Mann Whitney test was administered to study the differential effect of different degree of sloping SNHL with duration of hearing loss less than two years on unaided and aided ACC. Latency and amplitude of N1[1], P2[1]were compared separately between subgroup 'A' and 'C' in the unaided condition. N1, P2 peaks were absent in subgroup C, whereas, in subgroup A, the N1and P2 peaks were present only for two and three ears out of six ears respectively. Hence, these ACC peaks were not included for comparison in the unaided condition. In the aided condition, N1 peak was present in all ears except one ear in the same subgroup. However, in subgroup C, it was present for all four ears. P2 peak was obtained for all ears in subgroup A. But, in subgroup C it was absent for one ear out of four ears aided condition. The reason for absence of N1 and P2 in subgroup C and its presence in subgroup A is discussed below

The N1 of ACC represents the onset of consonant portion in the stimulus (syllable) (Ostroff, Martin & Boothroyd, 1998). The consonant portion (fricative portion) in the stimulus (/si/) has a spectral energy of 2 K Hz and above, more energy around 4 to 8 K Hz (shown in fig 3.3). The audibility at the higher frequency is important for the perception of fricatives (Annabelle, Sher & Owens, 1972), and it is also evident from studies that if proper audibility is present there is indemnity of generation of far field recordable cortical potentials. In the present study, the two ears

in the subgroup A had mean threshold at high frequency (from 1 K Hz to 4KHz) as 41.6 and 43.3 dBHL. Hence, the presence of N1is accounted to the intensity level of the presentation of stimulus, which was at 65 dBSPL. But, in group C N1 was absent for all subjects. This suggests, audibility at the higher frequencies (1 K Hz and above) is important for the generation of N1.

4.2.1.1 Latency of $N1^1$, $P2^1$

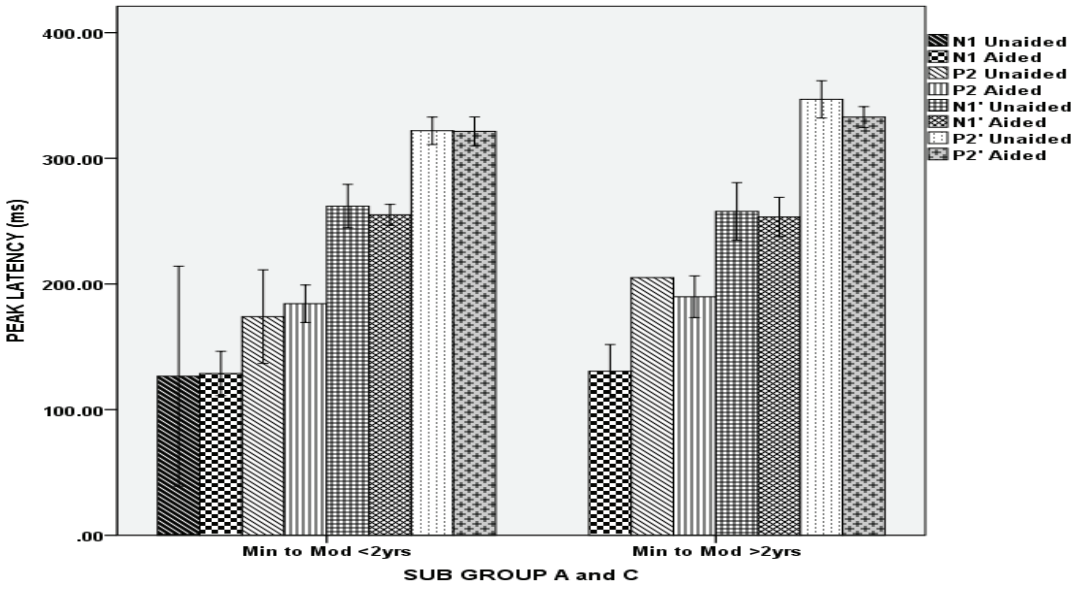
The results of the Mann Whitney test for latency of$N1^1$, $P2^1$ revealed that there is a significant difference in latency of $P2^1$ (Z = -2.07 p = .038) in unaided condition between subgroup A vs. C. But, aided condition did not show any statistical significance. The latency of $P2^1$ was shorter (mean latency of 321.93, shown in table 4.1)) for subgroup A compared to subgroup C (mean latency of 346.92ms, shown in table 4.3). There was no statistically significant difference in the $N1^1$ latency between subgroup A and C. The results of the Mann Whitney test for latency and amplitude for $N1^1$, $P2^1$ in unaided and aided condition are given Table 4.1.The effect of degree of sloping SNHL on unaided and aided ACC is depicted in graph 4.3 below.

Table 4.5: Z value and level of significance for latency and amplitude of N1, P2, $N1^1$, $P2^1$in unaided and aided condition

Peaks	***Z value***	***Level of significance***
latency of N1 (Aided)	-.447	.655
Latency of $N1^1$ (Unaided)	-.130	.897
Latency of $N1^1$(Aided)	-.516	.606
latency of $P2^1$(Unaided)	-2.07	.038*
latency of $P2^1$ (Aided)	-1.55	.121
Amplitude of N1(Aided)	-1.34	.180
Amplitude of $N1^1$(Unaided)	-2.32	.020*

Amplitude N1[1] (Aided)	-1.55	.121
Amplitude P2[1] (Unaided)	-.516	.606
Amplitude P2[1] (Aided)	-1.03	.300

Note: * indicates statistically significant.

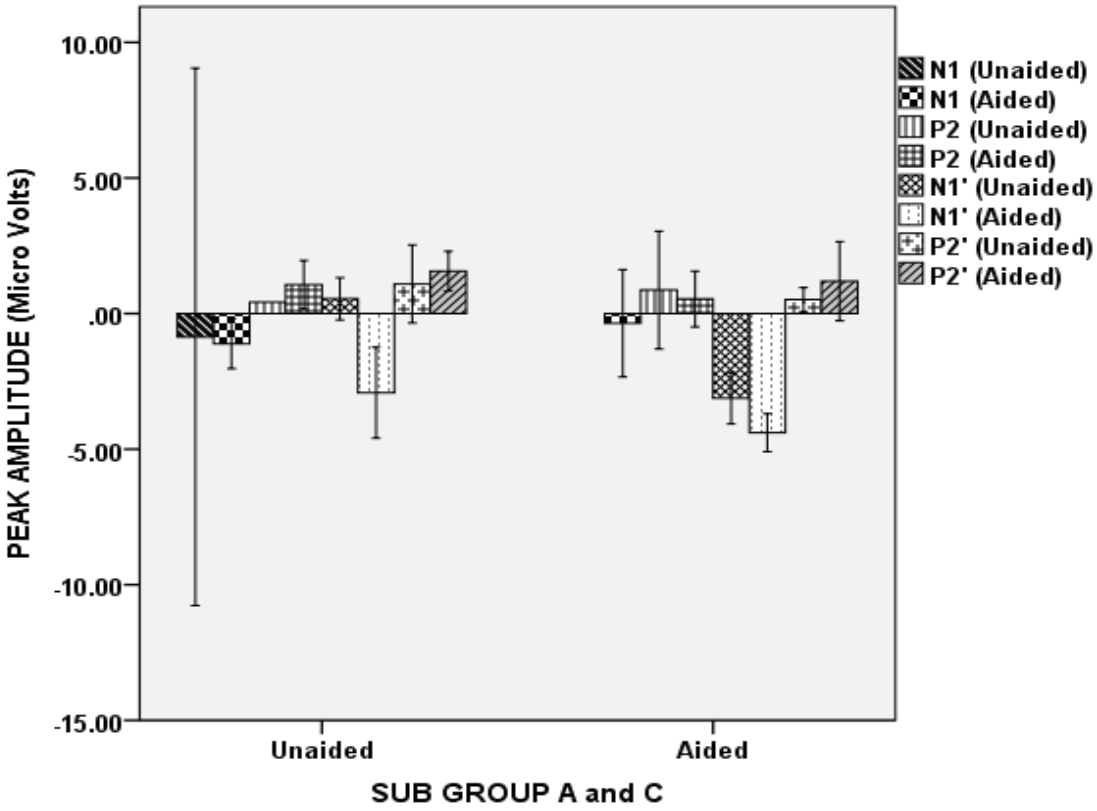


Graph 4.3: Effect of minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss less than two years on latency of unaided and aided ACC.

4.2.1.2 Amplitude of N1[1], P2[1]

Mann Whitney test revealed that there was no statistically significant difference in amplitude of P2[1] between subgroup A vs. C in both unaided and aided conditions. There was a statistically significant difference in amplitude of N1[1] ($p = .020$) in unaided condition between subgroup A vs. C, whereas, aided condition did not shown statistically significant differences. The amplitude of *N1[1]* was larger for subgroup A compare to subgroup C, individual with minimal to moderate sloping

SNHL showed larger amplitude compared to moderate to severe sloping SNHL. The results are represented in table 4.5 above and in graph 4.4 below.



Graph 4.4: Effect of minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss less than two years on amplitude of unaided and aided ACC.

To conclude, there is a significant difference in $P2^1$ latency and $N1^1$ amplitude in unaided condition between minimal to moderate and moderate to severe sloping SNHL with durations of hearing loss less than two years. The $P2^1$ is a response to the vowel portion of the stimulus i.e. syllables (Ostroff, Martin, Boothroyd, 1998) and thresholds at the 250Hz to 2 kHz is important for the generation of $P2^1$. In present study stimulus had a vowel (/i/) with more spectral energy in this region. Subgroup A had better thresholds at this frequency region compare to C as a result of this, the $P2^1$ latency was shorter for Subgroup A compare to C. Hence, it is possible to

electrophysiologicaly state that when the degrees of slope till 2 kHz increases, the vowel coding at the cortical level decreases. The onset portion of /i/ vowel has got low frequency content than its steady state portion and cortical response to this onset portion is represented by $N1^1$ peak of ACC. The low frequency thresholds (250 Hz and 500 kHz – mean of 20.44 dBHL) were better for subgroup A than C (mean threshold of 33.77). So, there is larger amplitude of $N1^1$ for minimal to moderate than moderate to severe sloping SNHL with duration of hearing loss less than two years. But, in aided condition, there was no significant difference between two groups. It says that the hearing aid in both degrees (higher and lower) works similarly.

4.2.2 *Effect of minimal to moderate vs. moderate to severe sloping SNHL with duration of hearing loss greater than two years on unaided and aided ACC.*

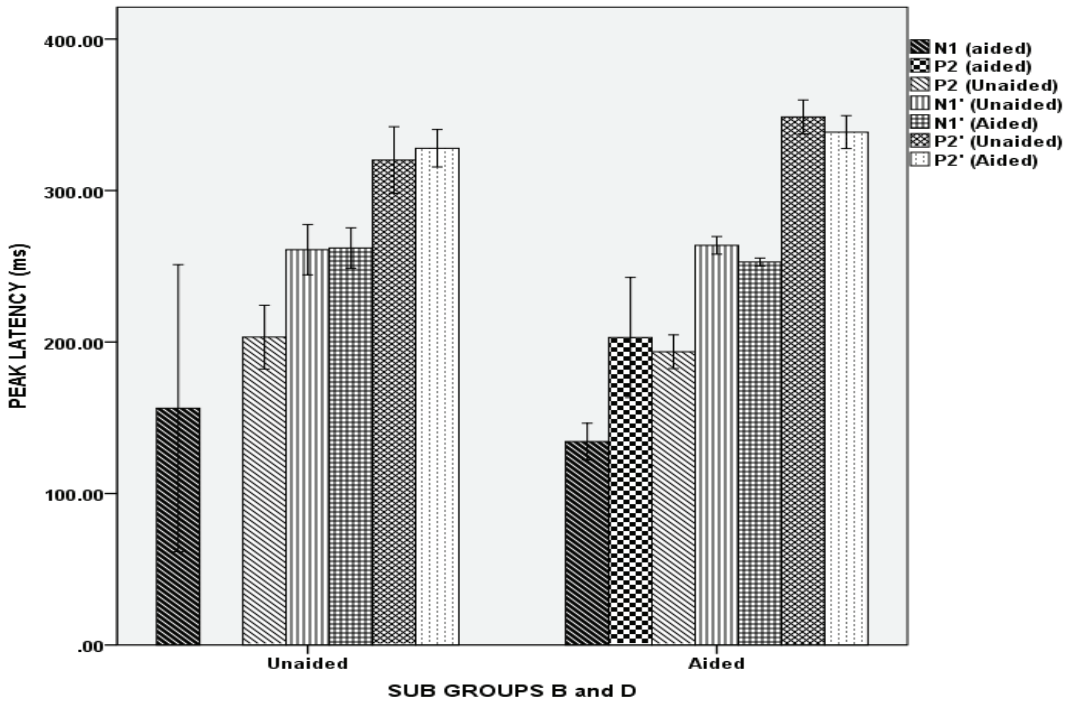
4.2.2.1 Latency of N1, P2, $N1^1$, $P2^1$

In unaided condition, N1 was absent for subgroup B and D, whereas, P2 was present only for two ear in the subgroup B with a mean latency of 203ms. Mann Whitney test revealed that there was no statistically significant difference in latency of N1, P2, between subgroup B vs. D in aided conditions. Latency of $N1^1$ ($p = .059$) in aided condition and $P2^1$inunaided condition ($p = 0.50$) between subgroup B vs. D is approaching statistical significance. The latency of $N1^1$ in aided condition was longer (mean latency of 262.07) for subgroup B compare to subgroup D (mean latency of 252.78), i.e., minimal to moderate sloping SNHL group showed longer latency compare to moderate to severe sloping SNHL. The results of Mann – Whitney test are represented in table 4.6 given below. The effect of minimal to moderate and moderate to severe SNHL on latency of unaided and aided ACC is depicted in graph 4.5 below.

Table 4.6: Z value and level of significance for latency and amplitude of N1, P2, $N1^1$, $P2^1$ in unaided and aided condition

Peak	***Z value***	***Level of significance***
latency of N1 (Aided)	-.258	.796
Latency of P2(aided)	-1.06	.286
Latency of $N1^1$(unaided)	-.686	.493
latency of $N1^1$(aided)	-1.89	.059#
latency of $P2^1$ (unaided)	-1.96	.050#
latency of $P2^1$(Aided)	-1.70	.089
Amplitude ofN1(aided)	.000	1.00
Amplitude of P2(aided)	-1.27	.201
Amplitude $N1^1$ (unaided)	-1.93	.053#
Amplitude $N1^1$ (aided)	-1.70	.089
Amplitude $P2^1$(unaided)	-1.96	.050#
Amplitude $P2^1$(aided)	.000	1.00

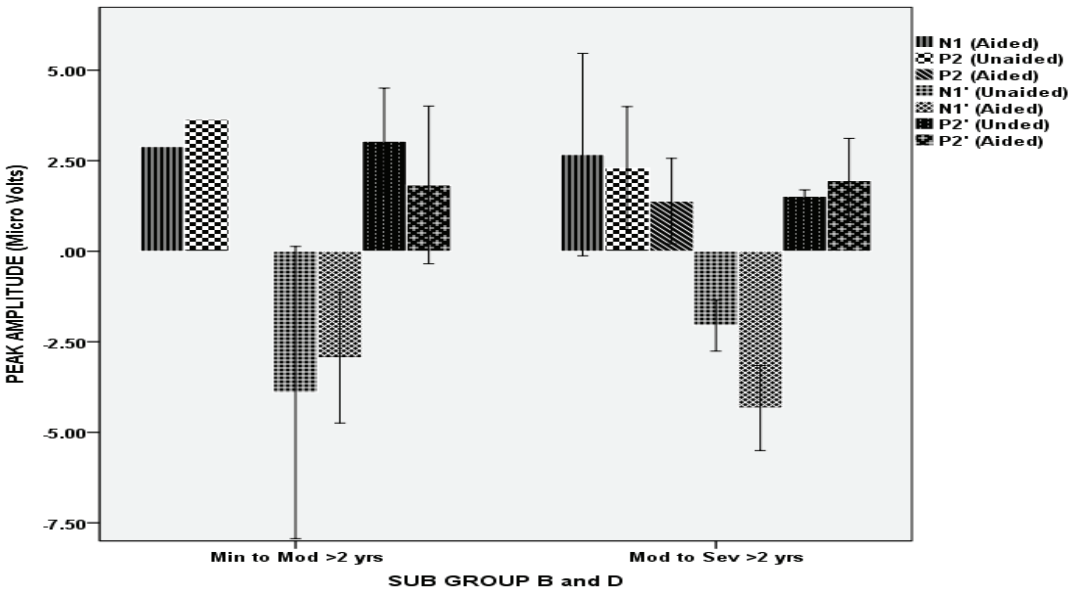
# indicates approaching significance.



Graph 4.5: Effect of minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss more than two years on latency of unaided and aided ACC.

4.2.2.2 Amplitude of N1, P2, N1[1], P2[1]

Mann Whitney test revealed that there was no statistically significant difference in amplitude of N1, P2, between subgroup B vs. D in aided conditions. In unaided condition, N1 was absent for subgroup B and D, and P2 was present only for two ear in the subgroup B with a mean amplitude of 2.67uv. Amplitude of $N1^1$ (p=0.053) and $P2^1$ (p=0.050) in unaided condition between subgroup B vs. D is approaching statistical significance. The amplitude of $N1^1$ in unaided condition was larger (mean amplitude of -3.9uv) for subgroup B compared to subgroup D (mean amplitude of -2.05uv), i.e., in individual with minimal to moderate sloping SNHL showed larger amplitude compare to moderate to severe sloping SNHL. The results of Mann Whitney test are represented in table 4.6 given above. Effect of minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss more than two years on amplitude of unaided and aided ACC is represented in graph 4.6 below.



Graph 4.6: Effect of minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss more than two years on amplitude of unaided and aided ACC.

The comparison of greater duration (greater than two years) with two different degree of sloping SNHL demonstrated statistically different amplitude of $N1^1$ and $P2^1$latency and amplitude in unaided condition. When, effect of greater duration of hearing loss added to degree of slope, $N1^1$ showed significant difference between minimal to moderate and moderate to severe sloping SNHL in unaided and aided condition. The auditory deprivation effect could be the probable reason for this. It indicates that if the duration of hearing loss is more, even with effective amplification also there is difference in cortical representation of speech sounds. The concept of auditory deprivation in individuals with moderate to severe SNHL as a measure of cortical potential was given by Buckley, 2003. The result of the present study is in agreement with Buckley's study in terms changes in amplitude of the cortical potentials with auditory deprivation.

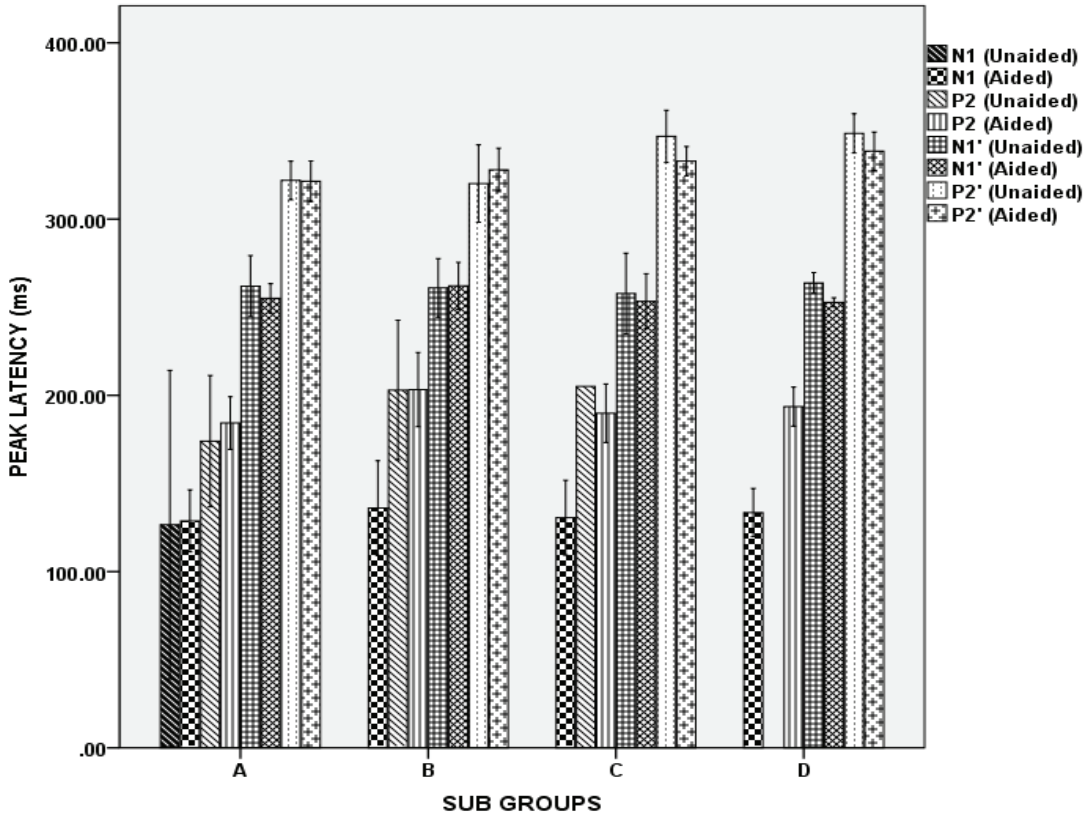
4.3 Effect of duration of hearing loss along with degree on unaided and aided ACC.

Mann Whitney test was administered to study the effect of duration of hearing loss on a particular degree of sloping hearing loss. The results of effect of duration of hearing loss on degree of sloping SNHL in unaided and aided ACC conditions are discussed under two headings.

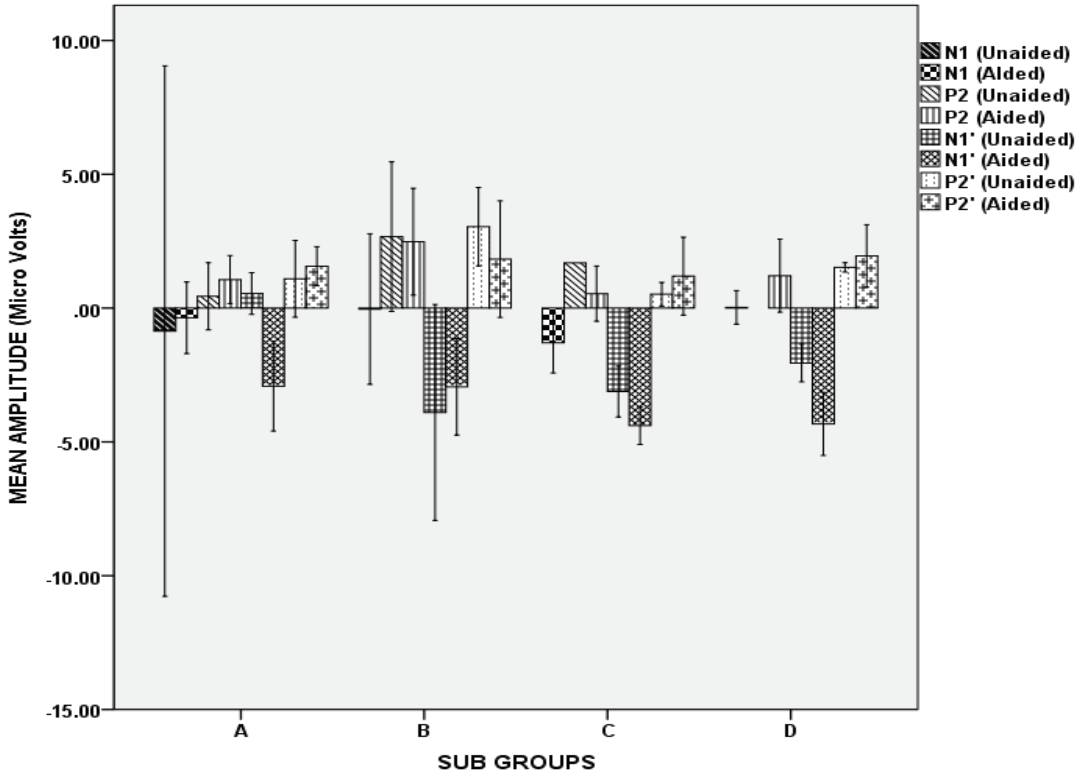
i. Effect of duration of hearing loss on minimal to moderate sloping SNHL
ii. Effect of duration of hearing loss on moderate to severe sloping SNHL

4.3.1 Effect of duration of hearing loss on minimal to moderate sloping SNHL

Mann-Whitney test was administered to study the two different duration of hearing loss; less than two years and greater than two years on minimal to moderate sloping SNHL. Latency and amplitude of N1, P2, $N1^{1}$,$P2^{1}$in subgroup A is compared with the same of subgroup B to study the effect of duration of hearing loss on ears with minimal to moderate sloping SNHL. Effect of duration of hearing loss of minimal to moderate sloping hearing loss on unaided and aided ACC is depicted in graph 4.7.



Graph 4.7: Effect of duration of hearing loss (less than two years and more than two years) of minimal to moderate and moderate to severe sloping SNHL on latency of unaided and aided ACC.



Graph 4.8: Effect of duration of hearing loss (less than two years and more than two years) of minimal to moderate and moderate to severe sloping SNHL on amplitude of unaided and aided ACC.

4.3.1.1 Latency of N1, P2, N1[1], P2[1]

Mann – Whitney test revealed that there was no statistically significant difference in latencies of N1, P2, N1[1], *P2[1]* between subgroup A vs. B in unaided condition. The N1 is present only for two ears in subgroup A (mean latency of 126.61ms) out of six ears, whereas, it was absent for all ears in subgroup B. P2, is present in three out six ears (mean latency of 174.01)in subgroup A and two ears out of four ears (mean latency of 203ms) in the subgroup B. But, latency of P2in aided condition is approaching statistical significance (p = .052) between subgroup A vs. B. The latency of P2was shorter (mean latency of 184.30ms) for subgroup A compared

to subgroup B (mean latency of 203.26ms).The results are represented in table 4.7 given below.

Table 4.7: Z value and level of significance for latency &amplitude of N1, P2, $N1^1$ and $P2^1$in unaided and aided condition

Peaks	***Z value***	***Level of significance***
latency of N1 (Aided)	-.745	.456
Latency of P2(aided)	-1.94	.052#
Latency of $N1^1$(unaided)	-.775	.439
latency of $N1^1$(aided)	-1.18	.240
latency of $P2^1$ (unaided)	-.130	.896
latency of $P2^1$ (Aided)	-1.70	.088
Amplitude ofN1(aided)	-.745	.456
Amplitude of P2(aided)	-1.49	.134
Amplitude $N1^1$ (unaided)	-2.32	.020*
Amplitude $N1^1$ (aided)	-.426	.670
Amplitude $P2^1$(unaided)	-1.80	.071
Amplitude $P2^1$(aided)	-.214	.831

* Indicates statistically significant, # indicates approaching statistical significance.

4.3.1.2 Amplitude of N1, P2, $N1^1$, $P2^1$

Mann Whitney test revealed that there was no statistically significant difference in amplitude of N1, P2, between subgroup A vs. B in aided conditions, but these peaks were not considered in the unaided condition as N1 was absent for all ears in subgroup B and P2 was only present for three (mean amplitude of 0.44uv) and two ears (mean amplitude of 2.67uv) in subgroup A & B respectively. Amplitude of $N1^1$ in unaided condition between subgroup A vs. B is statistical significant (p = .020). The amplitude of $N1^1$ in unaided condition was larger for subgroup A compare to subgroup B. The mentioned results are represented in table 4.7 given above.

To conclude, minimal to moderate sloping SNHL with different durations are not affecting the latency of unaided ACC, but, the amplitude of $N1^1$. This effect was more seen in minimal to moderate sloping SNHL with duration of hearing loss greater than two years. The probable reason is auditory deprivation. In aided condition, P2 showed a difference, this tells us that with amplification there is difference in consonant coding at cortical level in individual with more than two years of duration of minimal to moderate sloping SNHL. So, it could be that the auditory deprivation within minimal to moderate sloping SNHL has an effect on cortical neural coding of acoustical changes within speech sounds with amplification.

4.3.2 Effect of duration of hearing loss on moderate to severe sloping SNHL

Latency and amplitude of N1, P2, $N1^1$,$P2^1$insubgroup C is compared with D to study the effect of duration of hearing loss on ears with moderate to severe sloping SNHL.N1 and P2 were absent in subgroup C & D in unaided condition, hence these peaks were not taken for comparison in unaided condition. But, N1, P2, $N1^1$and $P2^1$ peaks were considered for comparison in aided condition.

4.3.2.1 Latency of N1, P2, $N1^1$, $P2^1$

In unaided condition, Mann Whitney test revealed that there was no statistically significant difference in latencies of $N1^1$ and $P2^1$ between subgroup C and D, whereas, in aided condition latencies of N1, P2, $N1^1$,$P2^1$ also does not shown any statistically significant differences. The results are represented in Table 4.8 given below.

Table 4.8: Z value and level of significance for latency and amplitude of N1, P2, $N1^1$, $P2^1$in unaided and aided condition

Peaks	***Z value***	***Level of significance***
latency of N1 (Aided)	-.640	.522
Latency of P2(aided)	-.516	.606
Latency of $N1^1$(unaided)	-1.13	.257
latency of $N1^1$(aided)	-.378	.705
latency of $P2^1$ (unaided)	.000	1.00
latency of $P2^1$ (Aided)	-.570	.569
Amplitude ofN1(aided)	-2.34	.019*
Amplitude of P2(aided)	-.775	.439
Amplitude $N1^1$ (unaided)	-1.89	.059#
Amplitude $N1^1$ (aided)	.000	1.00
Amplitude $P2^1$(unaided)	-2.12	.034*
Amplitude $P2^1$(aided)	-1.32	.186

* Indicates statistically significant, # indicates approaching statistical significance.

4.3.2.2 Amplitude of N1, P2, $N1^1$, $P2^1$

Mann Whitney test revealed that there is a statistically significant difference in amplitudes of $P2^1$ (p = 0.034), between subgroup C vs. D. Whereas, $N1^1$ is approaching statistical significance (0.059) in unaided condition. In aided condition, only the amplitude of showed statistically significant difference (p = 0.019) between subgroup C &D. The results are represented in Table 4.8, given above.

The individual with moderate to severe sloping SNHL with duration of hearing loss less than two years or greater than two years had no N1 and P2 peaks of ACC. This indicates that consonant (fricative /s/) is not coded at their auditory cortical structures which are responsible for the generation of ACC potential at usual conversational speech level (65 dBSPL). The reason for this is poor thresholds at the

higher frequencies (frequencies above 2 KHz) leading to lack of 'audibility' of high frequency, low intensity speech sounds like fricatives. Lack of audibility at high frequencies could not be the only reason even though it is the major one. The other probable reason could be that the auditory deprivation may induce changes in the amplitude and latency of ACC, which may act together with decreased audibility at an input level of 65 dBHL. Hence, to support this, further research with various input levels to the hearing aids, needs to be conducted which will give information about auditory deprivation of ACC, as at higher levels the effect of audibility is compensated.

However, in these individuals there was a presence of $N1^1$ and $P2^1$ peaks, which indicate audibility to the vowel portion, can be preserved to some extent in these individuals with sloping SNHL. But, there is a question which need to be answered is, why the vowel coding was not significantly different (no significant difference in latency and amplitude $N1^1$ and $P2^1$) between duration of hearing loss of less than two years and more than two years. The probable reason for this is the heterogenic cortical auditory response characteristics in individual with moderate to severe sloping SNHL, and also the variability in the participant selection (number of ears selected in subgroup C had only four, which, D had only seven). However, a proper amplification is given to these participants; there was a presence of N1, P2 peaks and statistically significant difference in $N1^1$ amplitude of aided ACC between moderate to severe sloping SNHL with duration of hearing loss less than two years (more amplitude)and more than two years (less amplitude). The reason for presence of N1, P2 indicates that with proper amplification the consonant (/s/) is coded in the cortical level of individual with moderate to severe sloping SNHL irrespective of duration of hearing loss. But, the reason for significant difference in $N1^1$ amplitude

(indicates vowel onset portion (/i/) in the stimulus coded differently in moderate to severe sloping SNHL with duration of hearing loss less than two years and greater than two years is probably auditory deprivation.

Only $N1^1$showed significant difference because this response is mainly elicited by the low frequency portions of vowel (/i/) and it is already evident from electrophysiological studies that cortical response to the low frequency stimulus is better than response to the high frequency stimulus (Picton, Woods & Proulex, 1978). The reason is that, inherent lower amplitude of the cortical response when elicited from stimulus containing high frequency content such as /ʃ/ and /s/ compared to /m/, /a/, /u/ and /i/ which had predominant low frequency content as documented by Agung, Purdy, McMahon & Newall, 2006. One of the stimulus related reason is that /i/ portion of /si/ stimulus used in the present study had larger amplitude compared to /s/ portion. Hence, the better amplitude of $N1^1$.To explain further, physiologically, individuals with cochlear pathology have wider auditory filter, is a reason they are more prone to the effects of upward spread of masking (Moore, 1998). The preceding high frequency signals (fricative /s/) in the stimulus of this study could have been masked to an extent by low frequency vowel portion. It is evident from studies that low frequency portions of speech like vowels may mask higher frequency components like frication noise as reported by Dillon in 2001. Hence, the $N1^1$ amplitude was higher compare to other peaks.

4.4 Comparison of aided and unaided ACC within each subgroup.

Latencies and amplitude of P2, $N1^1$, $P2^1$ (compared only within subgroup A) and $N1^1$, $P2^1$ (compared within subgroup B, C, D) were compared using Wilcoxon

Signed Ranks test between unaided and aided condition within each subgroup. Latency and amplitude comparison of unaided and aided peaks of ACC were done, in order to study the effect of amplification along with degree and different duration of hearing loss on cortical neural representation of acoustical changes within speech sounds (syllable). The results of Wilcoxon Signed Ranks test for each subgroup is given in Table 4.9 below. The each within group comparisons of latency and amplitude of unaided and aided ACC is shown in graph 4.1 and 4.2 respectively.

Table 4.9: Results of Wilcoxon Signed Ranks test for comparison of peaks of unaided and aided ACC within each subgroup (A, B, C, and D). Z value and level of significance is shown for each comparison within each group

Subgroups	***Comparisons***	***Z value***	***Level of significance***
A	*Latency of P2 (aided) – latency of P2 (unaided)*	-.535	*.590*
	Latency of $N1^1$ (aided) – latency of $N1^1$ (unaided)	-1.15	.249
	Latency of $P2^1$ (aided) – latency of $P2^1$ (unaided).	*-.420*	.674
	Amplitude of P2 (aided) –amplitude of P2 (unaided)	-.535	.593
	Amplitude of $N1^1$ (aided) – Amplitude of $N1^1$ (unaided)	-2.20	.028*
	Amplitude of $P2^1$ (aided) – Amplitude of $P2^1$ (unaided)	-1.15	0.25
B	*Latency of $N1^1$ (aided) – latency of $N1^1$ (unaided).*	.000	1.00
	Latency of $P2^1$ (aided) – latency of $P2^1$ (unaided)	-1.06	0.28
	Amplitude of $N1^1$ (aided) – Amplitude of $N1^1$ (unaided)	-1.06	0.10
	Amplitude of $P2^1$ (aided) – Amplitude of $P2^1$ (unaided)	-.535	0.59
C	*Latency of $N1^1$ (aided) – latency of $N1^1$ (unaided)*	-.730	0.46
	Latency of $P2^1$ (aided) – latency of $P2^1$ (unaided)	-1.82	0.06
	Amplitude of $N1^1$ (aided) – Amplitude of $N1^1$ (unaided)	-1.82	0.06

	Amplitude of $P2^1$ (aided) – Amplitude $P2^1$ (unaided)	-1.46	0.14
D	*Latency of $N1^1$ (aided) – latency of $N1^1$ (unaided)*	-2.19	0.028*
	Latency of $P2^1$ (aided) – latency of $P2^1$ (unaided)	-1.60	0.10
	Amplitude of $N1^1$ (aided) – Amplitude of $N1^1$ (unaided)	-2.36	0.01*
	Amplitude of $P2^1$ (aided) – Amplitude of $P2^1$ (unaided)	-1.60	0.10

* Indicates statistically significant

4.4.1 Comparison of aided and unaided ACC within subgroup A

The latency and amplitude of P2, $N1^1$, and $P2^1$peaks were compared separately between unaided and aided condition within subgroup A using Wilcoxon Signed Ranks test.

4.4.1.1 Latency of P2, $N1^1$, $P2^1$

The results of the test revealed that there are no statistically significant difference latencies of P2, $N1^1$, and $P2^1$ between unaided and aided condition within minimal to moderate sloping SNHL with duration of hearing loss less than two years.

4.4.1.2 Amplitude of P2, $N1^1$, $P2^1$

Amplitude of P2, $P2^1$ also does not shown any statistically significant differences, whereas, $N1^1$has shown a statistically significant difference ($p = 0.028$). The amplitude of (mean amplitude of 0.54, shown in table 4.1) $N1^1$in aided condition is larger than in unaided (mean amplitude of -2.91, shown in table 4.1) within subgroup A.

4.4.2 Comparison of aided and unaided ACC within subgroup B& C

The latency and amplitude of $N1^1$, $P2^1$ peaks were compared separately between unaided and aided condition within subgroup B & C using Wilcoxon Signed Ranks test.

4.4.2.1 Latency of $N1^1$, $P2^1$

The results of the test revealed that there are no statistically significant difference in the latencies of $N1^1$, $P2^1$ between unaided and aided condition within minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss greater than & less than two years.

4.4.2.2 Amplitude of $N1^1$, $P2^1$

Amplitude of $N1^1$ and $P2^1$ does not shown any statistically significant differences between unaided and aided condition within minimal to moderate and moderate to severe sloping SNHL with duration of hearing loss greater than & less than two years respectively.

4.4.3 Comparison of aided and unaided ACC within subgroup D

The latency and amplitude of $N1^1$, $P2^1$ peaks were compared separately between unaided and aided condition within subgroup D using Wilcoxon Signed Ranks test.

4.4.3.1 Latency of $N1^1$, $P2^1$

The results of the test revealed that there are no statistically significant difference in the latencies of $P2^1$ but, there is a statistically significant difference in

$N1^1$ latency ($p = 0.028<0.05$) between unaided and aided condition within moderate to severe sloping SNHL with duration of hearing loss greater than two years .

4.4.3.2 Amplitude of $N1^1$, $P2^1$

Amplitude of $N1^1$ is also shown statistically significant differences ($p = 0.018<0.05$) between unaided and aided condition within moderate to severe sloping SNHL with duration of hearing loss greater than two years. But, there is no statistically significant difference in amplitude of $P2^1$.

Justification for the results under within subgroup comparisons

The results of the within group comparison of unaided and aided ACC in subgroup A, clearly throws light on to the effectiveness of amplification in sloping hearing loss with duration of hearing loss less than two years. There was appearance of N1 in aided condition; which indicates that the audibility of (Korczak, Kurtzberg & Stapells, 2005; Oates, Kurtzberg & Stapells, 2000; Wall, Dallebout, Davidson & Fox, 1991; Polen, 1984), the fricative portion was enhanced by amplification. The reason for absence of N1 in subjects minimal to moderate sloping hearing loss can be the upward spread of masking (Moore, 1998). In the present study low frequency - high amplitude vowel could have been masked the onset portion of fricative /s/. So, even minimal to moderate sloping SNHL with duration of hearing loss less than two years needs to be provided with hearing aids, even if they have minimal speech perception problems. The above statement has to be supported by further behavioral and electrophysiological studies.

The results of the within group comparison of unaided and aided ACC in subgroup B, throws light on to the effectiveness of amplification in sloping hearing

loss with duration of hearing loss more than two years. It is indicated that latency and amplitudes of unaided and aided condition did not show any significant differences, even though there is a difference in the mean value. The probable reason for this is auditory deprivation.

The results of the within group comparison of unaided and aided ACC in subgroup C, gives information on the effectiveness of amplification in moderate to severe sloping hearing loss with duration of hearing loss less than two years. There was appearance of N1, P2 in aided condition because of audibility at the higher frequency provided by the hearing aid. It is indicated that latency and amplitudes of unaided and aided ACC did not show any significant differences, though there is a difference in the mean value. The probable reason could be the abnormal growth of loudness phenomenon that occurred at the threshold level (Buus &Florentine, 2001) in these participants in the unaided condition but not in aided condition with probably may be contributed to the amplification factors by the hearing aid.

The results of the within group comparison of unaided and aided ACC in subgroup D, clearly throws light on to the effectiveness of amplification in moderate to severe sloping hearing loss with a duration of hearing loss more than two years. There was appearance of N1, P2 peak in aided condition even in individual with more than two years of deprivation because of audibility at the higher frequency provided by the hearing aid. It is indicated that latency and amplitudes of unaided ACC peaks ($N1^1$, $P2^1$) did not show any significant differences but there was a difference in latency and amplitude of $N1^1$ of aided ACC compare to unaided ACC. The reason for this finding is presently unclear.

Table 4.10: Summary of Results

Effect of degree of sloping SNHL on unaided and aided ACC	***Effect of duration of sloping SNHL on unaided and aided ACC.***	***Effect of amplification on ACC within each subgroup***
Minimal to moderate vs. moderate to severe < 2 yrs.	***I. Minimal to moderate vs. moderate to severe < and > 2 yrs.***	***Minimal to moderate < 2 yrs.***
Subgroup A vs. C	*Subgroup A vs. B*	N1 is present for two ears in unaided condition but, present for all ears in aided condition.
Unaided ACC	**Unaided ACC**	Amplitude of $N1^1$ is significantly larger (0.54uv) in unaided condition than aided condition (-2.92uv).
N1, P2 peaks were absent in subgroup C but, it was present (N1 in two ears, P2 in three ears) in subgroup A.	N1 was present for two ears in subgroup A, but, absent in subgroup B.	***Minimal to moderate >2 yrs***.
Latency of $P2^1$ is significantly shorter (321.93ms) in subgroup A than subgroup C (346.92ms).	P2 is absent for three ears in subgroup A, it was present only for two ears in subgroup B.	N1 and P2 are present for all ears in aided condition.
Amplitude of $N1^1$ is significantly larger (0.54uv) in subgroup A than subgroup C (-3.11uv).	Amplitude of $N1^1$is significantly larger (0.54uv) in subgroup A than subgroup B (-3.9uv).	***Moderate to severe <2 yrs.***
Aided ACC	**Aided ACC**	N1 and P2 are present for all ears in aided condition.
N1 and P2 peaks were present for subgroup A and C.	All peaks were present.	All other peaks did not show significant differences in latency and amplitude.
No difference in latency and amplitude of all peaks	Latency of P2 is significantly shorter (174.01ms) in subgroup A than subgroup B (203ms).	***Moderate to severe > 2 yrs.***
Subgroup B vs. D	*Subgroup C vs. D*	N1 and P2 are present for all ears in aided condition.
Unaided ACC	**Unaided ACC**	There was a significant difference in latency of $N1^1$; prolonged in unaided (263.79ms) than aided condition
N1 was absent in both subgroups. But, P2 was present for two ears in the subgroup B.	N1, P2 were absent for subgroup C and D.	
	Latency of all peaks did not show statistically significant differences.	
	Amplitude of $N1^1$ of	

Latency of $P2^1$ is (approaching significance) shorter (320.17ms) in subgroup B than subgroup D (348.60ms). Amplitude of $N1^1$ and $P2^1$ is (approaching significance) larger (-3.9uv) in subgroup B than subgroup D (-2.05uv). **Aided ACC** All peaks were present. Latency of $N1^1$ is (approaching significance) larger (262.07) in subgroup B than subgroup D (252.78). Amplitude of all other peaks did not show statistically significant differences.	subgroup C was (-3.12 uv) larger than subgroup D (-2.05uv). **Aided ACC** All peaks were present. Amplitude of N1of subgroup C was (-1.29uv) larger than subgroup D (0.02uv).	(252.78ms). Amplitude of $N1^1$ was smaller in aided condition (-2.05uv) compared to the unaided condition (-4.33uv).

Chapter 5

SUMMARY AND CONCLUSION

The study focused on the effect of degree and duration of hearing loss on the cortical representation of acoustical changes within speech sounds in individual with sloping sensorineural hearing loss (SNHL), in unaided and aided conditions. Degree and duration are the two variables adopted for this study with the assumption that degree and duration can have combined or separate effects on the coding of acoustical changes within speech sounds, at the cortical level. Based on this, the present study was designed with following objectives.

Firstly, to compare the unaided and aided ACC separately between minimal to moderate and moderate to severe degree of sloping sensorineural hearing loss.

Secondly, to compare between less than or greater than two years of duration of hearing loss in minimal to moderate & moderate to severe degree of sloping sensori-neural hearing loss separately.

Thirdly, to compare unaided and aided ACC within the two degrees of sloping SNHL with durations of hearing loss less than and greater than two years.

As a first step towards the objectives, the individual with sloping hearing loss were sub categorized in to four groups based on degree and duration of hearing loss.

i. Minimal to moderate hearing loss with duration of hearing loss less than two years (subgroup A).
ii. Minimal to moderate hearing loss with duration of hearing loss greater than two years (subgroup B).

iii. Moderate to severe hearing loss with duration of hearing loss less than two years (subgroup C).

iv. Moderate to severe hearing loss with duration of hearing loss greater than two years (subgroup D).

After categorizing in to four different subgroups, the optimized fitting of the hearing aid (four channel digital behind the ear hearing aid) and gain verification of the hearing aid in ear canal was done for participants. Aided ACC was recorded with /si/ stimulus, subsequently; the hearing aid was removed from the test ear and unaided ACC was recorded. The latency and amplitude of the N1, P2, $N1^1$, and $P2^1$ peaks of ACC were the target parameters considered for statistical analysis.

The latency and amplitude values of each peak were tabulated for each subgroup and statistically analyzed using Man Whitney test to study the effect of different degree of sloping SNHL (minimal to moderate and moderate to severe) and duration (less than two years and greater than two years) on unaided and aided ACC. The same statistical test was used to study the effect of different duration of hearing loss within a particular degree of hearing loss. Wilcoxon's signs ranks test was administered to find the effect of amplification within a particular degree of hearing loss. For this, latency and amplitudes of each peaks of aided and unaided ACC were compared. So, to reach the results of the present study, across group comparison and within group comparison were done.

The results of the each objective are summarized below:

I. The results of the *First objectives* of the study demonstrated under the two headings:

1. Minimal to moderate vs. moderate to severe < 2 yrs. (Subgroup A vs. C)

Unaided ACC

1. N1 and P2 peaks were absent in subgroup C, but, present (N1 in two ears, P2 in three ears) in subgroup A.
2. Latency of $P2^1$ is significantly shorter (321.93ms) in subgroup A than subgroup C (346.92ms).
3. Amplitude of $N1^1$ is significantly larger (0.54μv) in subgroup A than subgroup C (-3.11μv).

Aided ACC

1. N1 and P2 peaks were present for subgroup A and C.
2. No difference in latency and amplitude of all peaks

2. Minimal to moderate vs. moderate to severe > 2 yrs. (Subgroup B vs. D)

Unaided ACC

1. N1 was absent in both subgroups. But, P2 was present for two ears in subgroup B.
2. Latency of $P2^1$ is (approaching significance) shorter (320.17ms) in subgroup B than subgroup D (348.60ms).
3. Amplitude of $N1^1$ and $P2^1$ is (approaching significance) larger (-3.9μv) in subgroup B than subgroup D (-2.05μv).

Aided ACC

1. All peaks were present.
2. Latency of $N1^1$ is (approaching significance) larger (262.07) in subgroup B than subgroup D (252.78).
3. Amplitude of all peaks did not show any statistically significant differences.

II. The results of the *Second objective* of the study is demonstrated under the two headings:

1. Minimal to moderate vs. moderate to severe <2 years and >2 yrs.

Unaided ACC

1. N1 was present for two ears in subgroup A, but, absent in subgroup B.
2. P2 is absent for three ears in subgroup A. It was present only for two ears in subgroup B.
3. Amplitude of $N1^1$ is significantly larger (0.54μv) in subgroup A than subgroup B (-3.9μv).

Aided ACC

1. All peaks were present.
2. Latency of P2 is significantly shorter (174.01ms) in subgroup A than subgroup B (203ms).

2. *Moderate to severe < 2 yrs. Vs moderate to severe > 2 yrs.*

Unaided ACC

1. N1, P2 were absent for the subgroup C and D.
2. Latency of all peaks did not show statistically significant differences.
3. Amplitude of $N1^1$ of subgroup C was (-3.12 μv) larger than subgroup D (-2.05μv).

Aided ACC

1. All ACC peaks were present.
2. Amplitude of N1of subgroup C was (-1.29μv) larger than subgroup D (0.02μv).

III. The results of the *third objective* of the study demonstrated under the four headings:

1) Minimal to moderate <2 yrs.

1. N1 is present for two ears in unaided condition but, present for all ears in aided condition.
2. Amplitude of $N1^1$ is significantly larger (0.54μv) in unaided condition than aided condition (-2.92μv).

2) Minimal to moderate >2 yrs.

1. N1 and P2 are present for all ears in aided condition.
2. All other peaks did not show significant differences in latency and amplitude.

3) Moderate to severe <2 yrs.

1. N1 and P2 are present for all ears in aided condition.
2. All other peaks did not show significant differences in latency and amplitude.

4) Moderate to severe > 2 yrs.

1. N1 and P2 are present for all ears in aided condition.
2. There was a significant difference in latency (prolonged in unaided)

From the study we can conclude the following:

1. Since most of the peaks of ACC are present in aided condition compared to unaided condition in individual with varying degrees of sloping sensorineural hearing loss, ACC can be used as an objective measure to quantify the benefit from amplification.

2. Vowel portion of the speech stimulus is better coded in the cortical level compared to consonant portion as evidenced from larger amplitude of $N1^1$ and $p2^1$ compared to N1 and P2 in individuals with varying degrees of sloping hearing loss.

3. It is evident from the results of this study that the hearing aids are not able to give adequate amplification at higher frequencies as the fricatives are not being coded well at the cortical level compared to vowels. Hence, measures should be taken to improve the spectral content of the higher frequency sounds by the amplification device for individuals with sloping sensorineural hearing loss.

Implications of the study

1. There is a change in ACC in aided and unaided condition between individuals in subgroup A, and C (with short duration hearing loss), hence, it is possible to electro physiologically state that minimal to moderate sloping and moderate to severe sloping sensorineural hearing loss has got a separate effect on aided and unaided ACC. So, there is definite relationship between the degree of hearing loss and cortical representation. This knowledge gives an insight to the professionals to manage individuals with sloping sensori-neural hearing loss effectively.

2. Duration of onset of cochlear hearing loss has got an effect on ACC. This finding will give an electrophysiological highlight about the duration of cochlear hearing loss which can lead to reduced benefit from hearing aids due to auditory deprivation.

3. Duration of hearing loss is an important factor that contributes to the changes in the auditory system as evident from the unaided and aided ACC. Hence, this finding has implication in providing amplification even with a minor degree of hearing loss in order to avoid late onset auditory deprivation.

Future directions

1. The effect of duration of hearing loss and its effect on ACC to different speech sounds can be studied in more number of individuals with different configuration of hearing losses.

2. Further studies can focus on ACC in response to acoustical changes in the different speech sounds and its correlation with Behavioral Speech perception measures in individual with different degree and configuration of hearing loss.

3. The benefit of amplification in individuals with different configuration of hearing loss can be studied in naïve hearing aid users and long term hearing aid users by using ACC and behavioral speech perception measures.

4. The effectiveness of amplification in individuals with different duration of sensori-neural hearing loss can be studied longitudinally at regular smaller intervals.

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