

# **EFFECT OF HEARING AID CHANNELS ON ACOUSTIC CHANGE COMPLEX**

Giten Eliza George

Register Number: 10AUD013

A Dissertation Submitted in Part Fulfillment of Final Year

Master of Science (Audiology)

University of Mysore, Mysore



**ALL INDIA INSTITUTE OF SPEECH AND HEARING**

**MANASAGANGOTTHRI, MYSORE-570 006**

**MAY, 2012.**

## **CERTIFICATE**

This is to certify that this dissertation entitled “*Effect of hearing aid channels on Acoustic Change Complex*” is the bonafide work submitted in part fulfillment for the Degree of Master of Science (Audiology) of the student (Registration No.: 10AUD013). 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 - 570 006

## CERTIFICATE

This is to certify that this dissertation entitled “*Effect of hearing aid channels on Acoustic Change Complex*” 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 - 570 006

## DECLARATION

This is to certify that this Master's dissertation entitled "*Effect of hearing aid channels on Acoustic Change Complex*" 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.

Mysore

Register No. 10AUD013

May, 2012

## ACKNOWLEDGEMENT

Thank you Father, for “You reached down from on high and took hold of me; you drew me out of deep waters. You brought me out into a spacious place; You rescued me because you delighted in me. *Psalms 18:16, 19.*

The data collection of this dissertation has been one of the most significant academic challenges I have had to face. Without the patience and kindness of the following people I have mentioned, the writing of this dissertation will never be complete.

Importantly, I would like to thank all those who took the time to participate in this study, to each and every one of you. May God bless each and every one of you.

I would like to thank the Director S. R. Savithri & H. O. D (Audiology) Dr. Animesh Barman, Manjula ma'am for allowing us to use the instruments; this institute is a wonderful place.

I wish to express sincere appreciation to my guide, Sreeraj sir, for always prioritizing the dissertation work, for your suggestions and encouragement despite having many academic commitments. I am deeply grateful for the long discussions on the final drafts. Thank you for being so patient, and always being calm. I would also like to thank Ajith sir, Mamatha ma'am, Sandeep sir, Sujeet sir, Haemanth sir, Ganapathi sir, Antony sir and Arunraj sir for helping in any possible ways they could for completing this work.

I would like to thank the staff in Hearing Aid Trial Dept. Ramadevi ma'am, for being so sweet and kind, to always clear my every doubt about the hearing aids. Baba sir, for teaching me how to use Fonix 7000. Vasanthalakshmi ma'am for doing the statistics for me so quickly.

Thank you Jobish for your help and support throughout this dissertation and Merry for helping me understand the statistics, during a really busy time. Thank you Deepthi M. K. God has blessed me to grow spiritually through you. I could not have accepted the circumstances this dissertation had put me through without the knowledge you pointed out to me in the Bible. Kadi Songa, this acknowledgement will not be complete without writing how much you have helped me, from the beginning of this year till the very end. I learnt to be patient and submissive because of you. Thank you Preethi Sahu for all the help through the semester. Mahima Gupta for being a loving friend.

Lastly, I would like to thank all my classmates, all those who have put in so much effort handling the class duties, to CR: Zubin V., Saravanan P., Seby M.

My family, George Daniel and Mariamma George, my parents, who have always supported, so lovingly and unselfishly cared and for me.

Thank you God for all these wonderful people you placed in my life.

## TABLE OF CONTENTS

<i>Chapter No.</i>	<i>Title</i>	<i>Page No.</i>
1	Introduction	1
2	Review of literature	6
3	Method	21
4	Results and Discussion	32
5	Summary and Conclusion	47
	References	52

## LIST OF TABLES

<i>Table No.</i>	<i>Title</i>	<i>Page No</i>
<i>Table 3.1</i>	Stimulus and Acquisition parameters of ACC	24
<i>Table 3.2</i>	Acoustic Features of the /si/ stimulus	25
<i>Table 3.3</i>	Electro-acoustic measurement of the three hearing aids	27
<i>Table 4.1</i>	Unaided parameters obtained for the control and clinical groups	33
<i>Table 4.2</i>	Latency, amplitude and morphology (mean and standard deviations for two, four and eight channels)	39

## LIST OF FIGURES

<i>Figure No.</i>	<i>Title</i>	<i>Page No.</i>
<i>Figure 3.1</i>	The wave form of the stimulus /si/.	26
<i>Figure 3.2</i>	Spectrogram of the stimulus /si/.	26
<i>Figure 3.3</i>	Equipment set-up for ACC measurement.	27
<i>Figure 3.4</i>	Set-up for aided ACC measurement – Electrode and speaker placement.	30
<i>Figure 4.1</i>	Mean latencies (in milliseconds, ms) for the control and clinical group.	34
<i>Figure 4.2</i>	Grand mean waveform acquired from the participants with hearing sensitivity within normal limits & sloping sensorineural hearing loss.	36
<i>Figure 4.3</i>	Mean amplitudes (in microvolts, $\mu\text{v}$ ) for the control and clinical group participants.	37
<i>Figure 4.4</i>	Mean latencies for the three aided conditions.	41
<i>Figure 4.5</i>	Mean amplitudes for the aided condition of two, four and eight channels.	42
<i>Figure 4.6</i>	Grand mean waveform of the ACC obtained for the aided condition using the two, four and eight channel device.	43
<i>Figure 4.7</i>	Mean speech identification scores obtained for different conditions	44



## Chapter 1

### INTRODUCTION

The acoustic change complex is a cortical auditory evoked potential that can be obtained from the auditory system when a time varying acoustic change occurs within the stimuli. The change can be in terms of amplitude, spectral envelope or periodicity. Speech is one such stimulus which has multiple time varying acoustic changes, and can be used to elicit this potential (Martin & Boothroyd, 2000). Cortical auditory evoked potentials are primarily categorized as obligatory and cognitive (Wunderlich & Cone-Wesson, 2006) or discriminative (Purdy, Kelly & Thorne, 2001). The obligatory potentials depend solely on the acoustic features of the stimulus and the integrity of the central auditory system. Whereas, the cognitive or discriminative potentials (P300 and MMN) requires the use of an odd-ball paradigm and the listener should have the ability to discriminate between the frequent and infrequent stimulus. The ACC is an obligatory response which does not require active involvement from the participants.

There is interest in using this potential as it can probe into supra-threshold auditory skills, such as speech sound processing. This potential may be able to dwell deeper into the perception of speech as an index of speech sound discrimination as it comprises not only a detection waveform but also one which arises due to a change in the acoustic characteristics of the signal. The P1-N1-P2 complex indicates that the stimulus has been detected at the cortical level, and has led to the use of this response in threshold estimation.

The cortical auditory evoked potential was also verified and found to be useful as an objective measure of hearing aid outcome. As the hearing aid may introduce variations in the stimulus, if it does not faithfully represent all the acoustic features. Digital hearing aids with WDRC processing will shorten the rise time due to the occurrence of overshoot at stimulus onset which happens because of the time required to determine the input level and to stabilize the gain for a rapid change in input level when the stimulus level is above the compression threshold (Easwar, Glista, Purcell & Scollie, 2012). This results in larger CAEP amplitude and shorter peak latencies. But, the responses in the aided and the unaided condition were not found to be significantly different. From this result it can be concluded that hearing aid processing did not introduce deleterious effects on the stimulus that could preclude CAEP from being used as an assessment tool.

Aided late latency responses can be reliably recorded according to Tremblay, Billings, Friesen, and Souza (2006), which will yield a better understanding of how device settings will affect the neural response patterns which in turn affects speech perception. After varying a device settings or a feature, if a cortical response will still mimic stimulus acoustic features, then a conclusion can be drawn about both, speech perception and how the hearing aid processes the signal. In the current study, one such parameter, the number of channels of a hearing aid has been varied and the results have been analyzed. By using the fricative-vowel syllable /si/, the ability of the hearing aid to process the higher frequencies as well as the transition from a fricative to a vowel can be studied. The ability to discriminate formant transitions was compared across configurations by Danaher, Osberger and Pickett in 1973 and found that thresholds in the context of /i/ was significantly poorer compared to /a/ for those with sloping hearing loss.

In a sloping hearing loss, as the frequency increases the degree of hearing loss also increases (Roeser & Clark, 2007). Persons with sloping hearing loss due to a cochlear pathology can face problems while listening due to decreased audibility and dynamic range, also secondary to reduced frequency and temporal resolution. While studying the distribution of hearing loss characteristics in terms of configuration, Margolis and Saly (2008), found that sloping hearing losses dominated the distributions of configuration and that sensori-neural was the most prevalent site of lesion. Similar findings were also reported by Demeester et al., (2008) for high-frequency gently sloping and steeply sloping to be 76% in males and 50% in females between 55 to 65 years. This means that majority of the persons with hearing loss miss the high frequency portions of speech, which are consonants. Consonants as compared to vowels were found to be approximately 30 dB lower in intensity and are hence less audible (Zeng & Turner, 1990). Hearing aids can help alleviate these issues by selective amplification, as in by increasing only the gain of higher frequency components.

Selective amplification can be accomplished through hearing aids by various means, one of which is the use of multichannel hearing aids which split the incoming signal into frequency bands. A band in a hearing aid refers to a frequency region where gain adjustments are made and a channel is where the same signal processing takes place. Individual compression circuits allow these multichannel instruments the flexibility to amplify each bandwidth of frequencies independently so as to correspond to the user's needs, preferences and their dynamic range. In addition, each channel may be set with unique attack and release time for compression across the frequency range as reported by Dillon, 2001.

Now, the question may arise as to the number of channels necessary for optimal speech perception. There are various factors involved that can dictate the amount of benefit a multichannel hearing aid can provide. For example, the configuration, age, degree of hearing loss, ability to combine temporal-envelope information and hearing aid experience (Jyoti, 2010; Rubina, 2008; Souza & Boike, 2006; Yund & Buckles, 1995). There are conflicting findings about the benefit derived from multichannel hearing aids as authors have found that these hearing aids may reduce the spectral contrast which aids in the perception of vowels (Bor, Souza & Wright, 2008). According to Plomp (1988), this occurs when there are multiple channels with large compression ratios. However, Yund and Buckles (1995), demonstrated improvement in speech recognition from four to eight channels, Jyoti (2010) while comparing two, four and eight channels in listeners with sloping hearing loss has also reported similar findings. For this reason, the number of channels in a hearing aid was chosen as a parameter and an electrophysiological verification of the benefits or detrimental effects of having multiple channels has been explored and behavioral speech identification scores were correlated with the findings. Therefore, conclusions can be drawn on the effect of multi-channel hearing aids on the central auditory system processing of speech.

### *Need for the study*

Firstly, P1-N1-P2 evoked neural response patterns are heavily influenced by the acoustic content of the evoking signal, and hearing aids alter the acoustic content. For example, the perception through hearing aids can blur the boundary between the aperiodic noise of consonants and the onset of voiced vowels, making these transitions less distinct (Stelmachowicz, Kopun, Mace, Lewis, & Nitttrouer, 1995). To understand the interaction between the digitally amplified signal and its neural

representation, physiologic detection of CV transitions needs to be studied in a group of individuals who are first time users of hearing aids.

Secondly, the number of channels was selected as a factor because as documented by Rubina (2008) and Jyoti (2010) the more the number of higher frequency channels allotted, the better the performance on wordlists containing predominantly high frequency content (>1000 Hz). The same needs to be verified electro-physiologically through the use of the /si/ stimulus which has frequency content between two and five kHz. Additionally, a high frequency wordlist will be used to behaviorally verify the results. The results of the study can be used as a counseling tool which can help clients make informed decisions and understand the why higher or lower number of channels are required to suit their need.

### ***Aim of the study***

The aim of the study is to find the effect of varying the number of channels on ACC in individuals with sloping hearing loss and to verify the same using the speech identification scores.

### ***Objectives***

Firstly, to compare ACC in individuals with hearing sensitivity within normal limits and those with sloping cochlear hearing loss.

Secondly, to compare the aided and unaided acoustic change complex in individuals with sloping cochlear hearing loss, the further comparisons need to be done:

- i. Unaided v/s different no. of channels of hearing aids
- ii. Across different no. of channels of hearing aids

Thirdly, to correlate the speech identification score and the ACC obtained across different number of channels.

## Chapter 2

### REVIEW OF LITERATURE

The interaction of the number of channels of a hearing aid and the acoustic change complex can be better appreciated in the light of various factors that could affect the latency, amplitude or morphology like sensorineural hearing loss and the presence of amplification. The contribution of each of these factors has been delineated to acquire a holistic understanding of this subject.

2.1 Sloping sensorineural hearing loss

2.2 Review on effect of number of channels in a hearing aid

2.3 Cortical Auditory Evoked Potential (CAEP)

2.4 Acoustic Change Complex (ACC)

#### ***2.1 Sloping Sensorineural Hearing Loss***

The sloping hearing loss is operationally defined as the air-conduction thresholds occurring at successively higher levels from 250 to 8000 Hz (Pittman & Stelmachowicz, 2003). A sloping hearing loss, described as being gradual will have a 5-12 dB threshold increase per octave, or 15-20 dB threshold increase per octave described as sharply sloping, and precipitously sloping which can be defined as threshold increasing at the rate of 25+ dB per octave as described by Lloyd and Kaplan, 1978.

##### *2.1.1 Perceptual consequences of sloping hearing loss*

Sher, and Owens (1974) compared performance on phoneme identification for listeners with sloping sensorineural hearing loss above 2 kHz. The phonemes found to be difficult to perceive were /s/, /π/, /p/, /t/, /k/ and /b/ in both the initial and final

positions from which the probability of error with the /s/ phoneme was found to be the highest. Probabilities of errors on phonemes are closely related to the pure-tone configuration according to Owens, Benedict and Schubert, 1972 when considering the perception of fricatives. Patients with flat configurations from 500 to 8000 Hz experienced little difficulty in identifying /s /, /f/, /tʃ/, /dz/. Identification of the /s/ and the initial /t/ and /ʃ/ were highly dependent upon thresholds in the frequency range above 2000 Hz. Consequently, high frequency sloping hearing loss results in poorer perception of fricatives which have a higher spectral content as compared to other consonants and vowels. Adding to this, the perception may not only depend on the spectral content of the consonant of interest but also on how these are modified in different vowel environments because of co-articulatory effects.

As a result of coarticulation a phonological segment is not identified to be the same in all environments, but varies to become more like an adjacent segment (Hardcastle & Hewlett, 1999). The ability to discriminate formant transitions was compared across configurations by Danaher, Osberger and Pickett in 1973 in the context of /s/ with different vowels. Results showed that discrimination of formant transition in the context of /a/ with mid-frequency spectral content was same for those with normal, flat and sloping sensorineural hearing loss. But, thresholds in the context of /i/ were significantly poorer for those with sloping hearing loss. Therefore, the ability to use co-articulatory cues will also be impaired. The prominent spectral peak is over 1000 Hz higher for the /i/ context than for the /u/ context as the frequency content averaged 4.9, 5.6, 6.0 kHz in the context of /u/, /a/ and /i/ according to Boothroyd and Medwetsky (1992).

### *2.1.2 Perception of fricatives and the corresponding transition cues by hearing impaired*

Zeng and Turner (1990) studied the perception of the fricative-vowel syllable-*/si/*. The fricative portion was 200 ms, with voicing starting at the offset of frication and immediately following the voicing was a 40-ms formant transition period followed by the vowel */i/*. The results from this experiment suggested that the transition cue was used by normal hearing subjects for recognizing the voiceless fricative at low presentation levels. In contrast, for the hearing impaired, the primary factor influencing perception was the low intensity of the frication cue (relative to the vowel) and their poorer than normal discrimination ability. To conclude, hearing impaired listeners do not effectively use transition cues which are rapid, and depend more on the direct cue which is the frication. Due to slope in the high frequency regions, when the vowel portion had been amplified the frication portion would still be inaudible because it is 30 dB lower than the vowels in intensity. The authors concluded stating that for */si/* perception, any effort to increase the audibility of the frication portion will result in the improvement of fricative intelligibility but the transition cues cannot be compensated through amplification.

Portions of the signal that are least changing (vowel nuclei or consonantal continuants) are less informative than portions that are more dynamic and influenced by co-articulation such as transitions. Steady-state spectral cues and speech envelope cues are perceived equally well by hearing-impaired listeners as they are by persons with normal hearing, when sufficient audibility is provided (Turner, Souza & Forget, 1995; Zeng & Turner, 1990). On the other hand, studies have shown that other types of speech cues like transitions with dynamic frequency changes are not perceived as well, even when audibility is provided (Zeng & Turner, 1990).



## *2.2 Review on effect of number of channels in a hearing aid*

A channel in a hearing aid is a bandwidth within which an independent amplifier is assigned to control the compression characteristics. In a single channel hearing aid, the entire dynamic range is optimized across all frequencies by a single compressor. In multichannel hearing aids, this dynamic range is optimized at discrete frequencies by using multiple compressors. During multichannel compression the input to the hearing aid is passed through band pass filters, compression is independently applied to the output of each channel, and the processed signals are summed into a single broadband signal, which is the hearing aid output (Edwards, 2004).

### *2.2.1 Upper frequency limit and high frequency emphasis*

With the purpose to determine the upper frequency limit of a hearing aid that will provide access to spectral cues for all sounds, Boothroyd and Medwetsky (1992) reviewed the /s/ stimulus. They concluded that for a hearing aid to reproduce the lowest frequency prominent spectral peak in /s/ for most adult talkers an upper frequency limit of 10 kHz or a limited frequency transposition scheme will be required. For stimuli /s/, the prominent spectral peak can vary from 2.9 kHz to 8.9 kHz. If a hearing aid has an upper frequency limit of 6 kHz, acoustic information above 6 kHz will not be amplified, potentially making the frequency content more similar to /Σ/. Even when hearing aids are capable of amplifying higher frequencies, this process alters timing cues.

Yund, and Buckles (1995) did a detailed analysis of consonant discrimination and how perception of the stimulus changed with increasing channels. Increasing the number of channels yielded more improvement for middle consonants than front or

back consonants, and improved fricative perception than nasals and glides, and voiceless stops more than voiced stops. The authors analyzed the frequency responses of the multichannel instruments and found the average amplification at 4 kHz and above increased as the number of processing channels increased from 4 to 8 to 16. The improved high frequency response for instruments with more channels of processing, which resulted in better transmission of high-frequency speech cues helped to account for the noted improvements in consonant discrimination. At higher frequencies, the greater ability of the 8 and 16 channel hearing aids to respond to spectral variation is most evident compared to the four channel hearing aid.

### *2.2.2 Speech perception across 2, 4 and 8 channels for different configurations*

Jyoti (2010) found the effect of number of channels on speech perception for flat, moderately sloping and steeply sloping configurations. Performance of subjects with digital hearing aids of two, four and eight channels was verified with a phonetically balanced (PB) and a high frequency word list. A trend towards an increase in the speech identification scores with increase in the number of channels within each group was noted with this increase being greater for the two sloping hearing losses compared to the flat hearing loss group with the PB wordlist. Use of the high frequency word list showed no significant change in the speech perception scores with increase in the number of channels from two to four. However, there was a significant change increase in the scores with increase in number of channel to 8. In this study, increase in the number of channels resulted in improved audibility and better frequency shaping. Significantly better performance was observed with the 8 channel hearing aid for the high frequency wordlist which has an energy concentration beyond 1000 Hz, which was attributed to the greater number of

channels (five) in the high frequency region. For the two and four channel hearing aids, the number of channels in the high frequency region was one and two respectively which resulted in poorer scores. These effects were better realized for the sloping hearing loss configurations. The author concluded stating that individuals with high frequency sloping hearing loss are likely to benefit from hearing aids with more than four channels.

### *2.3 Cortical Auditory Evoked Potentials*

N1/P2 complex reveals the synchronous neural activity of structures in the thalamo-cortical segment of the central auditory system. The time after stimulus presentation, or latency, is recorded in milliseconds and reflects neural travel time. The N1, a negative peak occurring approximately 100 ms after stimulus onset, was found to represent sound detection by Näätänen and Picton (1987), since it is sensitive to onset sound features, such as intensity and inter-stimulus interval. P2, a positive peak occurring approximately 200 ms after stimulus onset, is assumed to reflect sound content properties like acoustic or phonetic structures.

The CAEP's have been used successfully to serve purposes like threshold estimation (Lightfoot & Kennedy, 2006) with 94% of individual threshold estimates within 15 dB of the behavioral threshold and 80% within 10 dB. The effects of sensorineural hearing loss on CAEP's have been investigated by Oates, Kurtzberg and Stapells (2002) for N1, N2, P3 and MMN. Prolongations in the latencies relative to responses from normal-hearing subjects began with mild (25 to 49 dB HL) threshold elevations. The CAEP's are also sensitive to stimulus features in the temporal domain.

### *2.3.1 Temporal characteristics of the stimulus*

Cortical auditory evoked potential can encode categorical perception of voice-onset time (VOT) as revealed by Sharma and Dorman (1999). A sharp category boundary was revealed between /da/ and /ta/ for normal hearing listeners using N1 CAEP. Two peaks appeared (N1 and N1') when the temporal spacing between the burst and voice-onset was 50 ms which is also the VOT value at which listeners' behavioral identification of sounds changed to /ta/. Latency of these components imply that N1 occurred in response to the burst at syllable onset, while N1' was elicited by onset of voicing. Similar findings were reported by King, Campbell, Sharma, Martin, Dorman and Langran (2008), for /ta/ and /da/ stimulus.

The same precision in encoding temporal cues was noted for fricatives /si/ and /Σi/. Significantly earlier positive and negative peaks were elicited by the /Σi/. Reflecting the earlier onset of the vocalic portion, because the fricative portion is shorter in duration, and the onset of the vowel earlier, for the /Σi/ stimulus compared with the stimulus /si/ (Tremblay, Friesen, Martin & Wright, 2003). Just as the CAEP's can accurately encode temporal features evidenced through latency differences in the peaks obtained, the spectral content of the stimulus can also dictate the latency and the amplitude of the peaks acquired.

### *2.3.2 Frequency content of the stimulus*

Effect of sensorineural hearing loss on speech evoked aided auditory late latency response (ALLR) was studied by Apeksha and Devi (2010). Three different speech stimuli were used /ba/, /ga/ and /da/ which have spectral energy concentration in low, mid and high frequency respectively. Individuals with normal hearing and moderate to moderately-severe sloping SNHL were compared. Aided ALLR recorded

by spectrally different speech sounds are different in individuals with normal hearing and sloping SNHL. This suggests that neurophysiological processes are different for different speech sounds. Longer latency for /da/ suggests that latency of the processing at the cortical center was also different depending on the frequency composition of the signal. The authors proposed the reason to be the tonotopicity of the primary auditory cortex, because according to Yetkin, Roland, Purdy and Christensen (2004), the cortical area responding to low frequency auditory stimuli are located more superficially than the deep layer of the cortical regions for high frequency. Hence, low frequency stimuli produce smaller latency of ALLR than high frequency speech sounds. Cortical tonotopical layering has also be quoted by Dimitrijevic, Michalewski, Zeng, Pratt and Starr (2008) to be one of the reasons why stimulus with higher frequencies do not evoke responses similar to those of lower frequencies.

Dimitrijevic et al., (2008) observed a larger N100 response to the low frequencies suggests that a larger population of neurons is activated for processing compared to higher frequencies. They reasoned that the amplitude differences were related to the low frequencies which cause a basal spread of activation along the basilar membrane and therefore a larger pool of neurons can be excited; and the generator of the high frequency response was located deeper below the surface of the cortex and therefore through volume conduction resulted in smaller amplitudes.

Agung, Purdy, McMahon and Newall (2006) obtained CAEPs produced by seven different speech sounds /a/, /u/, /i/, /s/, /Σ/, /m/ and /□/. Speech sounds dominated by higher-frequency spectral energy, such as /s/ and /Σ/, elicited CAEPs with smaller N1 and P2 amplitudes than speech sounds with dominant spectral energy

in the lower frequencies. N1 and P2 evoked by speech sounds may reflect the tonotopic organization of auditory cortical areas. Cortical areas that respond to low frequency auditory information are located more superficially (i.e., closer to the surface of the scalp) than cortical regions for higher frequencies according to Yetkin et al., (2004). The high front vowel such as /i/ evoked CAEPs that have earlier latencies than for low mid-back vowels /u/. Vowels with large F2-F1 differences such as /i/ (~2300 Hz) and /u/ (~1700 Hz) have larger areas of activation and therefore elicit a response that occurs at a different time compared to a vowel with a small F2-F1 distance such as /a/. Significant effects of stimulus duration (500 ms versus 100 ms) were also observed, with shorter duration stimuli producing larger amplitudes and earlier latencies than a longer duration one. This research demonstrates that CAEPs can be reliably evoked by sounds that encompass the entire speech frequency range. Further, CAEP latencies and amplitudes may provide an objective indication that spectrally different speech sounds are encoded differently at the cortical level. Lastly, the intensity of the stimulus in terms of audibility is important to consider as it is the basis of objective testing individuals with hearing loss.

### *2.3.3 Intensity of the stimulus*

The N1-P2 response is sensitive to changes in amplitude modulation and changes in intensity. Harris, Mills and Dubno (2007), elicited the N1-P2 by an intensity increase in a continuous pure tone presented at 70 dB SPL using intensity increments up to 5 dB at 500 Hz and 8 dB at 3000 Hz in 1-dB steps. Intensity discrimination threshold was defined as the smallest intensity change needed to evoke an N1-P2 response, discrimination thresholds in the study ranged from 2–3 dB at 500 Hz and 2–4 dB at 3000 Hz in younger subjects. A common conclusion which can be drawn from the literature on temporal, spectral and intensity encoding is that the

CAEP's are an accurate and sensitive measure of changes within each of these domains and can be explored as a tool for understanding neural encoding of changes within a stimuli.

#### *2.3.4 Aided CAEP's*

The validity of using the CAEP as an objective measure of hearing aid outcome was investigated because hearing aid processing can cause stimulus modifications which will result in erroneous findings. CAEPs were obtained to unprocessed and multichannel hearing aid processed tone bursts from individuals with normal audiometric thresholds. Shortening of rise time and overshoot at the onset of the tone burst were evident in the hearing aid processed stimuli. Digital hearing aids with WDRC processing will shorten the rise time due to the occurrence of overshoot at stimulus onset. This overshoot occurs because of the time required to determine the input level and to stabilize the gain for a rapid change when the stimulus level is above the compression threshold (Dillon, 2001). This results in larger CAEP amplitude and shorter peak latencies. The effect of altered rise time may influence aided CAEPs recorded in individuals with cochlear hearing losses differently since poorer temporal processing relative to individuals with normal hearing (Florentine, Fastl & Buus, 1988). Statistical analysis of data showed no significant effects of hearing aid processing on the latency or amplitude of CAEP peaks (Easwar et al., 2012). Therefore, the CAEP can be used as an objective measure of verifying hearing aid outcome.

Billings, Tremblay, Souza and Binns (2007), found that as a 1000Hz tone intensity increased from 30 to 90 dB peSPL; P1, N1, P2, and N2 latencies decreased significantly, and N1, P2, and N2 amplitudes enlarged significantly. There were no

effects of amplification for latency or amplitude for any of the CAEP components. Unaided and aided real ear recordings from the participant ear canals were used to verify that the hearing aid was producing the desired amount of gain. The difference between unaided and aided output demonstrated that ~ 20 dB of gain was provided by the hearing aid through most of the intensity range. Therefore, 20 dB of hearing aid gain affects neural responses differently than 20 dB of stimulus intensity change. A 60 dB stimulus presentation level and a 40 dB presentation level with 20 dB gain of the hearing aid did not produce similar waveforms. This suggests that the amplification from a hearing aid changes not only the intensity of the stimulus, which leads to differences in the way the aided stimuli are interacting with the CAS.

Compression characteristics can also affect stimulus rise-slope, rise time, and introduce overshoot of the onset (Billings, Tremblay & Miller, 2011). When the input level is constant in the aided condition with varied hearing aid gain, the experiment simulates a condition that might take place in a clinical setting where hearing aid gain might be altered to estimate hearing thresholds, or evoke a desired response. For instance, evoked potentials and real ear measures were recorded in normal-hearing individuals in unaided and aided conditions. In the aided condition, a 40dB signal was delivered to a hearing aid programmed to provide four levels of gain (0, 10, 20, and 30 dB). As a control, unaided stimulus levels were matched to aided condition outputs (i.e. 40, 50, 60, and 70 dB) for comparison purposes. When signal levels are defined in terms of output level, aided CAEPs were smaller and delayed relative to unaided CAEPs, resulting from increases to noise levels caused by the hearing aid. In contrast, these results show that hearing aids modify stimulus characteristics such as SNR, which in turn affects the CAEP and does not reliably reflect hearing aid gain.



## 2.4 Acoustic Change Complex

Ostroff, Martin and Boothroyd (1998) found two overlapping P1-N1-P2 responses in response to the naturally produced syllable /si/. The first P1-N1-P2 complex reflected the onset of the sibilant /s/; the second P1-N1-P2 response reflected the consonant vowel (CV) transition (/s/ to /ei/). In the year 1999, Martin and Boothroyd termed this evoked response the *Acoustic Change Complex (ACC)*. It has not yet been determined whether the onset P1-N1-P2 and the ACC (i.e., the second P1-N1-P2 complex) share the same generators and tap identical processes. ACC was observed in response to a fricative-vowel transition, which involved simultaneous changes of spectral envelope, periodicity, and amplitude. The following study considers the effect of amplitude alone which is capable of eliciting an ACC.

Martin and Boothroyd in 2000, probed into stimulus modifications with the purpose of confirming if the ACC can be elicited by an amplitude change alone, in the absence of changes of spectral envelope or periodicity and effect of simultaneous change in the amplitude and spectrum. The following observations were made: For the stimuli and protocols used in the study, an amplitude change of at least +2 or -3 dB was required in order to produce an ACC. The ACC amplitude is greater for amplitude increments than for amplitude decrements. To explain for amplitude increments, the ACC amplitude continues to increase as the amplitude change increases. Whereas, for amplitude decrements, there is no evidence to show that the ACC amplitude continues to increase with increasing amplitude change. The addition of an amplitude increment to a spectral change has the effect of increasing amplitude of the ACC. The ACC in a signal with constant rms amplitude, reflects the auditory system's response to the spectrum and not to accompanying changes of perceived

magnitude of the signal. Clearly, an ACC can be elicited with a change in amplitude; research on periodicity change alone has also revealed similar findings.

To determine whether the periodicity alone in the middle of an ongoing stimulus can elicit an ACC, Martin and Boothroyd (1999) used a tonal complex and a band of noise having the same spectral envelope and rms intensity. An ACC can be elicited by the change in periodicity, or by the abrupt intensity rise of the second portion of the stimulus. The noise-tone and tone-noise stimuli had two N1-P1 components in the waveform compared to the responses to the noise-only and tone-only stimuli which showed a single N1-P2 complex to the onset of stimulation followed by a sustained potential that continued until the offset of stimulation.

#### *2.4.1 Aided Acoustic Change Complex*

It is essential to understand the effects of amplification on the central auditory system as this can tap into how neural representation of speech perception is modified by a device. Hearing aids may modify the inherent spectral and temporal variations of a speech signal due to processing. But, these modifications have not been so detrimental as to circumvent the use of CAEP as an objective tool to measure hearing aid outcome.

Similar to the study done by Billings et al., (2007), aided ACC was performed by Tremblay et al., (2006) for individuals with normal hearing. This population was chosen because cortical potentials are sensitive to peripheral pathology, making it difficult to separate the effects of hearing loss and amplification. The purpose of their study was to determine whether 20 dB of hearing aid gain would alter ACC waveforms. They had also expected that adding 20 dB of hearing aid gain would have the same effect as increasing the stimulus intensity by 20 dB. On the contrary, their

results showed no significant differences between aided and unaided cortical potentials in response to both /s/ and /ʒ/ stimulus. If the consonant-vowel boundary is preserved by the hearing aid, it can also be detected neural level. For example, stimulus /si/ and /ʒi/ which differ in that the fricative portion as /ʒi/ contains lower spectral energy and is shorter in duration than the fricative portion of /si/. They found test-retest reliability to be good in both unaided and aided conditions. Mild high frequency emphasis hearing aids were used. Similar to unaided findings, the N1-P2 response corresponding to vowel onset was significantly earlier for the /ʒi/ stimulus by 30 msec. The differences found between the aided and the unaided conditions were the N1 corresponding to the acoustic onset of /s/ was greater in amplitude than /ʒ/ in the unaided condition but this differentiation was not seen once the speech tokens were processed by the hearing aid. /s/ and /ʒ/ stimulus portions did not significantly differ in response amplitude. Amplification could have modified the fricative portions, making them similar. It could also have been that the high-frequency energy in /s/ was not amplified, making it similar to /ʒ/. The findings could have been a result of hearing aid compression which limited the output. The small sample size (7 participants) might have been insufficient to observe more subtle effects of amplification (unaided versus aided). Stimulus presentation level was 64 dB peSPL, so the suprathreshold amplified stimulus sensation level approached 70 dB. According to Adler and Adler (1989), the latency and amplitude intensity functions for the N1 response asymptote at sensation levels approximating 70 dB SL. This could explain why subtle suprathreshold intensity increases, provided by the hearing aids, did not result in robust amplification effects in the study.

Aided ACC was performed by Tremblay, Kalstein, Billings and Souza (2006b) for individuals with sloping sensorineural hearing loss using a linear hearing

aid. Stimulus /si/ and /Σi/ were used to determine whether the amplification differentially altered the onset of consonants /s/ and /Σ/. Both stimuli elicited distinct ACC neural response patterns. The CV transition for /Σi/ evoked a negative peak that was earlier than the corresponding peak evoked by /si/. Only if the CV transitions were preserved by the hearing aid and audibility was sufficient for each listener, could the stimuli be correctly identified by each hearing aid user. Therefore, the hearing aid could faithfully represent the consonants with their corresponding transitions and the same could be verified with the aided ACC.

## Chapter 3

### METHOD

The purpose of this study was to learn the effect of the number of hearing aid channels in individuals with sloping hearing loss using ACC, which is a late latency auditory evoked potential generated in response to speech stimulus with inherent variation in spectral envelope, periodicity and amplitude. Additionally, the behavioral and electrophysiological responses were correlated across different number of channels.

To study the effect of sloping cochlear hearing loss alone, a control group with normal hearing sensitivity was included and their latency, amplitude and morphology were compared to that of the group with hearing loss. To achieve the mentioned aims, the participants progressed through the following phases.

#### *Participants*

*Control group:* 16 individuals (20 ears) with normal hearing in the age range of 25-59 years comprised the control group. The following criteria were used for subject selection.

- i. Hearing sensitivity of air conduction and bone conduction thresholds - less than 20 dB HL across frequency range from 250 Hz to 8 kHz in both ears.
- ii. Normal middle ear status
- iii. Native Kannada speakers.

*Clinical group:* A total of 10 participants (11 ears) in the age range of 25-59 years with cochlear hearing loss of a sloping configuration and have the following audiological profile.

- i. Air conduction thresholds increased by 5-12 dB per octave (Vogel & Kaplan, 1978)
- ii. Speech identification score of >55% in the test ear.
- iii. Normal immittance results, defined as peak pressure between -100 and +60 dapa, and admittance between 0.3 and 1.60 cc.
- iv. Native speakers of Kannada language.
- v. Naive hearing aid users.

### ***Instrumentation***

- a. A calibrated double channel audiometer, GSI- 61 was used to estimate the pure tone thresholds and to obtain speech identification scores.
- b. GSI Tymptstar (version-2) middle ear analyzer was used for immittance measurements.
- c. Bio-Logic Navigator Pro (version 7.0.0), a 2-channel diagnostic auditory evoked potential measuring instrument, was used for electrophysiological recording to obtain synchronous presentation of the stimulus and to record the corresponding ACC waveform.
- d. 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 speech stimulus to record ACC in aided and unaided condition.
- e. Fonix 7000 hearing aid test system was used for electro-acoustic measurement of hearing aids and for real ear measurement.

### ***Test environment***

The recording of the test stimuli and the audiological testing were done in an acoustically treated room, with the noise levels within permissible limits according to ANSI S3.1, 1991. Pure tone audiometry was done in a double room suite, and the ACC acquisition in a single room set-up.

### ***Procedure***

Procedure involved four phases:

- 1) Acquisition of ACC for participants in the control group
- 2) Hearing aid fitting for clinical group
- 3) Acquisition of aided ACC for clinical group
- 4) Obtaining speech identification score in clinical group

#### ***Phase 1: Acquisition of control group ACC***

As a pre-requisite to record ACC, the following audiological procedures were followed:

- i. Case history
- ii. Pure Tone Audiometry
- iii. Immittance

The procedure started with taking a detailed case history probing into any history of ear related pathologies. Pure tone thresholds were obtained in octave intervals between 250 Hz to 8000 Hz for air conduction and between 250 Hz and 4000 Hz for bone conduction using the modified Hughson-Westlake procedure (Carhart and Jerger, 1959). Tympanometry and reflexometry were done to exclude individuals with middle ear pathology. To accomplish the same, a group of 50

individuals were administered the diagnostic protocol mentioned above, after which 16 of them (20 ears) were included in the study.

The ACC was used in this study to learn the neural representation of a fricative /s/, its transition to a vowel, and perception of the vowel /i/. If these elements are perceived at the cortical level, two distinct waveforms will be observed, one in response to the consonant and the other for the vowel.

To learn the effect of sloping sensorineural hearing loss, the clinical group's unaided ACC waveforms were compared to that of the control group; the differences in latency, amplitude and morphology between the two responses gave information on the effect of sloping sensori-neural hearing loss on ACC. The inclusion of a control group can prevent extrinsic variables from influencing results, as both groups comprised native Kannada speakers, with no experience listening through an amplification device. Additionally, cognitive abilities that might influence the processing speech stimulus at the level of the auditory cortex can be taken into consideration. The stimulus and the acquisition parameters are detailed in Table 3.1.

Table 3.1 Stimulus and Acquisition parameters of ACC

<i>Stimulus parameters</i>		<i>Acquisition parameters</i>	
Stimuli	/si/	Mode of stimulation	Ipsi
Duration of stimulus	250 msec	Electrode montage	Cz, M1/M2 of test ear, ground at Fz
Intensity	65 dB SPL	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

### *Stimulus Preparation*

To elicit ACC, /si/ stimulus was used, which was naturally produced and recorded using Adobe Audition (version 1.5) at a sampling rate of 48,000 with 16 bit resolution. A dB Technologies-160 free field speaker was used to present the /si/ stimulus for recording ACC in both the aided and unaided conditions. The output of the speaker was calibrated using a Larson-Davis System-824 sound level meter to be presented at a level of 65 dB SPL. The speaker was positioned at a distance of one meter at 45° azimuth. With the use of Pratt© software (version 5.1.31) the acoustic features of the /si/ stimulus was analyzed and is presented in Table 3.1, Also, the stimulus waveform is shown in Figure 3.1, with the corresponding spectrogram in Figure 3.2.

Table 3.2 Acoustic features of the /si/ stimulus

<b><i>Acoustic Features</i></b>	<b><i>Values</i></b>
Total Duration	250 ms
Fricative Duration	143 ms
Vowel Duration	107 ms
Fricative Center Frequency	Energy spread from 2 to 5 kHz
Fundamental frequency - Vowel	75 Hz
First Formant	1059 Hz
Second Formant	2557 Hz

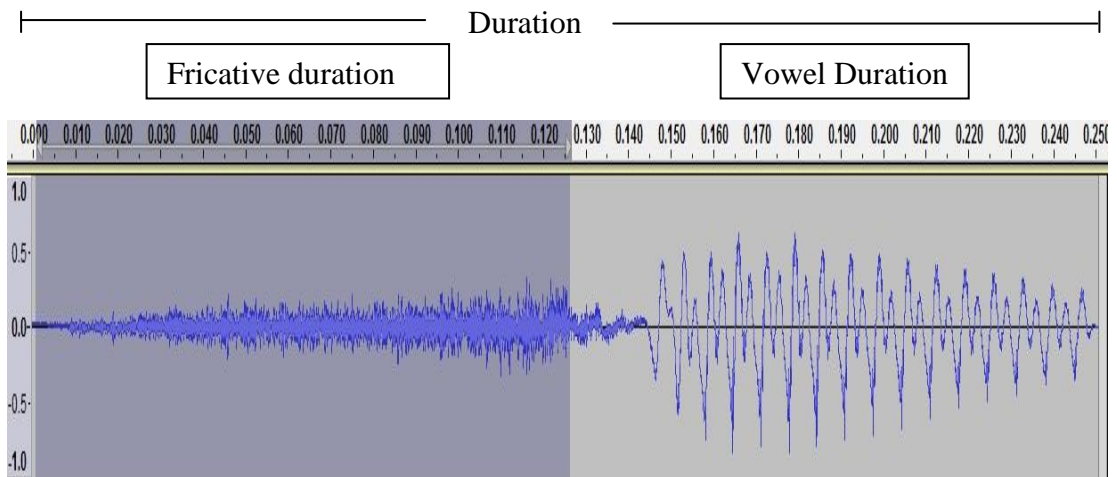


Figure 3.1: Wave form of stimulus /si/.

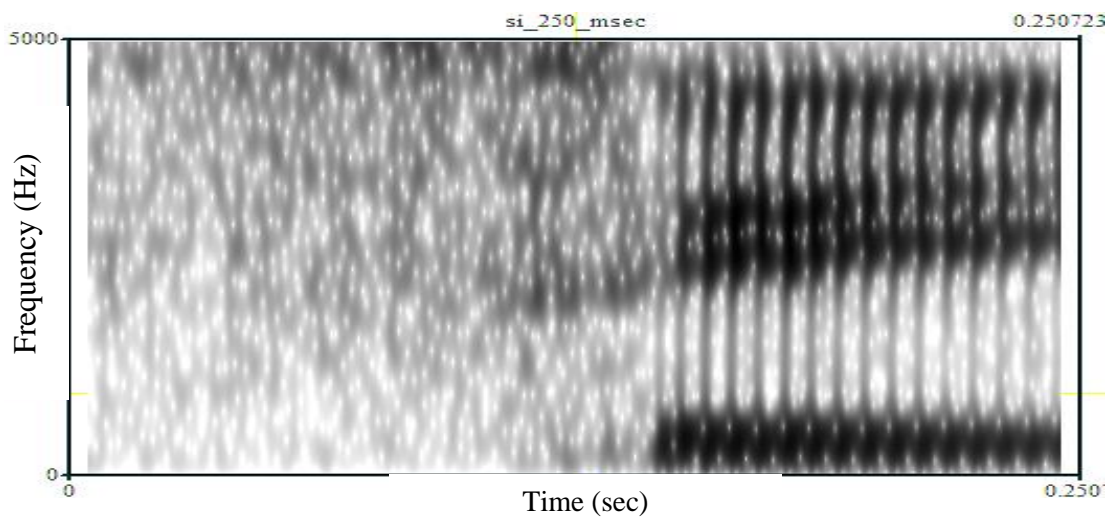


Figure 3.2: Spectrogram of stimulus /si/.

While acquiring the ACC, participants were seated comfortably on a reclining chair. The electrode site was cleaned with Nu-prep gel and gold disc electrodes were placed with Ten-20 conductive paste at the test site. The inter electrode impedance was maintained to be less than 2 K $\Omega$  and was monitored throughout the recording. The participants were instructed to ignore the stimulus and minimize head movements. A total of 300 sweeps were acquired for each participant. If the waveform was not found to replicate, a third recording was done.

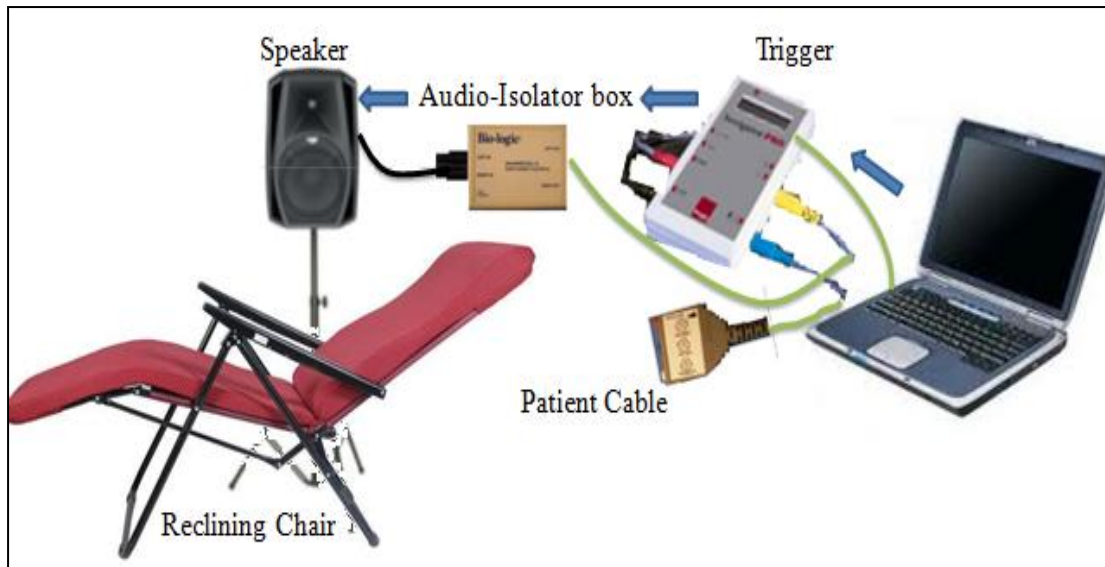


Figure 3.3: Equipment set-up for ACC measurement.

## ***Phase 2: Hearing aid fitting for the clinical group***

### *Electro-acoustic Measurement*

Two, four and eight channel hearing aids of the same make and manufacturer were utilized for the study. On electro-acoustic measurement with the hearing aid in test setting [ANSI S3.22-2003; (2 cc coupler)], the values mentioned in Table 3.3 were obtained.

Table 3.3 Electro-acoustic measurement of the three hearing aids

<i>Electro-acoustic measure</i>	<i>2 Channel</i>	<i>4 Channel</i>	<i>8 channel</i>
Output Sound Pressure Level (OSPL) at 2100 Hz	130.7 dB	129.8 dB	131.3 dB
HFA- FOG	53.2 dB	49.8 dB	59.3 dB
Reference Test Gain	47.1 dB	46.4 dB	46.3 dB
Frequency Range	200 – 5785 Hz	200 – 5787 Hz	200 – 6212 Hz
EIN	12.4 dB	10.1 dB	16.0 dB

THD			
500 Hz	2.0 %	1.2%	0.5%
800 Hz	1.4 %	1.3%	0.4%
1600 Hz	0.1 %	0.1%	0.2%

Note: THD = Total Harmonic Distortion, EIN = Equivalent Input Noise, HFA-FOG = High Frequency Average-Full On Gain.

### *Hearing aid programming*

The NOAH 3 software using the Connexx platform, connected to HiPro was used for programming, the client would be seated with the probe microphone and the programming system placed in the same room to simultaneously match the target while programming the hearing aid. The hearing aids were in omni-directional mode with enabled compression circuits, and compression ratio set according to NAL-NL1. The amount of gain provided by the hearing aid was verified in 2 cc coupler using hearing aid analyzer. At an input of 65 dB SPL, gain provided across 250 Hz to 6500 Hz was verified. Of the four programmable memories in each hearing aid, only one that is the speech in quiet mode was activated, also the volume control was disabled.

NAL-NL1 fitting formula was used to prescribe the gain across different channels and accordingly the hearing aid was programmed to match the target gain at acclimatization level two. To specify the amplification targets for speech and the maximum output necessary to provide loudness comfort, audibility of speech, and speech intelligibility, this prescriptive formula was used.

### *Real Ear Measurement*

To quantify the stimulus level at the output of the hearing aid, a probe-tube microphone system was placed in the ear canal, and sound level measurements were made with the hearing aid. In-the-canal acoustic recordings provide precise stimulus

intensity measurements because they take into account individual pinna, concha, and canal effects. In order to achieve measurement accuracy up to 6 kHz, the probe tube was placed within 5 mm of the tympanic membrane. This is accomplished by placing a probe tube inserted 5 mm beyond the medial portion of the ear tip. Computation of hearing aid delay was done to remove the effect of the hearing aid processing delay while analyzing the latency. Processing delay of the 2 - channel hearing aid was 1.5 ms, and the 4 and 8 channel aids had 1.8 ms and 3.3 ms delays respectively. However, as the delays were very small compared to the standard deviation of the ACC, it was not taken into consideration during final analysis.

### ***Phase 3: Acquisition of aided ACC for clinical group***

First, a participant of the clinical group was comfortably seated on a recliner in the sound treated room. The electrode placement is as mention in Table 3.1 and depicted in Figure 3.4. Instrumentation in the single sound treated room used is shown in Figure 3.3. Aided ACC waveforms were acquired, with the hearing aid which was programmed earlier placed on the test ear. The behind-the-ear hearing aid had to be placed in such a manner, that it caused no interference or artifacts as the inverting electrode was also placed on the test ear mastoid. Next, the speaker was positioned so that the hearing aid microphone, relative to the speaker was at an angle of 45°. A pre-stimulus electroencephalographic recording was first observed, and once the trace was found to stabilize the test recording was initialized. To obtain a baseline of the neurological activity unrelated to the stimulus in the waveform, a prestimulus recording was done for 100 msec. The waveforms were then analysed.

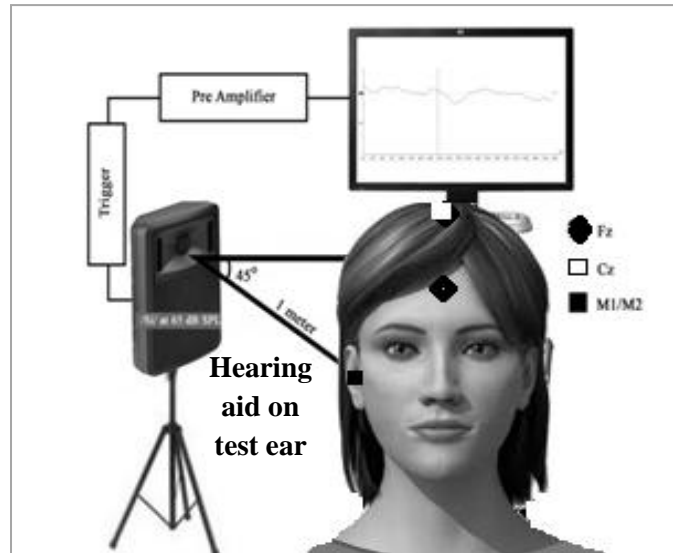


Figure 3.4: Set-up for aided ACC measurement – Electrode and speaker placement.

#### *Analysis of the waveforms*

The three-aided ACC waveforms (ACC obtained from 2, 4 and 8 channel) were compared based on their respective latency, amplitude and morphology. First, the waveforms were visually judged to be replicable after which peak latency and amplitude measures were made. The waveforms were analyzed by two experts in the electrophysiological measurement of auditory evoked potentials. Absolute amplitude and latency were chosen at the most negative or positive point or halfway point of a broad peak was considered.

Secondly, the morphology was rated based on three point rating scale. A score of 0 indicates poor morphology, 1 and 2 indicates average and good wave form morphology respectively. The data was tabulated for N1, P2, P1' and N1' and further statistical comparisons were done based on the objectives.

#### ***Phase 4: Obtaining the speech identification score in the clinical group***

For each individual of the clinical group, aided speech identification scores for high frequency word list adapted from the High Frequency Kannada Speech Identification Test (Mascarenhas, 2002) was utilized after a written consent was obtained, from the concerned author. Each channel of the hearing aid was evaluated with one of the four lists consisting of 25-words each; an unaided SIS score was also obtained.

This word list had been developed exclusively for high frequency sloping sensorineural hearing loss. In an acoustically shielded room, the voice of a native Kannada female speaker was recorded. A female voice was used as it has a higher fundamental frequency, and can better tap into perception of high frequency phonemes. Speaker effects were eliminated with the use of words which were recorded using Adobe Audition (version 1.5) with 16 bit resolution. To prevent familiarization three word list of the original test were utilized along with a fourth, that was developed through randomization of the words on the first list. Prior to each list, a 1000 Hz calibration tone was recorded in each word list, and was used to adjust the VU meter of the audiometer to zero. The words were presented at 65 dB SPL through a diagnostic 2-channel audiometer at 0° azimuth.

## Chapter 4

### RESULTS AND DISCUSSION

The objective of this study was to compare the aided ACC across different number of channels. The first objective was to compare the ACC in individuals with sloping hearing loss to those having hearing sensitivity within normal limits. A second portion of this study dealt with obtaining the speech identification score across two, four and eight channel hearing aids and comparing it with aided ACC peak. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) (version 16). The following statistical tests were used in the present study:

- Descriptive statistics which included the mean and standard deviations
- Mann-Whitney U test
- Friedman's Test
- Wilcoxon Signed Rank Test
- Spearman's Rho

#### *4.1 Comparison of unaided scores between the clinical and control groups*

The first objective was to compare the ACC of participants with sloping hearing loss (clinical group) with the participants having hearing sensitivity within normal limits (control group). With a descriptive analysis of the control and clinical group the mean and the standard deviation values could be obtained and is shown in Table 4.1. The N1-P2 complex was generated in response to perception of the fricative /s/ and the N1'-P2' complex in response to the vowel /i/. For statistical analysis these four peaks were considered.



The two groups could be compared based on the common variables they shared. To study if the outcome was statistically significant or not a Mann-Whitney U test was done to compare the two groups. The test revealed statistically significant differences between latency of P2 with  $|Z| = -02.405$ ,  $p < 0.05$  which was significantly shorter in the control group. The amplitude of N1' with  $|Z| = -0.377$ ,  $p < 0.001$ , was significantly larger in the control group. Although the mean latency of the N1' peak for the clinical group is shorter, it was not found to be significant on statistical analysis.

Table 4.1 Unaided parameters obtained for the control and clinical groups

<i>Parameter Analyzed</i>	<i>CONTROL</i>			<i>CLINICAL</i>		
	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>
N1 latency		127.05	15.34		-	
N1 amplitude	11	-01.35	.925			
P2 latency		179.74	26.59		201.07	11.68
P2 amplitude	20	1.610	.873	6	1.30	1.05
N1' latency		256.98	11.77		244.89	20.50
N1' amplitude	20	-2.84	1.20	8	-2.79	1.73
P2' latency		316.15	20.03		317.93	25.93
P2' amplitude	20	1.06	1.14	9	2.67	1.27
Waveform Morphology	20	1.55	0.51	11	1.40	.843

A noticeable feature depicted in Figure 4.1, which compares the mean latencies of the control and clinical group is the absence of the N1 peak in the clinical group participants. The N1 latency and amplitude values could not be considered for comparison as none of the clinical group participants had this peak present in their waveforms. As the N1 reflects detection of the stimuli at the cortical level (Martin,

Tremblay & Stapells, 2007), the absence of the same in the sloping cochlear hearing loss individuals may have arose due to three main reasons. The first reason is the inaudibility of the fricative portion of the /si/ stimulus. From a visual analysis of the /s/ portion shown in Figure 3.1, it is evident that the fricative portion has lower amplitude than the vowel portion. The first N1-P2 complex is generated in response to the fricative portion of the stimulus and the second N1'-P2' complex is generated in response to the transition from fricative to the vowel. The transition portion is audible to those with sloping cochlear hearing loss as evidenced by the presence of the N1'-P2' complex, but the initial /s/ portion is not audible.

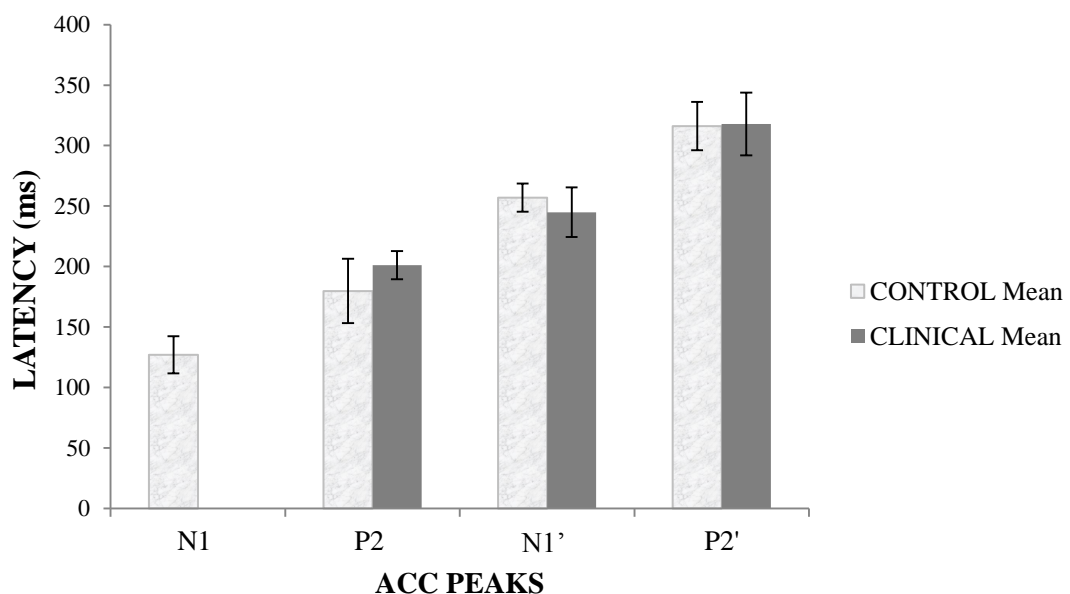


Figure 4.1: Mean latencies (in milliseconds, ms) for the control and clinical group. Error bars denote one standard error around the mean.

The P2 peak appeared earlier (early latency) in the control participants when compared to the clinical group, but, the P2 amplitude was similar for both the groups. This could be because the control group participants could perceive the initial

fricative /s/, but, due to reduction in audibility there was a delay in latency for the clinical group. There were no differences between the two groups in the amplitude of the P2 peak in the present study, and is comparable to results found by Wall, Dalebout, Davidson and Fox (1991). These results have also been confirmed in literature by Oates, Kurtzberg and Stapells in 2002, who found latency measures to be a more sensitive indicator of the effect of decreased audibility than a response strength measure such as amplitude. Similar findings have been reported by Polen (1984) who found significantly longer latencies for P2 in listeners with hearing loss compared to listeners with normal hearing. The author stated that this could be because this peak is sensitive to stimulus features and a reduction in audibility of these features causes the peak to diminish.

Although the frication portion was audible to the control group, nine of the twenty control group participants did not have the N1 peak in their waveform. Therefore, there could be features inherent to the stimulus as in how the vowel and consonant pair interact or the way the central auditory system responds to high frequency stimuli that may have caused the N1 peak to be absent. To explain further, the second reason considers the masking phenomenon; individuals with cochlear pathology are more prone to the effects of upward spread of masking due to wider auditory filters, which in turn results in reduced frequency resolution (Moore, 1998). The vowel portion of the stimulus could have sufficiently masked the fricative causing a response to arise only from the vowel portion. Low frequency portions of speech like vowels may mask higher frequency components like frication noise (Dillon, 2001). The /i/ portion being larger in amplitude could have masked the /s/ portion of the stimulus used in this study.

The third reason is the inherent lower amplitude of the cortical response when elicited from stimulus containing high frequency content such as / $\Sigma$ / and /s/ compared to /m/, /a/, /u/ and /i/ which had predominant low frequency content as documented by Agung et al., (2006). The N1 amplitude in their study was significantly smaller for the stimuli containing high frequency content. As responses evoked by a high frequency stimulus have inherent lower amplitude, an additional cochlear pathology could have resulted in an elimination of the response.

The vowel /i/ is capable of eliciting earlier latency. This is because the high front vowel such as /i/ can evoke CAEPs that have earlier latencies compared to low mid-back vowels /u/. Vowels with large F2-F1 differences such as /i/ (~2300 Hz) and /u/ (~1700 Hz) have larger areas of activation compared to a vowel with a small F2-F1 distance such as /a/ according to Yetkin et al., (2004).

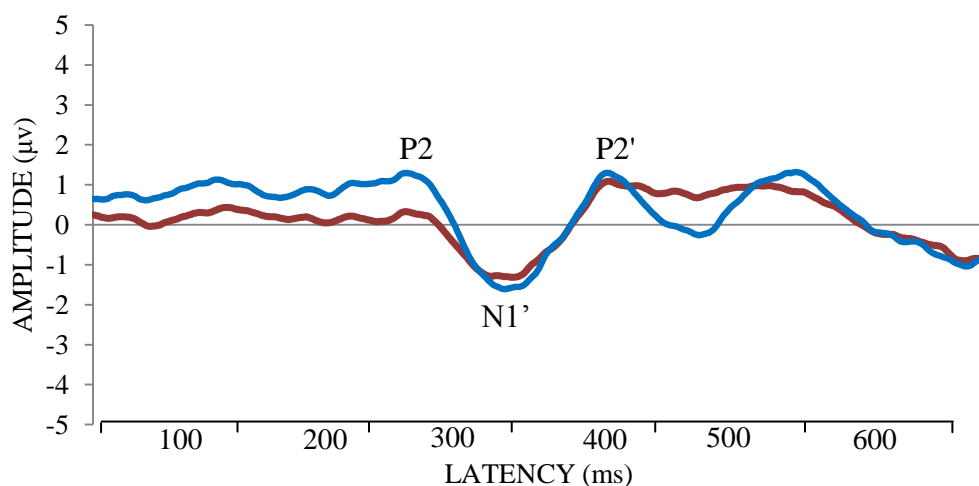


Figure 4.2: Grand mean waveform acquired from the participants with hearing sensitivity within normal limits (Blue) & sloping sensorineural hearing loss (Red).

In listeners with normal hearing sensitivity, the growth of loudness at mid and high level follow a compressive power function of intensity (Stevens, 1955). Whereas, in

cochlear hearing loss, a loss of compression occurs and is consistent with the presence of recruitment. According to Buus and Florentine (2001), this growth of loudness occurs near threshold; and they stated that on average for every 16 dB of hearing loss, the loudness near threshold doubled. The finding of a larger P2' amplitude in the clinical group can be explained based on the differences in the growth of loudness between the control and clinical group. The increase in the amplitude of P2 in the control group may be attributed to recruitment which could have occurred as a result of cochlear hearing loss. The grand mean waveforms of the control and the clinical group ACC has been depicted in Figure 4.2. The amplitude differences have been shown in Figure 4.3.

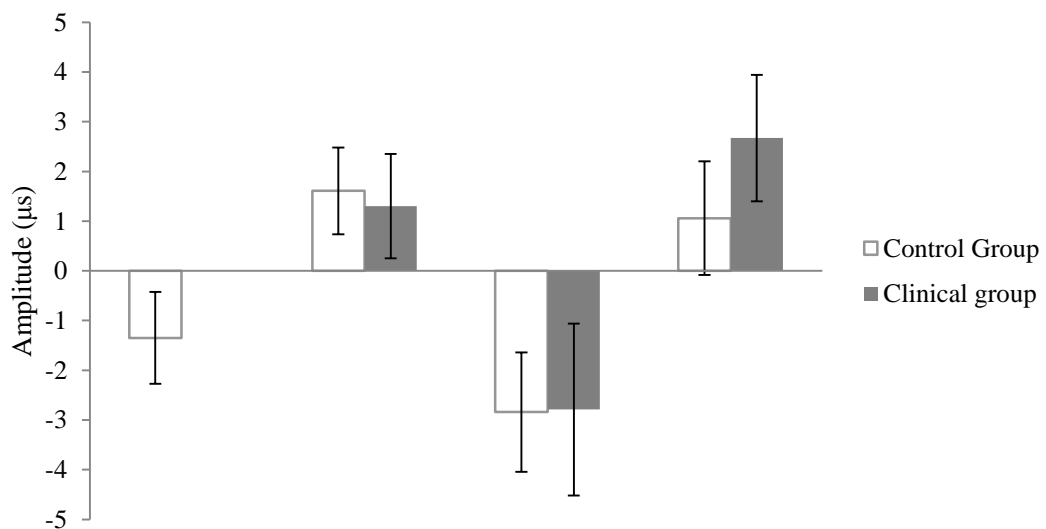


Figure 4.3: Mean amplitudes (in microvolts,  $\mu\text{V}$ ) for the control and clinical group participants. Error bars denote one standard error around the mean.

The second objective was to compare the unaided and aided ACC in individuals with sloping cochlear hearing loss. Comparison between the three aided conditions and the unaided condition was done. The aided conditions considered three hearing aids comprising two, four and eight channels.

The results of the second objective are discussed under two sub headings.

1. Comparison of the unaided and aided conditions within the clinical group
2. Effect of hearing aid channels

#### *4.2.1 Comparison of the unaided and aided conditions within the clinical group*

Wilcoxon Signed Ranks Test was used to make these pair-wise comparisons, and the result did not reveal any significant differences between the aided (two, four and eight channels) and unaided conditions ( $p > 0.05$ ). Therefore, for individuals with sloping cochlear hearing loss, the performance on the ACC did not differ with amplification or without it. While studying the neural representation of amplified speech sounds, similar findings were reported by Tremblay et al., (2006). An increase in hearing aid gain did not result in an amplitude increase or latency decrease as compared with unaided ACCs at the same input level. They found no significant differences between aided and unaided ACCs in response to the /si/ stimulus. Reasons suggested for the findings have been hearing aid compression effects, which is also applicable in this study as the compression circuit was active during ACC acquisition. To explain further, according to Easwar et al., (2012) inter stimulus interval between one and two seconds causes an abrupt increase in the input level each time the stimulus is presented. This causes compression to act each time, and may result in overshoot for every stimulus presented. This overshoot occurs when the stimulus is above compression threshold, because the hearing aid requires time to stabilize the gain when there is a rapid change in the input level.

Another reason could be that the amount of response change (improvements) seen in the ACC demonstrated considerable variability across subjects. This could

have lead to insignificant findings in the current study. One of the participants in the clinical group had no visible ACC in the unaided condition or in the aided although the stimulus was audible. The finding of this subject was not included for statistical analysis.

#### 4.2.2 Effect of hearing aid channels

Speech cues like transitions with dynamic frequency changes are not perceived well even when audibility is provided. Zeng and Turner (1990) stated the loss of transition cue perception which occurs in sensorineural hearing loss cannot be compensated by hearing aids. In contrast, the hearing aids in the present study could reflect the transition portion as evidenced by the prominent ACC N1'- P2' peak.

Each peak obtained in the ACC, was analyzed for latency, amplitude and morphology. The descriptive statistics has been shown below, in Table 4.2.

Table 4.2: Latency, amplitude and morphology (mean and standard deviations for two, four and eight channel hearing aids)

<i>Parameter Analyzed</i>	<i>8-Channel</i>			<i>4-Channel</i>			<i>2-Channel</i>		
	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>
P2 Lat		201.72	19.00		209.04	13.24		190.47	17.21
P2 Amp	8	1.00	0.67	8	1.49	0.93	6	0.84	0.69
N1' Lat		251.48	14.62		254.63	19.03		251.10	17.54
N1' Amp	10	-2.64	1.49	10	-2.61	1.10	9	-2.78	1.50
P2' Lat		314.00	20.58		317.33	24.20		316.92	27.80
P2' Amp	10	2.30	1.37	10	1.97	1.44	9	2.22	1.29
Morph	11	1.45	0.69	11	1.28	0.79	11	0.82	0.75

Note: Lat = Latency (ms), Amp = Amplitude ( $\mu$ v), Morph = Morphology

To learn the effect of hearing aid channels on the central auditory system, the ACC latency, amplitude and morphology across 2, 4 and 8 channels were compared. The Friedman's test was used and the results obtained have been discussed below.

The following variables were considered for each channel:

- i. P2 - Latency and amplitude
- ii. N1' - Latency and amplitude
- iii. P2' - Latency and amplitude
- iv. Overall waveform morphology

No significant differences were found between the two, four and eight channels in terms of latency and amplitude; this means that the three hearing aids had equivalent performance. The same has been shown in Figure 4.4 and Figure 4.5. It has been reported that the vowel-consonant differences will be reduced due to spectral contrast reduction caused by a higher number of channels (Plomp, 1988). Vowels are more susceptible to this effect than consonants. Consonants in the initial position are more susceptible than those in the final position (Boothroyd, Mulhearn, Gong & Ostroff, 1996). However, the results of this study contradicts the findings of Plomp (1988) and Bor et al., (2008), as the two, four and eight channel hearing aids were not found to have significant differences in latency and amplitude. The effects of spectral contrast reduction which would have reduced the difference between the vowel and the fricative would have otherwise led to increased latency, reduced amplitude and poor morphology for the eight channel hearing aid.



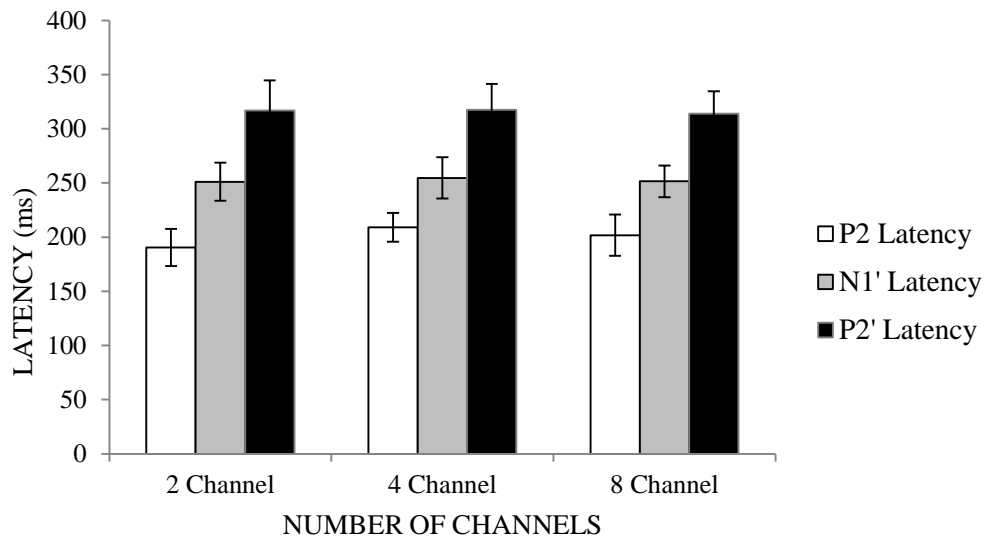


Figure 4.4 Mean latencies for the three aided conditions. Error bars denote one standard error around the mean.

Among the four parameters analyzed, the waveform morphology was found to be statistically significant across the three aided conditions with  $\chi^2 (11) = 7.00$ ,  $p < 0.05$ . Next a Wilcoxon Signed Rank test was conducted and the results showed that morphology obtained with the four and eight channel devices were equivalent. However, between the eight and two channel hearing aids there was a significant difference in morphology,  $Z = -0.111$ ,  $p < 0.05$ . The mean of the ranks in favor of a 2 channel hearing aid were 4.00, while the mean of the ranks in favor of eight channels were 4.57. From the descriptive statistics, mean value of the 8 channel hearing aid was higher than that of the 2 channel hearing aid. Therefore, it can be concluded that an 8 channel hearing aid results in better waveform morphology relative to the 2 channel hearing aid, Figure 4.6 compares the grand mean waveforms of the three aided conditions. Since poor waveform morphology may not have a physiological basis, and can be affected by a number of factors from sweep to sweep

such as muscular artifacts, eye blink and state of arousal, it may not be consistent with changes in signal processing.

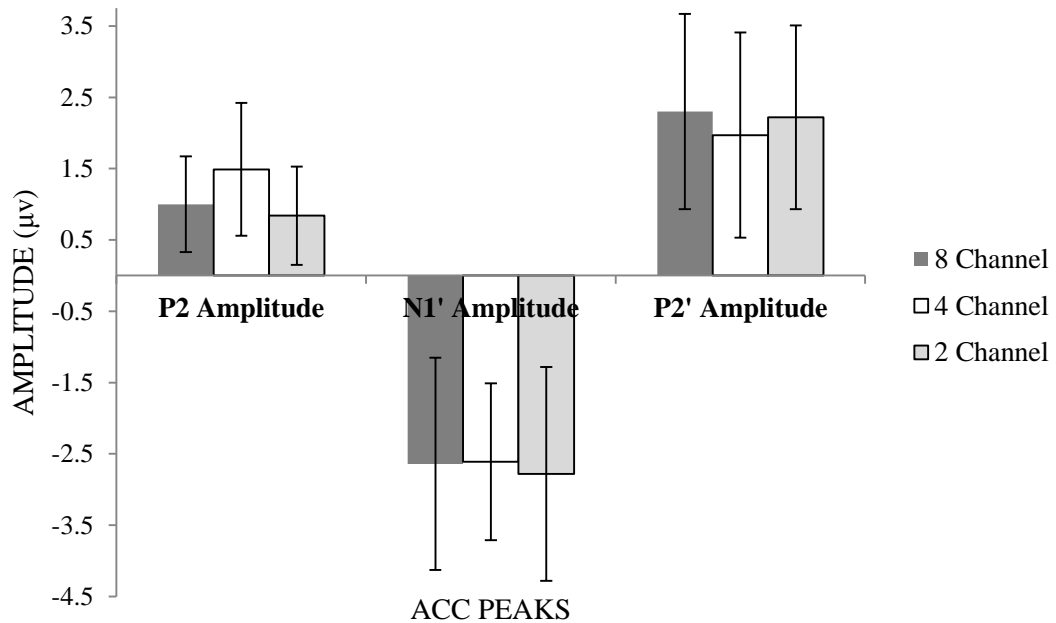


Figure 4.5 Mean amplitudes for the aided condition of two, four and eight channels. Error bars denote one standard error around the mean.

Multichannel hearing aids do not have rectangular analysis bands and compression channels, but instead the actual analysis bands and compression channels in hearing aids are non-rectangular, and may have very gradual filter slopes. The more gradual the filter slope of the channels, it will result in correlated compression across channels. Therefore, a hearing aid with higher number of channels may be functioning similar to a hearing aid with lower number of channels. This could be a probable reason for why no significant changes were noted across two, four and eight channels.

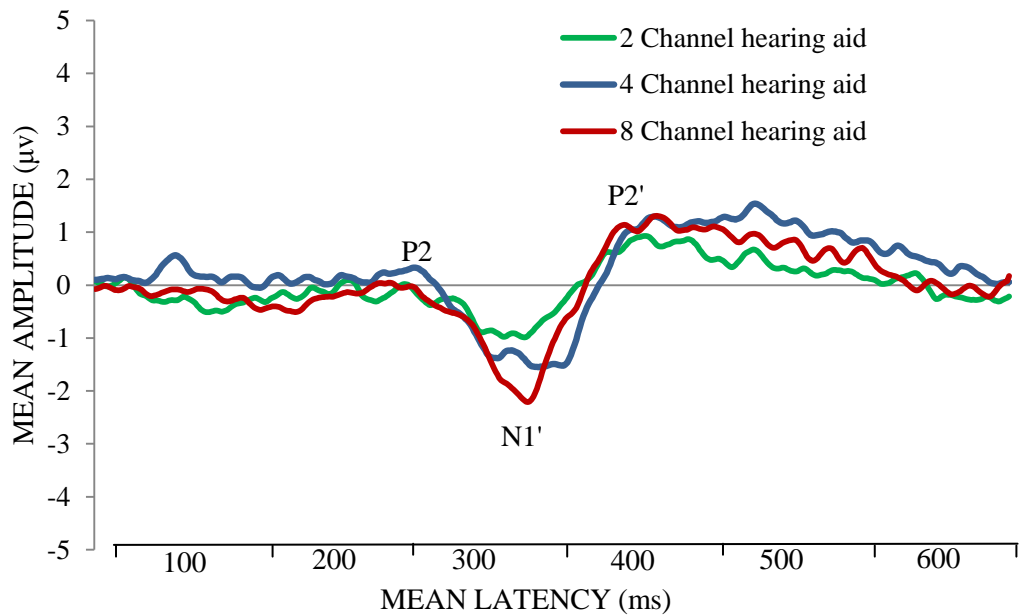


Figure 4.6 Grand mean waveform of the ACC obtained for the aided condition using the two, four and eight channel device.

Another reason for no significant differences across channels could be due to the compression circuit of the hearing aid. According to Korczak, Kurtzberg and Stapells (2005), when the compression circuit is active, a ceiling effect occurs for aided responses at higher intensities. Therefore, subtle variations in signal processing brought about by an increase or decrease in the number of channels, may not be accurately reflected if other processing schemes such as compression are active.

#### ***4.3 Correlation of speech identification scores and ACC parameters***

Non-parametric correlations were administered to achieve the fourth objective which was to compare the SIS and ACC across different number of hearing aid channels. Spearman's correlation was done, considering the peaks in the ACC waveform namely; P2, N1' and P2' which was correlated with the speech identification score obtained using two, four and eight channel hearing aids. The mean

scores obtained for the two, four and eight channels were 83, 84 and 81% with standard deviations of 8.48, 10.90 and 9.78 respectively. The difference between the unaided and the aided condition approached significance at  $\chi^2(8) = 7.541$ ,  $p = 0.057$ . The same has been depicted in Figure 4.7.

A positive correlation between the amplitude of P2' with the four channel device and the speech identification scores ( $\rho = 0.822$ ,  $p < 0.05$ ). This could be attributed to the inherent redundancy of words compared to a syllable. Word discrimination improves with increase in word length (Black, 1952). The speech identification material used in the present study consisted of monosyllabic words and the ACC stimulus was a single syllable /si/. The difference in redundancy between the two stimuli could have resulted in a lower correlation between the two. The probable reason may be attributed to the subjective differences in perception through hearing aids with varying number of channels. This needs to be clarified by further research in the same area.

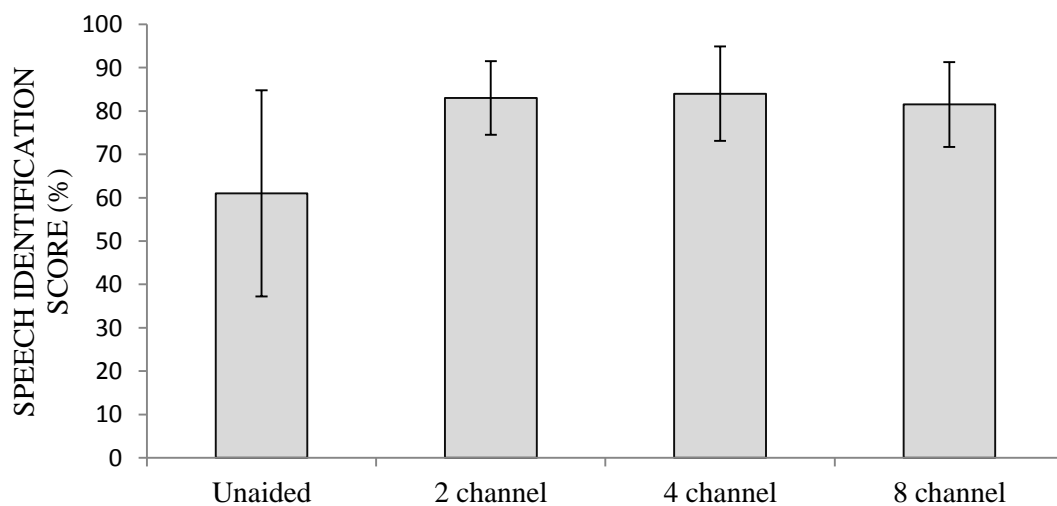


Figure 4.7 Mean speech identification scores obtained for different conditions.

Error bars denote one standard error around the mean.

In contrast to the present study, behavioral measures of discrimination and its correlation to CAEP have been assessed by Korczak et al., (2005). This study has found a correlation between the behavioral or discrimination score and the electrophysiological testing.

A reason for no significant changes seen between the number of channels and speech identification scores could be that an increase in the number of channels negatively affects the perception of diphthongs and vowels (Bor et al., 2008) and not to a large extent the perception of consonants according to Boothroyd et al., 1996. High Frequency Kannada Speech Identification Test (Mascarenhas, 2002) was used to assess the speech identification score. The high frequency content in the wordlist was contributed by consonants which were semivowels (/j/, /r/, /l/), stops (/t/, /p/, /k/), fricatives (s, /f/, /Σ/) and the affricate /tΣ/. Therefore, only minor variations in the scores were seen across different number of channels.

### ***Summary of Results***

*1) Comparison between individuals with normal hearing sensitivity to those with sloping cochlear hearing loss:* The group waveform characteristics which included the latency, amplitude and morphology differed between the two groups. The P2' latency was found to be significantly shorter in individuals with normal hearing sensitivity. Additionally, the N1' amplitude was significantly larger in individuals with hearing sensitivity within normal limits.

*1) Comparison of the unaided and aided conditions:* There were no significant differences noted between the aided and the unaided conditions, in terms of latency, amplitude and morphology. In other words, there were no major differences noted in the ACC with the hearing aid or without it.

2) *Comparison between the two, four and eight channel hearing aids:* The three hearing aids were programmed based on the probe microphone measurement through which the gain of the hearing aid was matched to the target curve. When compared based on the latency and amplitude, the hearing aids had equivalent performance. The morphology of the eight channel and four channel devices were equivalent. But, when compared to the two channel device, the morphology of the eight channel device was found to be significantly better.

3) *Correlation of speech identification scores and ACC parameters:* A positive correlation between the amplitude of the P2' and the speech identification score was found only with four channel device. Other peaks did show any significant correlation.

## Chapter 5

### SUMMARY AND CONCLUSION

The acoustic change complex is a slow latency potential which can be elicited with a stimuli containing ongoing changes in terms of amplitude, spectral envelope or periodicity (Martin and Boothroyd, 2000). /si/ has variations in all the above mentioned and hence, was chosen for the study which aimed to find the effect of varying the number of channels on ACC in individuals with sloping hearing loss.

The effect of the number of channels on speech perception was studied in two ways, electrophysiologically through the ACC and behaviorally through speech identification scores. Participants with sloping hearing loss were included as the benefit across channels can be better observed with this configuration as compared to a flat cochlear hearing loss. The effect of sloping sensori-neural hearing loss was found out by comparing it with the ACC obtained in the control group, which was the first objective of the study.

The two groups were compared based on their latency, amplitude and morphology. The P2 latency (in response to the /s/) was significantly delayed in the clinical group as compared to the control group, but, with no significant differences in amplitude which confirms to literature that latency measures are more sensitive indicators of the effects of decreased audibility than are response strength measures like amplitude. However, the N1'- P2' complex (in response to /i/) showed no significant differences in latency, amplitude or morphology. This portion of the stimuli could easily be perceived by both groups and hence these findings were obtained. The P2 amplitude was larger in the clinical group as compared to the control

group; this could have been caused due to abnormal loudness growth also known as recruitment in cochlear pathology.

The second objective was to find the differences between the aided and the unaided response within the clinical group, significant differences were not noted between the two conditions. A possible reason for this result could be due to the effect of recruitment in the unaided condition (which increased the amplitude) and the effect of compression by the hearing aid in the aided condition (which decreased amplitude). Therefore, in the aided and unaided conditions, an equivalent performance was noted, which needs confirmation through further research by varying the input levels to the amplification device.

Next, within the aided condition, the difference across two, four and eight channels were obtained. The latency and amplitude comparison did not reveal any significant difference. Differences can be expected in terms of spectral contrast reduction which would in turn reduce speech perception and/or better frequency shaping which should improve speech perception when the number of channels is higher. However, the results across the channels were found to be equivalent in contrast to previous findings.

No significant differences between the two, four and eight channel hearing aid could have been because of the gradual slope between the channel filters, which will result in correlated output from the hearing aid. The three hearing aids will function similarly if this is the case. The active compression circuit in the hearing aids could have caused a ceiling effect to occur for aided responses. Therefore, subtle variations in signal processing brought about by an increase or decrease in the number of



channels, may not be accurately reflected in the presence of other hearing aids processing strategies like compression.

The last objective was to correlate the speech identification score with the ACC. A positive correlation between the amplitude of the P2' and the speech identification score was found for the four channel device. The speech identification material used in the study consisted of polysyllabic words and the ACC stimulus comprised of a single syllable /si/. The difference in redundancy between the two stimuli could have resulted in a lower correlation because of which the eight and the two channel hearing aids did not have any significant correlation. The positive correlation between the amplitude of P2' and the SIS obtained via 4 device needs to be probed further.

### *Conclusions*

When compared to individuals with hearing sensitivity within normal limits, those with cochlear hearing loss have delayed latencies on the ACC; amplitudes were found to be similar between the groups. For sloping cochlear hearing loss, with or without amplification the responses were similar. When the two, four and eight channel hearing aids were evaluated electrophysiologically through the ACC, the performance was equivalent. When the electrophysiological findings were correlated with the speech identification scores for each aid, ACC obtained with the four channel device was found to correlate with the SIS; but, there was no correlation found between the two and eight channel hearing aids.

### ***Clinical Implications***

1. As individuals with sloping cochlear hearing loss have difficulty in perception of fricatives, results of this study can help understand the neural representation of fricatives at the cortical level, and how amplification will affect this response.
2. The comparison between individuals with hearing sensitivity within normal limits and those with sloping sensorineural hearing loss, can give an insight into the delay in the information transferred to the cortical structures as a result of cochlear hearing loss.
3. The cortical responses like N1 and P2 are sensitive to stimulus related changes. Hence, the changes in the stimulus which will result in earlier latencies, increased amplitudes and better morphology will be activating more neurons at the cortical level. The amplification device can incorporate such output modifications, thereby improving the perception of speech. The results obtained will also assist Audiologists in devising strategies to optimize the amplification device for better speech perception.

### ***Future Research***

1. It would be valuable to investigate the correlation between the behavioral and electrophysiological testing with stimuli that have similar in terms of external redundancy. This will aid in understanding how different hearing instruments work, and its effect on speech perception of high frequency sounds which significantly contribute to speech intelligibility.
2. To better understand the effect of the number of channels, this study needs to be done on a larger group of individuals in order for significant results to be clearly illustrated. This is because of heterogeneity within individuals with cochlear hearing loss.

3. As the slope of the hearing loss increases, the benefit of multichannel hearing aids can be better understood as there will be greater variability in frequency shaping and compression with increase in number of channels. Greater number of channels will bring out the effect of dividing a signal through separate amplifiers, with facility to alter parameters for independent channels. Therefore, the benefit or shortcomings in the performance due to varying number of channels can be well understood if the same study is replicated in individuals with steeply sloping sensori-neural hearing loss.

## REFERENCES

- Adler, G., & Adler, J. (1989). Influence of stimulus intensity on AEP components in the 80- to 200-millisecond latency range. *Audiology*, 28, 316–324.
- Agung, K., Purdy, S. C., McMahon, C. M., & Newall, P. (2006). The use of cortical auditory evoked potentials to evaluate neural encoding of speech sounds in adults. *Journal of the American Academy of Audiology*, 17(8), 559-572.
- Apeksha, K., & Devi, N. (2010). Effect of sensorineural hearing loss on speech evoked aided auditory late latency response. *Journal of All India Institute of Speech and Hearing*, 29(2).
- Billings, C. J., Tremblay, K. L., & Miller, C. W. (2011). Aided cortical auditory evoked potentials in response to changes in hearing aid gain. *International Journal of Audiology*, 50, 459-467.
- Billings, C. J., Tremblay, K. L., Souza, P. E., & Binns, M. A. (2007). Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. *Audiology and Neurootology*, 12(4), 234-246.
- Black, J. W. (1952). Accompaniments of word intelligibility. *Journal of Speech and Hearing Disorders*, 17, 409-418.
- Boothroyd, A., & Medwetsky, L. (1992). Spectral Distribution of /s/ and the frequency response of hearing aids. *Ear and Hearing*, 13(3), 150-157.
- Boothroyd, A., Mulhearn, B., Gong, J., & Ostroff, J. (1996). Effects of spectral smearing on phoneme and word recognition. *Journal of Acoustical Society of America*, 100, 1807-1818.
- Bor, S., Souza, P., & Wright, R. (2008). Multichannel compression: Effects of reduced spectral contrast on vowel identification. *Journal of Speech Language and Hearing Research*, 51, 1315-27.

- Buus, S., Florentine, M. (2001). Growth of loudness in listeners with cochlear hearing losses: Recruitment reconsidered. *Journal of the Association of Research in Otolaryngology*, 3, 120-139.
- Carhart, R., & Jerger, J. J. (1959). Preferred method for clinical determination of pure-tone thresholds. *Journal of Speech and Hearing Disorders*, 24, 330-345.
- Danaher, E. M., Osberger, M. J., & Pickett, J. M. (1973). Discrimination of formant frequency transitions in synthetic vowels. *Journal of Speech and Hearing Research*, 16, 439-451.
- Demeester, K., van Wieringen, A., Hendrickx, J., Franssen, E., van Laer, L., Van Camp, G., & Van de Heyning, P. (2009). Audiometric shape and Presbycusis. *International Journal of Audiology*, 48, 222-2332.
- Dillon, H. (2001). Hearing aids. Turrumurra, Australia: Boomerang Press.
- Dimitrijevic, A., Michalewski, H. J., Zeng, F. G., Pratt, H., & Starr, A. (2008). Frequency changes in a continuous tone: Auditory cortical potentials. *Clinical Neurophysiology* 119(9):2111-24.
- Easwar, E., Glista, D., Purcell, D. W., & Scollie, D. S. (2012). Hearing aid processing changes tone burst onset: Effect on cortical auditory evoked potentials in individuals with normal audiometric thresholds. *American Journal of Audiology* (In Press).
- Edwards, B. (2004). Hearing aids and hearing impairment. In Greenberg, S., Ainsworth, W., Popper, A. N., Fay, R. R. *Speech Processing in the Auditory System*. (pp. 339 – 421) New York: Springer-Verlag.
- Florentine, M., Fastl, H., & Buus, S. (1988). Temporal integration in normal hearing, cochlear impairment, and impairment simulated by masking. *Journal of the Acoustical Society of America*, 84, 195-203.
- Hardcastle, W. J. & Hewlett, N. (1999). *Coarticulation: Theory, Data and Techniques in Speech Production*. Cambridge: Cambridge University Press.

- Harris, K. C., Mills, J. H., Dubno, J. R. (2007). Electrophysiologic correlates of intensity discrimination in cortical evoked potentials of younger and older adults. *Hearing Research*, 228, (1-2) 58-68.
- Jyoti (2010). *Effect of number of channels of hearing aids on speech perception in different degrees of sloping hearing loss cases.* Unpublished Masters's Dissertation. University of Mysore, Mysore.
- Korczak, P. A., Kurtzberg, D., & Stapells, D. R. (2005). Effects of sensorineural hearing loss and personal hearing aids on cortical event-related potential and behavioral measures of speech-sound processing. *Ear and Hearing*, 26(2), 165-185.
- Lightfoot, G., & Kennedy, V. (2006). Cortical electric response audiometry hearing threshold estimation: accuracy, speed, and the effects of stimulus presentation features. *Ear and Hearing*, 27(5), 445-456.
- Lloyd, L. L., & Kaplan, H. (1978). *Audiometric interpretation: A manual of basic audiometry*, Baltimore: University Park Press.
- Margolis, R., & Saly, G. (2008). Distribution of hearing loss characteristics in a clinical population. *Ear and Hearing*, 29 (4), 524-532.
- Martin B. A., & Boothroyd, A. (1999). Cortical, auditory, event-related potentials in response to periodic and aperiodic stimuli with the same spectral envelope. *Ear and Hearing*. 20, 33-44.
- Martin, B. A., & Boothroyd, A. (2000). Cortical, auditory, evoked potentials in response to changes of spectrum and intensity. *Journal of the Acoustical Society of America*, 107, 2155-2161.
- Martin, B. A., Tremblay, K. L., & Stapells, D. R. (2007). Principles and applications of cortical auditory evoked potentials. In Burkard, R. F., Don, M. & Eggermont, J. J. (Eds.) *Auditory Evoked Potentials. Basic Principles and Clinical Application* (pp. 482-507). Philadelphia: Lippincott, Williams & Wilkins.

- Mascarenhas, K. (2002). *A high frequency Kannada speech identification test (HF-KSIT)*. Unpublished Masters's Dissertation. University of Mysore, Mysore.
- Moore, B. C. J., (1998) Cochlear hearing loss. London: Whurr Publishers Ltd.
- Naatanen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology*, 24, 375-425.
- Oates, P. A., Kurtzberg, D., & Stapells, D. R. (2002). Effects of sensorineural hearing loss on cortical event-related potential and behavioral measures of speech-sound processing. *Ear and Hearing*, 23, 399-415.
- Ostroff J. M., Martin B. A., & Boothroyd A. (1998). Cortical evoked responses to spectral change within a syllable. *Ear and Hearing* 19, 290-297.
- Owens, E., Benedict, M., & Schubert, E. D. (1972). Consonant phonemic errors associated with pure-tone configurations and certain kinds of hearing impairments. *Journal of Speech and Hearing Research*, 15, 308-322.
- Pittman, A. L., & Stelmachowicz, P. G. (2003). Hearing loss in children and adults: audiometric configuration, asymmetry, and progression. *Ear and Hearing*, 24, 198-205.
- Polen, S. B. (1984). Auditory event related potentials. *Seminars in Hearing*, 5, 127-141.
- Plomp, R. (1988). The negative effect of amplitude compression in multichannel hearing aids in the light of the modulation transfer function. *Journal of the Acoustical Society of America*, 83, 2322-2327.
- Purdy, S. C., Kelly, A. S., & Thorne, P. R. (2001). Auditory evoked potentials as measures of plasticity in humans. *Audiology and Neurootology*, 6(4), 211-215.
- Roeser R. J., & Clark, J. L. (2007). Pure tone tests. In Roeser R. J., Hosford-Dunn, H., Valente, M. (2 Ed.), *Audiology Diagnosis* (pp. 238-260). New York: Thieme Medical Publishers, Inc.

- Rubina (2008). *Effects of Degree of Loss and Age on Speech Identification with Multichannel Hearing Aids*. Unpublished Masters's Dissertation. University of Mysore, Mysore.
- Sharma, A., & Dorman, M.F. (1999). Cortical auditory evoked potential correlates of categorical perception of voice-onset time. *Journal of the Acoustical Society of America*, 106(2), 1078-1083.
- Sher, A. E., & Owens, E. (1974). Consonant confusions associated with hearing loss above 2000 Hz. *Journal of Speech and Hearing Research*, 17, 669-681.
- Souza, P.E., & Boike, K.T. (2006). Combining temporal cues across channels: Effects of age and hearing loss. *Journal of Speech, Language and Hearing Research*, 49, 138-49.
- Stelmachowicz, P. G., Kopun, J., Mace, A., Lewis, D. E., & Nittrouer, S. (1995). The perception of amplified speech by listeners with hearing loss: Acoustic correlates. *Journal of the Acoustical Society of America*, 98, 1388-1399.
- Stevens, S. S. (1955). The measurement of loudness. *Journal of Acoustical Society of America*, 27, 815-829.
- Tremblay K. L., Billings C. J., Friesen L. M., & Souza P. E. (2006). Neural representation of amplified speech sounds. *Ear and Hearing*, 27, 93-103.
- Tremblay, K. L., Friesen L., Martin B. A., & Wright R. (2003). Test-retest reliability of cortical evoked potentials using naturally produced speech sounds. *Ear and Hearing*, 24; 225-232.
- Tremblay, K. L., Kalstein, L., Billings, C. J., & Souza, P. E. (2006b). The neural representation of consonant vowel transitions in adults who wear hearing aids. *Trends in Amplification*, 10, 155-162.
- Turner, C. W., Souza, P. E., & Forget, L. N. (1995). Use of temporal envelope cues in speech recognition by normal and hearing-impaired listeners. *Journal of the Acoustical Society of America*, 97, 2568-2576.



- Van Dun B., Carter L., Dillon H. (2011). The relationship between cortical auditory evoked potential (CAEP) detection and audibility assessed behaviorally in infants with sensorineural hearing loss.
- Wall, L., Dalebout, S. D., Davidson, S. A., & Fox, R. A. (1991). Effect of hearing impairment on event-related potentials for tone and speech distinctions. *Folia Phoniatrica*, 43, 265–274.
- Wunderlich, J. L., & Cone-Wesson, B. K. (2006). Maturation of CAEP in infants and children: A review. *Hearing Research*, 212(1-2), 212-223.
- Yetkin, F. Z., Roland, P. S., Christensen, W. F., & Purdy, P. D. (2004). Silent fMRI of tonotopicity and stimulus intensity coding in human primary auditory cortex, *Laryngoscope*, 114, 512-518.
- Yund, E. W., & Buckles, K. M. (1995). Multichannel compression hearing aids: Effect of number of channels on speech discrimination in noise. *Journal of the Acoustical Society of America*, 97, 1206-1223.
- Zeng, F. G., & Turner, C. W. (1990). Recognition of voiceless fricatives by normal and hearing-impaired subjects. *Journal of Speech and Hearing Research*, 33, 440-449.