Representation of Amplified Speech at the Brainstem and Cortical levels of the Auditory Pathway in Individuals with Sensorineural Hearing Loss

A DOCTORAL THESIS

Submitted to the University of Mysore,
for the award of degree of
Doctor of Philosophy (Ph.D.) in Audiology.

By

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DECLARATION

I declare that this thesis entitled **'Representation of Amplified Speech at the Brainstem and Cortical levels of the Auditory Pathway in Individuals with Sensorineural Hearing Loss'**, being submitted herewith for the award of the degree of Doctor of Philosophy in the field of Audiology to the University of Mysore, Mysore, is the result of work carried out by me at the All India Institute of Speech and Hearing, Mysore, under the guidance of Dr. Manjula P, Professor of Audiology, All India Institute of Speech and Hearing, Mysore. I further declare that the results of this work have not been previously submitted for the award of any degree.

Place: Mysore Hemanth, N.

Date:

CERTIFICATE

This is to certify that the thesis entitled **'Representation of Amplified Speech at the Brainstem and Cortical levels of the Auditory Pathway in Individuals with Sensorineural Hearing Loss'** submitted by Mr. Hemanth,N. for the degree of Doctor of Philosophy in Audiology to the University of Mysore was carried out at the All India Institute of Speech and Hearing, Mysore under my guidance. I further declare that the results of this work have not been previously submitted for any degree.

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ABSTRACT

Statement of problem: One among the rehabilitative options to alleviate the hearing problem is the hearing aid. Though having same type, degree and configuration of hearing loss, only some individuals benefit from a hearing aid, while others reject the hearing aids. To know the extent of changes provided from amplification device in clinical group, the aided response was studied at different levels of the auditory pathway.

Method*:* Hundred participants, 60 with sensorineural hearing impairment and 40 with normal hearing in the age range from 15 to 65 years, were included. The participants from each group were divided into four sub-groups, based on age. Each participant of clinical group was classified into good and poor hearing aid performers. The study was conducted in three phases. Phase 1 was utilized for participant selection criteria. In Phase 2, the probe tube microphone measurement was carried out to record the hearing aid output at the ear canal of participants from clinical group. In Phase 3, electrophysiological responses were obtained from auditory brainstem and cortical levels at 65 dB SPL for /ɖa/ and /si/ speech stimulus from the participants of clinical (unaided and aided) and normal hearing groups.

Results: The hearing aid preserved the spectral parameters at the ear canal. However, there was a minimal alteration in temporal parameter. Each response obtained, especially from older adults, at different levels of auditory pathway was relatively less precisely represented in clinical group than in the normal hearing group. Further, the

responses at each level were compared between good and poor hearing aid performers. The responses at each level were relatively well represented in good hearing aid performers than in poor hearing aid performers.

Conclusion: Though the hearing aid alleviates the hearing problem to an extent, minimal alteration after being processed through the hearing aid and altered physiological mechanism due to hearing loss were noted in the representation of the speech stimuli at each level of auditory pathway in the clinical group. Further, the speech stimuli were represented better at brainstem and cortical levels, in good hearing aid performers.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Acknowledgement

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CHAPTER - 1 INTRODUCTION

The hearing aids amplify the syllables. They have been successful at restoring hearing abilities for people with hearing impairment to some extent. However, even with some of the most advanced technologies, patients still have a degree of difficulty understanding speech. This is because, in addition to providing amplification, the hearing aids modify the inherent spectral and temporal aspects of speech signals (Eggermont, 1995).

Stelmachowicz, Kopun, Mace, Lewis, and Nittrouer (1995) have reported that the high frequency roll-off of the hearing aid response limits the high frequency consonant cues relative to the unprocessed signals. The low frequency roll-off essentially removes the first formant of certain vowels. According to Stelmachowicz, Mace, Kopun, and Carney (1993), a hearing aid can also blur the boundary between the aperiodic noise of consonants and the periodic onset of voiced vowels, making these transitions less distinct.

In older adults, Tremblay, Piskosz, and Souza (2002) have reported impaired temporal coding, due to the reduction of simultaneous discharge of neurons. This asynchronous firing in the auditory system of older adults have significant problem in encoding subtle acoustic cues. Coughlin, Kewley-port, and Humes (1998) investigated the relationship between identification and discrimination of vowels in older adults. They reported inability to extract temporal cues and indirectly suggested difficult to process vowel-format discrimination. In a similar line of investigation on temporal processing in older adults, Price and Simon (1984) have noticed deficits in discrimination of voice

onset time. This leads to difficulty in understanding speech, though this has no relationship with elevated thresholds (Willott, 1996). Thus, acoustic cues from impaired physiology at peripheral system relay the altered input to central auditory system through the auditory pathway (Chisolm, Willott, & Lister, 2003).

Altogether, the amplified speech signal alters the neural response patterns in the central auditory system by factors such as damaged mechanism in the peripheral auditory system, aging, complex interaction of aging and hearing loss, and alteration of inherent cues after processing by a hearing aid. Considering these and many more variables, audiologists are aware of the range of performance variability among individuals using hearing aids. Kochkin (2010) reported that performance variability could be because of factors anywhere from the microphone of the hearing aid till the processing of the signals in the brain. Much research is being undertaken focusing on hearing aid technology and the way the device might be modified to ensure better use. In spite of similar degree, type of hearing loss, and audiogram configuration, two individuals may not benefit from a hearing aid to a same extent. The reason for difference in improvement in speech understanding with similar hearing device by two individuals with a similar type, degree, and configuration of hearing loss is still unclear. Thus, it would be interesting to note how the processing of syllables at different levels of auditory system in a hearing aid user differs from that of a person with normal hearing.

Yund and Buckles (1995) reported wide variation in the degree of benefit that a person receives from a hearing aid. According to Kochkin (2010), 62.3 % are dissatisfied

with hearing aids of which 25.3 % reject their hearing aid because of background noise. Though there are various techniques to determine the extent of satisfaction with hearing aid viz., speech in noise test (SPIN), hearing in noise test (HINT), and connected speech test (CST), there is no strong relationship between the score obtained on speech intelligibility in noise of a person and his/her real world benefit / satisfaction with hearing aids.

Nabelek, Tucker, and Letowski (1991) developed a test procedure to determine the acceptable noise level (ANL) while listening to speech, as some individuals are unwilling to wear the hearing aid due to an inability to withstand the background noise. The ANL is calculated by subtracting the background noise level (BNL) from the most comfortable level (MCL). The ANL measure predicted the successful hearing aid use with 85% accuracy. However, the ANLs are not affected by age, hearing sensitivity (Branstrom, Lantz, Nielsen, & Olsen, 2011), content of speech signal, speaker (Plyler, Alworth, Rossini, & Mapes, 2011), speech presentation levels (Freyaldenhoven, Plyler, Thelin, & Hedrick, 2007) and different types of background noise (Nabelek, Tucker, & Letwoski, 1991). The ANL ranges from 2 to 27 dB (Nabelek, Freyaldenhoven, Tampas, Burchfield, & Muenchen, 2006). In yet another study by Plyler, Alworth, Rossini, and Mapes (2011) reported ANL range -3.5 to 27 dB on an average. Harkrider and Smith (2005) suggested that the acceptance of background noise is related to individual variation in the afferent and / or efferent function of central auditory system. Thus, it is interesting to investigate, how the neural representation of amplified speech in good hearing aid users differed from poor hearing aid users.

Thus, in the present study, the research questions formulated were to investigate how the acoustic features of speech syllables are represented at the ear canal after being processed by a hearing aid? If the hearing aid preserves the spectral and temporal content of speech, then to what extent is the representation of amplified speech similar to the representation of speech in individuals with normal hearing at each level of the auditory pathway? If there is a significant effect on physiological response at each level of the auditory system, then how are the speech syllables encoded in good and poor hearing aid performers? These are the research questions being formulated for investigation in the present study.

In search of answers for these research questions, changes in signals in the output of the hearing aid should be recorded in the ear canal. Further, the representation of acoustic cues available at the brainstem and cortical levels also needs to be investigated using electrophysiological measurements. These provide information about the effect of amplification and hearing loss along the auditory pathway. It also improves our understanding regarding the representation of amplified speech at the different levels of auditory pathway. This information will in turn help the clinician to account the extent of success with auditory rehabilitation.

Need for the study

Despite improvements in technology and with well selected and appropriately fitted hearing aids, audiologists are not fully aware of the possible performance variability posed by the hearing aids, aging and hearing loss. Hence, there is a need to

study the representation of amplified speech along the impaired auditory pathway. This allows the audiologist to acknowledge the confronting variables. It might help in developing strategies in hearing aids to rectify some of these problems.

Need for the measurement of the output of the hearing aid in the ear canal. The acoustic cues of the incoming signal serve as the template using which the output of the hearing aid is compared to know the extent of modifications induced by the hearing aid. The probe tube microphone (PTM) recordings are often used to measure the amplified signals at the ear canal because of their clinical efficiency in studying the output of the signals recorded close to the tympanic membrane and it gets done quickly. Bray and Nilsson (2002) reported that recording of the PTM reflects the acoustic effects of pinna, ear canal and the electroacoustic performance of the hearing aid.

The probe microphone measurements have limitation especially with the signals to verify the hearing aid gain using a pure tone sweep, a composite signal or a broadband noise as input to the hearing aid. The static signals that are generally used as input for the measurement cannot adequately describe the effect of some features of a hearing aid such as compression. The pure tone sweeps have constant level over longer duration than the brief speech component, which varies in intensity at each frequency band rapidly across time (Keidser & Dillon, 2003).

The human ears are exposed to speech signals in day-to-day life. Thus, utilizing speech signal as input for measurement facilitates in knowing the way in which the hearing aid represents the spectral and temporal properties of speech in the ear canal of

the participant during PTM measurement. In order to obtain the information about spectral and temporal parameters of the speech stimulus in the ear canal, the output from the PTM is recorded on to a computer with Praat software installed in it. Later, the recorded output is analyzed for spectral and temporal aspects of speech signal. Hence, there is a need to study the hearing aid output at the ear canal.

In some individuals, the critical cues appeared to be available at the output of the hearing aid. However, the errors exhibited by some participants can not be resolved by acoustic analysis of hearing aid output alone. Thus, it is for this reason that the study concerning the representation of acoustic cues at the brainstem and cortical levels become imperative (Stapells, 2000; Hall, 1992).

Need for studying the representation of acoustic cues available at the brainstem level. Auditory brainstem response (ABR) is most commonly used in the assessment of auditory sensitivity in infants, children and adults who cannot participate in behavioral hearing evaluation (Hood, 1998) or in evaluating the status up to the brainstem level. Several types of stimulus and recording methods have been proposed to provide frequency specific information such as tone bursts, filtered clicks, tone bursts and clicks mixed with various types of noise and high pass masking of clicks (Hood, 1998). While normal click- and tone burst- ABRs are an indication of the integrity of the cochlea and the ascending auditory pathway up to the level of the brain stem, they do not provide further information about the encoding of more temporally complex signals such as speech.

On the other hand, the brainstem response to speech has proven to be a mechanism for understanding the neural bases of normal attention-independent auditory function (Johnson, Nicol, & Kraus, 2005) and in assessing the integrity of the neural transmission of acoustic stimuli (Russo, Nicol, Masacchia, & Kraus, 2004). Recording brainstem response to sound has long been established as a valid and reliable means to assess the integrity of the neural transmission of acoustic stimuli.

Speech stimuli have also been used in humans to study the response characteristics of the frequency following response (FFR) (Galbraith, 1994). FFR is a phase-locked response that 'follows' the waveform of the stimulating sound up to a frequency of 1000 Hz (Hoormann, 1992). The brainstem neural synchrony is well tuned to temporal and spectral characteristic of speech syllables (Banai, Nicol, Zecker, & Kraus, 2005) and imprecise encoding of speech syllables at the neural level contributes to communication problem (Kraus & Nicol, 2005). Johnson, Nicol, Zecker, Bradlow, Skoe, and Kraus (2008) have studied the brainstem response to voiced consonant vowel (CV) stop syllables /ba/, /ḍa/, and /ga/. Spectro-temporal information distinguishes these voiced consonant-vowel syllables. This information is contained within the burst and the first few milliseconds of the formant transition to the vowel. The spectro-temporal variations among stimuli were represented by the timing of the neural response. Hence, FFR to speech provides an objective way in which the sound structure of speech syllables is encoded at the brainstem level. Further, literature has documented that aging (Clinard, Tremblay, & Krishnan, 2010; Hemanth & Manjula, 2012; Anderson, Parbery-clerk, White-Schwoch, & Kraus, 2012) and hearing loss (Musser, 2010; Prabhash & Sandeep,

2011) alter the input before it reaches brain. The findings from studies on biological aging and hearing loss recognized the changes in the brain processing (latencies) and strength (amplitudes). There is a dearth in literature on the effect of biological aging and hearing loss in spectral processing at the brainstem level. Additionally, the way in which the amplified speech is represented at the brainstem level after fitting an individual with a hearing aid is not clear. If the brainstem neuron follows the time varying cues of amplified speech, the extent to which the representation of amplified speech at brainstem level lessens the problems posed by hearing loss needs to be evaluated by comparing the encoding of amplified speech in individuals with hearing impairment with that of speech encoded in individuals with normal hearing. This needs to be studied in different age groups. In addition, the way in which the representation of amplified speech differed in good and poor hearing aid performers needs to be evaluated. Thus, there is a need to know the way in which these variables affect the representation of available acoustic speech cues at the level of auditory brainstem.

Need for studying the representation of acoustic cues available at the cortical level. Cortical auditory evoked potentials (CAEP) such as the N1–P2 complex are frequently used to assess the neural detection of sound. The N1– P2 complex is thought to represent the synchronous neural activity of structure in thalamic-cortical segment of the central auditory system. Many investigators have used brief stimuli such as clicks (Ponton, et al. 2000), tones (Pantev, Ross, Fujioka, Trainor, Schulte, & Schulz, 2003) and synthetic speech stimuli (Sharma, Dorman, & Spahr, 2002) to evoke this response. Although synthetic speech syllables allow the investigator to control stimulus

dimensions, these stimuli are not representative of everyday speech. Hence, naturally produced speech syllables which vary with time and are highly complex are more useful as they evoke a complex neural response pattern (Polen, 1984). Tremblay, Billings, and Rohila, (2004) utilized naturally produced CV syllables having plosives and shorter pre-transition duration to record the neural activity at the cortical level. The conventional N1-P2 complex of LLR was elicited for these stimuli. Thus, LLR at cortical level can be recorded using naturally produced speech syllables.

Goodin, Squires, Henderson, and Starr (1978), and Pfefferbaum, Ford, Roth, and Kopell (1980) studied neural representation of speech at the cortical level in individuals with biologic aging. The results revealed prolonged latency and reduced amplitude as a factor of aging. They speculated reason for this as decrease in rate of neural transmission, excitatory and inhibitory neurotransmitter, and conduction velocity. Tremblay, Piskosz, and Souza (2002) investigated the combined effect of aging and age-related hearing loss. They concluded that elderly individuals with hearing impairment have pronounced effect in encoding of speech. In yet another study Oates, Kurtzberg, and Stapells (2002) recorded LLR and P 300 in individuals with different degrees of sensorineural hearing loss. They reported that cognition was affected more than solely due to audibility. Further, LLR was studied after fitting individuals having sensorineural hearing loss with a hearing aid. The brain processing and response strength was better in the aided than in unaided condition. Collectively, the findings suggest that CAEPs are a sensitive tool to obtain information on aging, hearing loss and amplification.

Naturally spoken speech syllables contain rapid temporal and spectral changes. The speech evoked cortical response elicits N1-P2 obligatory response for voiced and voiceless consonant vowel (CV) stop syllables (Tremblay, Friesen, Martin, & Wright, 2003). In these types of CV syllables, multiple response patterns temporally overlap, obscuring the presence of the rapid changes in the ongoing stimulus. Ostroff, Martin, and Boothroyd (1998) compared the cortical response to the syllable /si/ with the cortical responses to the sibilant /s/ and to the vowel /i/. They ascertained that the response to /si/ was a combination of the CAEPs to the onsets of the two constituent phonemes /s/ and /i/. These overlapping CAEPs within a single response for a stimulus have been termed the acoustic change complex (ACC) (Martin & Boothroyd, 1999).

Acoustic change complex (ACC) is one such potential that reflects the complex changes contained in the stimulus. These potentials are sensitive to time-varying cues such as changes in spectrum, amplitude, and periodicity (Ostroff, Martin, & Boothroyd, 1998), change from a harmonic tonal complex to a noise band with the same spectral envelope (Martin & Boothroyd, 1999) and formant frequency changes in an ongoing vowel (Martin & Boothroyd, 2000). Therefore, ACC response to speech provides objective information about the way in which the sound structure of speech syllables is encoded at the cortical level. However, there is a dearth of literature on how the speech encodes at cortical level in good and poor hearing aid performers.

From the literature, it is revealed that both hearing loss and amplification can alter the temporal and spectral contents of speech signal (Martin, Tremblay, & Korczak,

2008). For a given signal, the input to the hearing aid is known. After amplification, the extent to which the hearing aid preserves temporal envelope and spectral cues was analysed in their study. The Envelope Difference Index (EDI) was utilized in order to study the difference in temporal envelope in the unaided and aided conditions. The spectrogram was used to measure the fundamental frequency and the first two formant frequencies (at onset and offset of transition) in unaided and aided conditions. In addition, spectra were also noted to know the intensity as a function of octave frequency. It will be interesting to find out how the spectral and temporal aspects of the speech sound are relayed at the brainstem and cortical levels influenced by aging, hearing loss and amplification. At brainstem level, the response was captured using FFR; and at cortical level the response obtained using LLR and ACC. Thus the present study is being investigated.

Aim of the study

The main aim of the study was to investigate the representation of amplified speech along the ear canal, brainstem and cortical levels of the auditory pathway in individuals with sensorineural hearing loss.

Objectives of the study

The following objectives were formulated

1. At the ear canal level, to measure the spectral and temporal parameters of speech syllables in unaided and aided conditions.

2. At the brainstem level, to compare the representation of speech syllables between clinical group and group with normal hearing.
    a. To compare between the aided slope of V-A from clinical group and unaided slope of V-A from group with normal hearing.
    b. To compare FFR in terms of aided F_0, F_0 energy and F_1 energy in clinical group with unaided F_0, F_0 energy and F_1 energy in group with normal hearing.
3. At the cortical level, to compare the representation of speech syllables between clinical group and group with normal hearing.
    a. To compare the LLR in terms of aided slope of N1-P2 in clinical group with unaided slope of N1-P2 in group with normal hearing.
    b. To compare latency of ACC components in aided condition from clinical group with unaided condition from group with normal hearing.
    c. To compare amplitude of ACC components in aided condition from clinical group with unaided amplitude from group with normal hearing.
4. Comparison of brainstem responses and cortical responses in good and poor hearing aid performers.
    a. To compare the brainstem responses between good and poor hearing aid performers.
        i. To compare slope of V-A between good hearing aid performers and poor hearing aid performers.
        ii. To compare FFR in terms of F_0 of FFR, F_0 energy and F_1 energy between good hearing aid performers and poor hearing aid performers.

b. To compare the cortical responses between good and poor hearing aid performers.
    i. To compare LLR in terms of slope of N1-P2 between good hearing aid performers and poor hearing aid performers.
    ii. To compare latency of ACC components between good hearing aid performers and poor hearing aid performers.
    iii. To compare amplitude of ACC components between good hearing aid performers and poor hearing aid performers.

Statement of the problem

The input to auditory system is altered by the impaired physiological mechanism and then the altered input is relayed to the central auditory pathway. One of the rehabilitative tools to overcome the hearing problem is the use of hearing aid. Only some individuals with hearing problem benefit from a hearing aid, while others reject the hearing aids. Kochkin (2010) speculated the reason for rejection of hearing aid. He said that factors affecting the hearing aid use can be located anywhere from the hearing aid microphone till the integrity of neurons along the auditory pathway. From literature, it is also well documented that majority of individuals reject hearing aid due to background noise (Kochkin, 2010). Hence, it is necessary to classify the users of hearing aids into either good or poor hearing aid performers using a measure that can predict the hearing aid use.

The willingness to listen in noise may be more indicative of hearing aid use than understanding speech (Nabelek, Freyaldenhoven, Tampas, Burchfield, & Muenchen, 2006).The acceptable noise levels (ANL) is a measure that can be used at the time of audiological evaluation rather than speculate the reason for rejection through the measurement of outcome, i.e., after a period of use of the hearing aid. The ANL test can be administered prior to the hearing aid evaluation to predict the hearing aid use, as there was a good correlation between ANL scores and satisfaction and / or improvement with hearing aid in an individual. Nabelek, Freyaldenhoven, Tampas, Burchfield, and Muenchen (2006) have reported that the ANL differentiates good (<7) from poor (>13) hearing aid users.

Further, to know the extent of alteration from hearing aid, it is important to have an account on representation of the acoustic content of the incoming signal along the auditory pathway. As noted earlier the hearing aid alters some acoustic cues in terms of temporal and spectral parameters. Thus, the output of hearing aid has to be recorded in the ear canal to see the alteration in the signal after being processed by the hearing aid. Even when the speech cues are preserved after amplification, some listeners fail to recognize speech. Thus, the ability to measure electrical activity in the auditory system in response to sound provides information about the representation of the signal along the central auditory system.

The present study intends to provide direct evidence of electrophysiological changes along the auditory pathway in individuals with acquired sensorineural hearing

loss associated with aging, hearing loss and amplification. The finding from this study helps an audiologist to understand the extent of improvement provided by the hearing aid; and also the variability involved among good and poor hearing aid performers based on the integrity of the peripheral and central auditory system. Further, the findings on electrophysiological measurements throw light on the encoding of available acoustic cues by the auditory system of the listener.

Hypotheses

The null hypotheses were framed for each main objectives of the study. They were

1. At ear canal level, there is no difference in the spectral and temporal parameters of speech syllables between unaided and aided conditions.
2. At brainstem level, there is no difference in the representation of speech syllables between aided responses obtained from clinical group and unaided responses obtained from group with normal hearing.
3. At cortical level, there is no difference in the representation of speech syllables between aided responses obtained from clinical group and unaided responses obtained from group with normal hearing.
4. There is no difference in the brainstem responses between good and poor hearing aid performers and
5. There is no difference in the cortical responses between good and poor hearing aid performers.

CHAPTER - 2 REVIEW OF LITERATURE

Speech is a complex sound dealt by the auditory system. The neural representation of speech needs to capture the features of the signal for understanding. The general features of the neural representation of speech have been studied for over 25 years (Sachs & Young 1979; Delgutte 1980; Reale & Geisler 1980). Information in speech is encoded in a rapid sequence of different sound segments. The individual segments can be characterized by their frequency spectra, i.e., the distribution across frequency of the energy making them up. The spectra change with the speech segment across time, hence the resulting speech signal has a complex spectrotemporal pattern.

The spectral and temporal parts of speech vary rapidly with time. In order for the speech to be perceived, the neurons in the auditory system should respond to the subtle changes in the speech signal. Any damage to the auditory system resulting in hearing impairment alters the physiological process. This in turn modifies the representation of signal at different levels of the auditory system before it reaches the brain.

There are many rehabilitative procedures available to overcome the effects of hearing impairment including the use of appropriate hearing aids. Though, the prescribed hearing aid is well selected and optimized to the listening needs of the client, some users of the hearing aid report of only a minimal improvement. However, it is hard to determine whether the limited benefit was due to the limitation of the hearing aid technology to preserve the acoustic information or inability of the listeners to process the amplified information. Hence, in order to investigate the possible source of variability in

performance of individuals with hearing loss, the present study was undertaken. In addition, aging is supposed to influence the way in which the auditory system processes the signal. In light of this, the present study also intended to investigate the influence of aging on processing at brainstem and cortical levels of the auditory system. In this connection, the relevant literature has been categorized under different headings.

Acoustics of speech

An understanding of acoustics of speech necessitates the audiologist to discuss about the response, especially obligatory response, in terms of the way the auditory system encodes speech. A hearing aid recommended for an individual with hearing impairment should preserve the spectral and temporal features, in addition to making the soft syllables audible and loud syllables below the level of discomfort (Dillon, 2001).

It is important to remember that speech is not static, rather a complex signal that varies in temporal and spectral aspects over time. Recognition of speech is partly contributed by temporal cues (Healy & Warren, 2003). According to Rosen (1992), speech comprises of three main temporal features based on dominant fluctuation rates. They include the envelope (2 to 50 Hz), the fine structure cues and the periodicity cues. The envelope is the overall fluctuations in the amplitude, and this provides segmental cues for voicing, manner, vowel identity as well as prosody. The fine structure cues are rapid fluctuations in amplitude (less than 600 Hz) that provide information about voicing, place and manner of articulation. The periodicity cues include periodic (50-500 Hz) and

aperiodic (2-10 kHz) cues that provide information about voicing, manner, stress and intonation. Drullman, Festen, and Plomp (1994) reported that amplitude envelope cues are most important for speech recognition with smaller contribution from fine structure and periodicity.

Along with envelope cues, the spectral cues contribute to the identification of specific speech syllables. Kewley-Port (1983), and Stevens and Blumstein (1978) reported that spectrum of the burst is an important cue for the place distinction of stop consonants. According to Jongman, Wayland, and Wong (2000) fricatives are represented by interval of aperiodic noise, and are identified by spectral peak location.

From the above literature, it can be inferred that unique cues are responsible for perception of each category of speech syllables. The encoding of these unique acoustic cues in identifying the specific speech syllables will be contributed by each part of the auditory system. Consequently, to understand this phenomenon, it is reasonable that one should know the way in which the auditory system encodes speech. This would provide important insight into the basis of speech perception. Thus, in the current study spectral information was analyzed in each of the two syllables recorded in the unaided and aided conditions through spectrogram. In addition, spectra was measured to investigate the distribution of energy as a function of frequency in octave and also to determine the differences in unaided and aided spectra for each target test stimulus. Further, change in temporal envelope between unaided and aided versions of the speech syllables was examined by envelope difference index (EDI). In the present study, the representation of

speech was studied at the ear canal, brainstem and cortical levels of the auditory system. At the level of ear canal, the spectrum was measured in octave frequencies. In addition, fundamental frequency and the first two formant frequencies were analyzed. Further, temporal envelope difference of unaided and aided was computed. At the level of auditory brainstem level, the temporal information was measured using slope of V-A and the spectral information was measured by frequency following response (FFR). Further, at the cortical level, temporal information encoded in terms of slope of N1and P2, latency and amplitude were measured. These investigations were conducted at different levels of auditiory pathway using two consonant vowel stimuli (/ɖa/ and /si/), in both unaided and aided conditions. Thus, the inference made from this study is limited to the speech stimuli used in the study.

Representation of Speech in the Auditory System

The physiological mechanism involved in each part of the auditory system contributes to understanding the meaning attached to the speech. The external ear acts as a funneling mechanism (Shaw, 1997), helps in localization of speech syllables and provides gain at its resonance frequency. The syllables from external ear impinge on tympanic membrane to reach the middle ear. The middle ear efficiently transmits the sound energy to the cochlea by impedance matching of the transfer function. From the transduction process in cochlea, the speech signals reach the brain via the auditory pathway.

External and Middle ear. The characteristics of the acoustic speech signal are modified by the transmission characteristics of the outer ear. Shaw (1997) reported that the pinna of the outer ear contributes to localization and funneling the syllables into the ear canal. The transfer function of outer ear peaks with a gain of 15-20 dB from 300 Hz to1500 Hz (Primary peaks), and with a gain of 10 dB at and above1500 Hz (Secondary peaks). This is due to the resonance frequency of the external auditory canal and concha respectively (Wiener & Ross, 1946). The ear mould or ear tip used along with the hearing aid alters the resonance by reducing the SPL at the primary and secondary peaks and sometimes the peaks are even absent (Ewertsen, Ipsen, & Nielson, 1957) or the secondary peaks shift downward in frequency (Studebaker, 1974). Thus, the amount of energy from external environment impinges on tympanic membrane (TM) depends on cytoskeleton of ear canal and also the amount of resonance property of ear canal reduced by ear mould.

The output from the earmould in the ear canal reaches the tympanic membrane. The tympanic membrane transduces the sound from acoustic energy into mechanical energy and the vibration of the tympanic membrane in turn is passed on to the middle ear. The middle ear efficiently transmits the sound energy to cochlea through impedance matching (i.e., from air to fluid medium). The transfer characteristic of middle ear has resonance frequency in the range from 0.5 to 2 kHz (Homma, Du, Shimizu, & Puria, 2009).

To summarize, the pinna is important in sound localization. The sound energy in the ear canal is augmented by the resonance. However, in hearing aid users, the ear

mould or ear tip alters the resonance frequency of the ear canal. The sound pressure level in the ear canal reaches the middle ear, when it impinches on tympanic membrane. The transfer characteristic of middle ear stabilizes the sound, as it travels from air to fluid medium in the inner ear.

Inner ear. The encoding of signal at the level of cochlea has been extensively studied and well understood (Davis, 1958; Bekesy, 1960; Yates, 1995; Ruggero, Rich, Recio, Narayan & Robles, 1997). The cochlea is arranged tonotopically, such that the apical end of the basilar membrane is tuned to lower frequencies and the basal end is sensitive to higher frequencies (Bekesy, 1960). According to Bekesy (1960), this is partly related to a stiffness gradient that increases toward the base, which results in maximum displacement dependent on frequency. At moderate intensities, the vibration of the basilar membrane grows non-linearly as the stimulus frequency moves towards characteristic frequency (Ruggero, Rich, Recio, Narayan & Robles, 1997). The energy at characteristic frequency is reduced by frequencies below characteristic frequency of suppressor tones. Thus, the mechanism of non-linearity is initiated at the level of cochlea. The consequent displacement of hair cells from the vibration of basilar membrane results in the transduction process (Dallos & Harris, 1978). Yates (1995) reported that the outer hair cells have an influence on the mechanics of cochlea (i.e., changing their length, shape and stiffness in response to stimulation), and were connected by efferent nerve fibers that carry information from auditory system to the cochlea. Thus, a higher center (superior olivary complex of auditory brainstem) regulates the analysis of auditory signals and the

tonotopic arrangement of the cochlea is preserved in the auditory nerve and throughout the auditory system (Harrison, 1985).

Movement of the cochlear partition produces deflection of stereocilia of inner hair cells. The deflection opens ion channels in the stereocilia resulting in intracellular voltage fluctuations and release of neurotransmitter (Davis, 1958). Thus, the acoustic signal is neurally transduced by sensory part of inner ear (inner hair cells), connected by afferent nerve fibers, which generate action potential with respect to the displacement of the basilar membrane.

To summarize, the disturbance of fluid in the cochlear duct set the vibrations in basilar membrane. These vibrations bend the stereocillia of sensory hair cells (outer and inner hair cells), which open (depolaraise) and close (hyperpolarise) the ionic channels and there by release the neurotransmitters. The outer hair cells are innervated by efferent nerve fibers which provide feedback and also fine tune the sensory input. The inner hair cells synapse with the afferent nerve fibers and the information is successively transferred or relayed to different levels of the auditory pathway (Harrison, 1985).

Auditory Nerve. The auditory nerve captures the important features of signal and relays the information to brain via the auditory pathway for understanding the intended meaning. Sachs and Kiang (1968) used two tones named primary and secondary. The secondary tones are having broad range of frequencies to study the non-linearity. They recorded discharge rate of neurons with respect to their characteristic frequency (i.e., the

neuron responsible to process desired frequency). The each characteristic frequency of neuron was activated by primary tone and simultaneously secondary tone at different levels was presented. Similar procedure was carried out by presenting different frequencies of secondary tone and noted the amount of inhibition on neuron at characteristic frequency in terms of discharge rate. They noted discharge rate at characteristic frequency of neuron reduced when secondary tone is in closer with primary tone. This mechanism of two tone inhibition helps to encode complex stimuli in the auditory nerve. They inferred that two tone inhibition exists in all the fibers of auditory nerve. However, in realistic condition secondary tone, which is delivered for long time continuously varing its frequency leads to adaptation. This study provided benchmark to conduct auditory neural activity using complex stimuli, as the tones used as target stimuli in the present study were acoustically different from speech stimulus.

Sachs and Young (1979), and Liberman (1978) recorded the activity of high spontaneous auditory nerve fiber in response to synthetic vowel stimuli. They analyzed the average discharge rate as a function of fiber characteristic frequencies (CF). At low stimulus levels, the rate of discharge was maximum in the fibers of CFs close to the formant frequencies. The extent and position of discharge rate near the CFs of auditory nerve fibers (ANFs) depends on the pattern of formant frequencies (Delgutte & Kiang, 1984). In yet another study by Sachs and Young, (1979) recorded discharge rate for vowels at moderate sound levels. The vowel spectra were well represented by rate-place code and ANFs discharge maximum at its CFs close to a formant frequency. Further, inhibition noted in those fibers especially away from CFs. However, the recording of

Sachs and Young (1979) were made without considering low spontaneous nerve fibers and the explanation on encoding of vowel spectra at higher level is not known. In their experiment they utilized anesthetized cats, in which the efferent activity could have reduced. Activation of efferent fibers might have improved the rate-place representation (Hillenbrand, Getty, Clark, & Weeler, 1995). Stimuli being used were synthetic vowels, though vowels are rarely static in real speech.

Liberman (1982) recorded discharge pattern of ANFs in response to rapid changes in the temporal and spectral content of speech. The response was analyzed in post-stimulus time histogram (PSTH). PSTH represents the average discharge rate of auditory nerve at its characteristic frequency (CF) over short interval or bins as a function of time. Consonant such as stops and fricatives are characterized by a maximum discharge rate at high frequency region. However, to the onset of vowel the discharge rate was maximum at the low frequency. Further, the activity of ANFs is modulated by medial olivocochlear (MOC) bundle of the auditory brainstem. The cell bodies of MOC neurons are located in the superior olivary complex (SOC). The axons of SOC terminate on outer hair cells in the cochlea (Warr, 1992; Guinan, 1996). Guinan and Gifford (1988) reported that CF of ANFs shifts its dynamic range by 15-30 dB with respect to stimulus frequency, in response to MOC stimulation. Especially, in the presence of background noise and also at higher levels of intensities, the stimulated MOC neurons exert an anti-masking effect. This mechanism shapes the auditory input, leading to next level of processing and also protect from hazard (Winslow & Sachs, 1987).

To summarize, the mechanism of two-tone inhibition, discharge rate function and place coding in the auditory pathway drive the precise association between the characteristic frequency of an ANF and its place of innervations along the cochlea. These associations continue along the major part of auditory pathway up to the auditory cortex (Brugge & Reale, 1985).

Auditory brainstem. The fibers of auditory nerve (AN) terminate in the cochlear nucleus (CN), the first stage of central auditory processing (Blackburn & Sachs, 1990). The cells of CN contain various units of response and operate in parallel mode (Rhode & Greenberg, 1994). These cells receive input from auditory nerve, but differ in terms of synaptic organization, processing properties and also synaptic connection to next processing part of the auditory system (Young & Oertel, 2004). The response to speech has been studied in stellate cells and bushy cells of anterior part of ventral cochlear nucleus (AVCN). The response analyzed in PSTH from the Stellate cells in AVCN fire repetitively at a rate unrelated to the period of stimulus. In PSTH, the discharge rate across time resemble chopper like response. Bushy cells in AVCN fire initially and gradually its rate reduces as a function of time and the response resemble primary like response. The responses of chopper unit (from stellate cells) and primary like unit (response from bushy cells) of AVCN process the information carried by AN from cochlea (Rhode & Greenberg, 1994). The response property of AVCN is different from AN interms of pattern of discharge rate and phase lock capacity. In AVCN, spike trains were regular (Young, 2007) and phase lock to stimuli approximately 2 kHz. (Winter & Palmer, 1990). May, Prell, and Sachs (1998) conducted a study on vowel representation

in VCN and inferred that primary unit of response resembles auditory nerve representations. Blackburn and Sachs (1990) compared the response of chopper unit from CN and AN fibers. The results indicated that chopper unit retains good rate representation in the high spontaneous nerve even at higher stimulus level.

At each stage of sequential auditory pathway, upper limit of frequency for phase locking decreases (de Ribaupierre, Rouiller, Toros, & de Ribaupierre 1980). The possible reasons would be synaptic jitter and different ionic channels (Trussell, 1999). Liu, Palmer, and Wallace (2006) studied phase locking response to pure tones at inferior colliculus (IC). It was reported that the upper limiting frequency of phase-locking varied greatly between anatomical divisions. The upper limit of phase locking was greater than 1kHz in central nucleus; 700 Hz in dorsal cortex; and 320 Hz in external nucleus. However, the acoustic characteristics of tones were static across time. Hence, the sensitivity of the IC neuron to modulation frequencies was investigated by Joris, van De Sande, and van der Heijden, (2005). The discharge rate pattern was investigated at each modulated frequency. The findings inferred that the IC neurons fire precisely to each period of modulated frequency less than 1 kHz. However, the neurons are unable to fire precisely at higher modulation frequencies. Further, the neurons of IC were investigated to speech segments in sentence. The strongest responses were noted in the onset and burst portion of speech; whereas, the nerve fibers fire at steady and low discharge rates especially for vowels. These results suggest that the central representation of speech may emphasize transients over steady-state responses (Delgutte, Hammond, & Cariani, 1998).

To summarize, at the auditory brainstem level, the speech signals are phase locked. The phase locking is different in parts of the CN, which was due to the dendritic sparse, synaptic jitter, and transduction of ionic channel. The phase locking was narrowed at the higher auditory centers.

Auditory Cortex. Neurons in auditory cortex are tonotopically arranged. That is neurons in each row of auditory cortex are fine tuned to particular frequency and arranged side by side in order from low to high frequencies (Eggermont, 1991). Schreiner (1998) reported that for lower level tone, neurons which are tuned to particular frequency or best frequency (BF) would respond. However, at higher input level of desired frequency, the neurons other than BF neurons respond and further inhibitory mechanism reshape the response. Nelken (2004) suggested that cortical neurons respond to spectrotemporal aspects of the stimulus. In yet another study by Eggermont (1995) the discharge rate in response to stop consonants vowel /pe/ stimulus on anaesthetized cat was investigated. Two peaks of response was noted i.e., higher discharge ate was time locked to one on release of stop consonant and another on the onset of voicing. The above studies were conducted on animals using invasive procedure by placing electrode directly on neurons of best frequency and analyzed the response in terms of discharge rate. This procedure is impossible to adopt on human participants, such that non invasive mechanism such as elctrophysiological approach was involved to study the auditory cortical behavior in response to tones and complex speech stimulus. Eggermont (1991) reported that neurons in the auditory cortex are phase locked to slow rates of stimulation, i.e., less than or approximately 25 Hz. This physiological process is represented in an

abstract manner at the cortex level, which is transformed from phase locked response of auditory brainstem level. Makela, Alku, Makinen, Valtonen, May, and Tiitinen (2002) studied N100 m (Magnetoencephlogram) cortical response to variation of F_0 with constant formant frequency and pure tone stimuli of 100 Hz, 200 Hz, 300 Hz and 400 Hz. Results of N100m responses were extremely similar in spatial activation irrespective of F0. However, intriguing finding of N100m responses in terms of processing and strength differed with pure tone stimuli. They opined that at auditory cortex, the response is insensitive to the F0 variations. Shestakova, Brattico, Soloviev, Klucharev, and Huotilainen (2004) reported that the spectral distance between F_1 and F_2 was reflected in the dipole location of N100m responses. The authors suggested that cortical sensitivity to F1-F2 differences can be explained by inhibitory mechanism. If the separation in Hz between two formant frequencies were closer, greater would be the neural inhibition. This in turn influences the location of the dipole source as measured by Magneto-encephlo-gram (MEG). To sum up, this section throws light on how each part of normal auditory system contributes to the identification of speech syllables from acoustic cues embedded in each category of speech syllables. The auditory system of individuals with aging and hearing loss suffers from understanding the speech. Now, the question arises on how speech cues are encoded at the different levels of the auditory pathway in individuals who are aging.

Physiological changes affecting the perception of speech - biological aging and hearing loss related factors. To design appropriate hearing aid to overcome the difficulty faced by biological aging and hearing loss individuals, it is important for an

audiologist and an engineer to understand the physiology of biological aging and damage to cochlea and further its concomitant changes along the auditory system.

Biological aging. The effect of age-related hearing loss on peripheral auditory system and its concomitant changes at the central auditory level were difficult to study as there are numerous variables beyond the control of the researcher. The variability includes inherent genetics (Gates, Couropmitree, & Myers, 1999), environmental exposures accumulated over lifetime, drug injuries (Helzner, 2005). These predisposing variables account for hearing loss and sometimes it may even accompany with biological aging. It is difficult to point out the cause of hearing loss either from particular predisposing factor or from biological aging, as these variables may interact in many ways. Hence, the animal models of age-related hearing loss have been used frequently in aging research as each component of auditory system can be studied thoroughly by controlling individual variable.

Mills, Schmiedt, and Kulish (1990) conducted study on age-related changes in the cochlea of mongolian gerbil through auditory potentials. The result revealed 15-35 dB HL threshold shifts, with greater loss at the high frequencies. Gratton and Schulte (1995) attributed that the hearing loss due to age is associated with damage in the lateral wall of cochlea including stria vascularis; and spiral ligament fibrocytes (Spicer & Schulte, 2002). Stria vascularis innervates blood supply to the cochlea. Damage to stria vascularis, decreases endocochlear potential and in turn restricts the inner hair cell transduction process (Schulte & Schmiedt, 1992). The spiral ligaments support basilar membrane and

consist largely of ion-transport fibrocytes which help in recycling of K^+ ions. The spiral ligament damage leads to disruption in the mechanism of recycling of K^+ ions efflux from the hair cells back to endolymph (Buckiova, Popelar, & Syka, 2007). Cochlear cell dysfunction, alterations in intracellular organelles, chemical alteration in the endolymph and reduced number of synapse in the hair cell result in impaired metabolism and finally lead to diminished input into the central auditory nervous system.

Bao and Ohlemiller (2010) reported that neurons of spiral ganglion functions as relay station in transferring the auditory information from hair cells to central nervous system. However, losses of neurons are the major cause for the age-related hearing loss rather than loss of neuronal connections (Morrison & Hof, 2007). The reason attributed was all living cells produce free radicals, which react with damaged proteins and lipids causing neurons malfunction (Willott, 1991). Further, central component of auditory system undergo direct and / or secondary changes induced by age-related hearing loss. Caspary, Schattenan, and Hughes (2005) conducted role of inhibitory response in dorsal and ventral cochlear nucleus. The vertical cells in the cochlear nucleus provide inhibitory input to fusiform cells through glycinergic circuitry. This mechanism declines with age, resulting in altered rate level function and fails to control over the output, which leads to serious consequence on processing of temporal cues.

The nuclei in the superior olivary complex involves in encoding complex features of sound through modulating the ascending information from cochlear nucleus. Zettel, Zhu, O' Neill, and Frisina (2007) investigated potassium and calbindin immuno reactivity

at superior olivary complex in mice. They found that significant loss of immuno reactivity in older mice, which attributed to reduction of total number of cells and in turn fail to process stimuli with high frequency. The altered physiological processes due to aging at the peripheral system block the inhibitory neurotransmitters, GABAergic and glycinergic, at the inferior colliculus. The resultant impact reflected in the imbalance of excitation and inhibition of neurons and finally disrupts the temporal processing (Walton, Frisina, Ison, & O' Neill, 1998). Further, these neurotransmitters were investigated at the different layers of primary auditory cortex in rat. They found a significant alteration in inhibitory neurotransmitter in older rats at layer 2, which contribute to loss of temporal processing.

The age-related hearing loss from animal model cannot be fully generalized to human auditory aging, as it differed from lifespan, disease pathogens during the course of life time, hearing loss onset and its duration, nature of age-related hearing loss. Among animals that were used for the investigation of age-related hearing loss had varied life span. Each animal has different duration of onset and progression of hearing loss. Further, the range of complex task cannot be implemented in animal model, whereas the humans are exposed to those tasks.

Schuknecht (1962) conducted a study by correlating the audiograms and temporal bones of elderly human participants. He reported four types of presbycusis, viz., sensory, neural, metabolic and cochlear conductive. Schuknecht and Gacek (1962) investigated the incidence and lesions in each type of presbycusis by analysing the

temporal bones of elderly humans. They concluded that damage to stria vascularis resulted in metabolic presbycusis and this was found to be predominant type of hearing loss due to aging. Lee, Mathew, Dubno, and Mills (2005) explored the audiometric findings in each type of presbycusis. The audiometric findings in metabolic presbycusis showed a 10-40 dB HL hearing loss with slope of -5 to 5 dB per octave at low frequencies (0.25 - 1.0 kHz) and gradual sloping loss at high frequencies (1 - 8.0 kHz), with slope ranging from 10 to 20 dB octave per octave. This suggests that from the audiogram configuration in older adults, one can predict the possible region of damage at the sensory or the neural level. The temporal asynchrony in older adults accompanied with hearing loss often complain of difficulty to understand speech (Dubno, Dirks, & Morgan, 1984). The reason for unable to follow message in older adults was studied using behavioral experiment in which temporal parameter of the test stimuli were varied to investigate its importance on speech perception (Coughlin, Kewley-port, & Humes,1998; Price & Simon, 1984). Coughlin, Kewley-port, and Humes (1998) investigated the relationship between identification and discrimination of vowels in elderly population. They reported inability to extract temporal cues and indirectly suggested difficult to process vowel-format discrimination. In a similar line of investigation on temporal processing in elderly individuals, Price and Simon (1984) noticed deficits in discrimination of voice onset time. Hence, there is a need to study the representation of speech at different levels of auditory system through non-invasive methods such as evoked potentials, as the direct investigation of observed findings at each level of the auditory system is impossible on human participants.

To summarize, stria vascularis and spiral ligament fibrocytes are the common sites of cochlear aging. This results in inhibition of transduction process and ion-transport imbalance. At higher auditory centre, the free radicals produced in healthy neuron cells combined with lipids and proteins of malfunctioning neurons lead to loss of neurons in biologic aging. Further, loss of immune reactivity leads to reduction of total number of neurons. This altered physiological process inhibits neurotransmitter. It is well documented that physiological variation arises with respect to age. To understand physiological variation across age in the auditory system, the non-invasive evoked potentials are useful.

Hearing loss. Individuals suffering from hearing impairment of cochlear origin frequently complain of difficulty in understanding speech, at low levels (Glasberg & Moore, 1989). Harrison (1986) reported that cochlear hearing loss resulted from damage to outer hair cell (OHC), thereby there is a loss of the biological active mechanism of amplification known as electromotile properties (Ashmore, 1987). Dallos and Harris (1978), and Glasberg and Moore (1989) reported that disruption of outer hair cells primarily resulted in elevated thresholds, softness imperceptions, wide auditory filters, and normal or near normal temporal resolution.

Absloute threshold. The absolute threshold of sound is the minimum level at which an individual detects sound (Moore, 2007). Absolute thresholds were elevated due to OHC dysfunction, which impairs the active mechanism resulted from the reduced vibration of the basilar membrane for low level syllables. Hence, higher sound pressure

level is required to just detect the vibration in the ear with cochlear damage. The consequence of the impaired active mechanism leads to 50 dB hearing loss at low frequency and 60 dB loss at high frequency (Moore, 2007).

Frequency resolution. Frequency selectivity is the ability of the auditory system to resolve the frequency components of a complex sound (Evans, 1978). Florentine, Buus, Scharf, and Zicker (1980) conducted a study on frequency selectivity in individuals with normal hearing and with hearing impairment of different degrees. They utilized four methods, pyshcoacoustical narrow band masking, tuning curve, critical bandwidth, and maximum loudness summation to identify frequency selectivity and also to explore the possible interaction among procedures. They concluded that frequency selectivity was reduced and were positively correlated among procedures. Reduced frequency selectivity revealed as an increased in the bandwidth of auditory filters with marked shallow low frequency skirts. However, the implication of reduced frequency selectivity on speech understanding has not been investigated. Glasberg and Moore (1989) utilized the speech for recognition and correlated with different psychoacoustic measures of frequency selectivity in different listening conditions. They used three procedures; frequency discrimination of tones; frequency discrimination of complex tones; and frequency selectivity as a measure of notch noise method. The result revealed a positive correlation among frequency selectivity and speech recognition in quiet condition. Further, the thresholds in dB HL were correlated with the SRT. This is because the subjects selected for the study had a relatively narrow range of threshold, i.e., uniform losses as a function of frequency. In addition, the stimuli were presented at relatively higher sound pressure

level to all the ears with impairment, which is generally > 20 dB above their absolute threshold (20 dB SL). In the above experiments frequency selectivity was measured using simultaneous masking method assuming the auditory filters were symmetrical. However, the auditory filters are asymmetric and have rounded top, rather steep skirts as in normal hearing. In many maskers method, auditory filters were shown asymmetric with respect to the signal frequency. The reason would be off-frequency listening, i.e., signal-to-noise ratio is higher for a filter adjacent to the signal frequency.

Patterson (1976) used a notch noise method to describe the filter shape. He reported that an auditory filter is symmetrical on a linear frequency scale. Moore and Glasberg (1983) reported that the non-simultaneous masking eliminates off-frequency listening. Glasberg, Moore, and Nimmo-Smith (1984) compared the auditory filters with simultaneous and non-simultaneous maskers. They reported that the auditory filter shape measured in the non-simultaneous masking was typically sharper than that measured in simultaneous making though the suppression mechanism is damaged in cochlear impairment.

Turner and Henn (1989) conducted a study to predict vowel recognition from forward making paradigm, a measure of frequency resolution obtained from each subject with cochlear hearing impairment. Their finding suggests that the frequency resolution predicted the recognition of vowel. Further, they speculated that confusion among vowels were less likely, as the differences in formant frequencies between vowels are large enough so that they are easily resolved even by listeners with poorer than normal

frequency resolution. Thus, the loss of OHC produces relatively wider auditory bandwidth than normal, which smoothen the spectral peaks and troughs in speech stimuli (Bacon & Brant, 1982).

Temporal Resolution. Temporal acuity is the ability of the auditory system to discriminate order of event in the minimum time interval (Green, 1971). Patterson (1976) conducted a study on temporal acuity using a Huffman sequence of stimuli, presented at different sensation levels (SL re: PTA), on ten subjects with sensorineural hearing loss and three subjects with normal hearing. Their finding revealed that eight of the ten listeners showed better temporal acuity, similar to the results of subjects with normal hearing.

Irwin, Hinchcliff, and Kemp (1981) investigated temporal acuity using the gap detection method. The gap detection threshold was obtained at different sound pressure levels (SPL) on seven subjects with sensorineural hearing loss and six subjects with normal hearing. The results indicated poorer temporal acuity. The reason for mixed results in temporal acuity could be the presentation levels i.e., equal SPLs or SLs. Glasberg and Moore (1989) investigated temporal gap detection thresholds in both SPLs and SLs at different listening conditions. Further, the relationship between speech recognition threshold (SRT) and temporal gap detection thresholds was measured. The results revealed that the difference in gap detection threshold reduced, when the normal ear compared at the same SLs as used for impaired ear. However, at equal SPLs significant difference was noticed in gap detection between the ears with normal hearing

and impairment. This can be attributed to the lower SLs in ears with impairment. In addition, the gap detection was positively correlated with the SRTs in quiet condition.

From the review it was confirmed that psychoacoustic methods are sensitive test to investigate the frequency resolution and temporal resolution in ears with impairment. In summary, elevated threshold, softness imperception and poor frequency resolution are likely to be the direct consequences of a loss of cochlear non-linearity. Hearing aid is being utilized to overcome the underlying problems to some extent.

Acceptable noise level

Hearing impairment is often the handicapping condition that may go unnoticed. Untreated hearing loss is associated with decrease in physical and psycho-social well-being (Bess, Lichtenstein & Logan, 1991), ease of communication (Arlinger, 2003), cognitive function (Cacciatore, Napoli, Abete, Marciano, Trinssi, & Rengo, 1999), social interactions (Resmick, Brant, & Verbugge, 1997) and reduced speech perception skills due to deprivation from auditory stimulation (Arlinger, 2003). Thus, one among the rehabilitation options for such problems is hearing aid.

Most of hearing aid users complain background noise as the major reason for the dissatisfaction with the hearing aids (Surr, Schuchman, & Montgomery, 1978 ; Kochkin, 2010). In other words, the hearing aid rejection is directly related to not willing to accept the noise while listening to speech (Nabelek, Tucker, & Letwoski, 1991). Traditionally, hearing aid is fitted based on the principle of quantifying speech intelligibility in the

presence of noise. There are many other similar tests viz., Speech in Noise (SPIN), Hearing in Noise Test (HINT), Competing Speech Test (CST), and Quick Speech in noise (QSIN) test. Irrespective of the tests selected, wide range of scores was noted among those who obtain hearing aids. Additionally, there was no relationship between the score on speech intelligibility-in-noise and his or her real-world benefit and / or satisfaction with hearing aids.

The investigators evaluating the hearing aid outcome measure reported that there is need to have tests that correctly predict the hearing aid use (Nemes, 2003). The hearing aid outcome measure has its own practical limitation in that these scales cannot be administered prior to the hearing aid fitting or even during the hearing aid evaluation. Thus, Nabelek, Tucker, and Letwoski (1991) have introduced the new concept called Acceptable Noise Level (ANL). In this, the willingness of the patient to accept the background noise level (BNL) was quantified in the presence of speech at most comfortable level (MCL). The difference between MCL and BNL was calculated to obtain ANL in dB. In other words, ANL=MCL-BNL.

The premise of ANL is that some listeners reject hearing aids because of not willing to accept the background noise. The ANL can be estimated prior to hearing aid evaluation to predict the successful use of a hearing aid, as there is a good correlation between the value of ANL and satisfaction and / or improvement in hearing aid use. Various ranges of ANLs have been reported in literature. The range of ANL as reported by Nabelek, Freyaldenhoven, Tampas, Burchfield, and Muenchen (2006) is from 2 to 27

dB. Plyler, Alworth, Rossini, and Mapes (2011) have reported that the ANL can range from -3.5 to 27 dB on an average; whereas Freyaldenhoven, Plyler, Thelin, Nabelek, and Muenchen (2008) have reported that the ANL values range from -2 to 18 dB.

Further, Nabelek, Freyaldenhoven, Tampas, Burchfield, and Muenchen (2006) have demonstrated that listeners with low ANLs (<7 dB) were more likely to become successful hearing aid users, and listeners with high ANLs (>13 dB) were more likely to become unsuccessful users. Thus, the ANL measure predicted the successful hearing aid use with 85% accuracy. In their study, the prediction of ANL for individuals with midrange values between 7 and 13 dB was not helpful, as the probability of success was 50%. However, the ANL is not affected by age, hearing sensitivity, and language (Branstrom, Lantz, Nielsen, & Olsen, 2011); content of speech signal and speaker gender (Plyler, Alworth, Rossini, & Mapes, 2011); speech presentation levels (Freyaldenhoven, Plyler, Thelin, & Hedrick, 2007); different types of background noise (Nabelek, Tucker, & Letwoski, 1991); native language (von Hapsburg & Bahng, 2006). The ANL measure is reliable (Nabelek, Tampas, & Burchfield, 2004), even though studies report of large inter-subject differences among homogenous populations.

Roger, Harkrider, Burchfield, and Nabelek (2003) suggested gender of the listener as a possible factor contributing to the inter-subject differences. The results indicated a statistically significant difference between the MCLs of male and female participants. Males accepted a higher intensity of background noise while listening to speech at MCL than females, by approximately 7 dB. Though, male participants had a higher MCL and a

higher BNL than females, the ANL was same for the two genders. These inter-subject variability observed in MCL and BNL of ANL accounts from differed physiological activity in the peripheral and central auditory systems (Nabelek, Tampas, Burchfield, 2004).

In order to study the effect of ANL on a physiological process involved in higher auditory center, electrophysiological measurement was carried out. Tampas and Harkrider (2006) examined auditory evoked potentials in participants having low and high ANLs. The results revealed that in subjects with low ANL, the amplitude difference become increasingly more remarkable in wave V of the auditory brainstem response (ABR), middle latency response (MLR) and late latency response (LLR). The amplitude of each response was higher in high ANL group than those with low ANL. Conversely, in latency of each response was earlier in low ANL than high ANL. This could be due to the stronger efferent mechanism such that sensory inputs are suppressed and / or central afferent mechanism is less active. Harkrider and Tampas (2006) investigated the physiological activity of peripheral and central auditory nervous system in participants with normal hearing having low- and high- ANL. The results indicated no difference in contra-lateral otoacoustic emission and ABR peaks of I and III. However difference emerges in wave V component of ABR, and Na and Pa components of MLR suggesting that ANL is of central origin.

To summarize, the hearing aid is prescribed to overcome the problem faced by hearing loss. However, the amount of benefit received from the hearing aid is varied.

The ANL test can be administered prior to the hearing aid recommendation to predict the successful benefit from hearing aid, as there was a good correlation between person's ANL and satisfaction and / or benefit from a hearing aid. Further, an objective electrophysiological measure is well correlated with the ANL value, suggesting that the ANL is sensitive to central auditory mechanism. Thus, good and poor hearing aid performers can be classified before hearing aid trial. It would be interesting to further understand how the neurally encoded acoustic cues are represented at different levels of the auditory pathway in individuals with low and high ANLs.

Analysis of Hearing Aid Output

Despite having the same degree and configuration of hearing loss, satisfaction from hearing aids varies among individuals. The variability in the satisfaction from a rehabilitative device might probably account from compression parameter of hearing aid and / or at the interaction between hearing aid output and physiological mechanism in ear with impairment.

Effect of amplification characteristics on acoustic cues. Individuals with hearing impairment of cochlear origin suffer from softness imperception, in which an individual with elevated thresholds will perceive loud syllables as louder than a person with normal hearing. Thus, the dynamic range between threshold and discomfort is reduced (Moore & Glasberg, 1986). One among the rehabilitative options available to individuals with hearing impairment is hearing aid. Earlier linear amplification was

prescribed, in which a constant gain was provided irrespective of the input levels. However, the linear amplification has its own drawbacks as it provided same gain across frequencies which resulted in the upward spread of masking i e., vowel masking over consonants. Additionally, even the softer syllables perceived to be louder, as the gain provided were constant irrespective of input intensities, and at higher intensities the signals were distorted due to pick clipping. This was overcome by compression circuit in the hearing aid (Hawkins & Naidoo, 1993). Hence, the linear amplification was replaced by the compression amplification system.

Compression in hearing aids provides option to deliver appropriate gain at each frequency band depending on the level of the input signal. Compressor in hearing aid reduces upward spread of masking, by assigning lower speech weight at lower frequency than at mid and high frequencies (Byrne, 1996). Thus, it helps to reduce low-frequency noise interference and provides better quality and speech intelligibility. In addition, compression in hearing aid amplify speech optimally well within dynamic range of client i.e., softer speech making soft by amplifying well above his threshold and louder sound compressed well within dynamic range) as the compression threshold activated at the lower level (Dillon, 2001).

However, there was equivocal evidence in improvement of speech recognition using non-linear hearing aids. van Tasell, (1993) reported the hearing aids alter the inherent temporal envelope. Temporal envelope alteration after hearing aid processing reduces the level difference between consonant and vowel leads to blur the boundary

between consonant and vowel. This led negative impact on speech perception in individuals with hearing impairment. In yet another study by Hickson and Byrne (1997) reported detrimental effect especially on stop consonant followed by vowel at the shorter release time. The author attributed possible reasons that the compression activated for the vowel, continued to be active on consonant due to shorter release time, thereby reducing the audibility of consonant and consequently blurs the boundary between consonant and vowel, and eventually resulted in misperception. Further, the stronger level of vowel leads to upward spread of masking on weaker level of consonants, where compression continued to be activated on consonants, leads to deterioration perception.

Conversely, there are studies documented that increasing the consonant level and reducing the vowel level, though the level difference between vowel and consonant was lesser, it improve speech intelligibility. Hickson and Byrne, (1997); Hickson and Thyler, (2003); Souza and Turner, (1996, 1998); and Drullman, Festen, and Plomp, (1994) studied consonant and vowel level difference on speech perception. Their finding infer that though the consonant vowel level difference reduced, the level of consonant audibility was present and also level of vowel decreased to avoid the upward spread of masking, it increases the speech intelligibility. Thus, it was noted that temporal alteration has both positive and negative effects on speech perception.

Hawkins and Naidoo (1993) confirmed that the listeners prefered non-linear hearing aids, as the varying levels of speech fits into the narrow dynamic range through a compression algorithm (Dillon, 1999). There were many experiments conducted on

speech recognition using two channel hearing aids with linear and varying compression parameters (i.e., compression ratio, crossover frequency) in individuals with cochlear hearing loss. Laurence, Moore, and Glasberg, (1983); Moore, Laurence, and Wright, (1985); Moore and Glasberg (1986) investigated speech recognition of key words embedded in a sentence and also speech intelligibility using linear aid and two-channel compression hearing aid in individuals with sensorineural hearing loss in quiet and different types of noise such as speech spectrum noise and speech babble noise with varying signal-to-noise ratios (SNR) conditions. In their study, the participants appreciated speech recognition and intelligibility in two channel compression hearing aid than with a linear aid, in both quiet and different SNR conditions. Hickson, Dodd, and Byrne (1995) utilized individuals with mild to moderate sensorineural hearing loss to study the consonant perception by recording the output of nonsense syllables from both linear and compression hearing aids with varying compression ratios (1.3 and 1.8) in quiet and noise conditions. The results showed that perception of frication and plosive was adversely affected with varying compression ratio (1.8) of amplifier.

It was noted from the above literature that the behavioral responses were analyzed after fitting the hearing aids. However, the alteration of inherent acoustic cues after being processed by a hearing aid has not been investigated. Hickson, Thyer, and Bates (1999), and Hickson and Thyer (2003) conducted studies on the effect of compression on the level difference between consonant and vowel. Syllables consisted of voiceless stops; fricatives and affricates combined with vowels /a/, /i/ and /u/ served as test stimuli. These stimuli were presented at 60 and 75 dB SPL through the loud speakers. The output was

recorded from two channel (compression ratio set independently) compression hearing aid (12 compression condition variable in 11 steps from 1.0 linear to 3.0), which was connected to 2 cc coupler and the output was recorded subsequently. The results indicated that the level difference between consonant and vowel was increased with compression ratio of 3 especially at high frequency channel. However, the output of the hearing aid was recorded from 2 cc coupler, which does not reflect the acoustic effects of pinna, ear canal and torso.

In another study by Ellison, Harris, and Muller (2003) used Knowles Electronic Mankin Acoustic Research (KEMAR) to investigate the interactions of hearing aid release time and fitting formula on consonant and vowel level difference. That is effect of hearing aid compression release time and fitting formula on speech acoustics. NAL-NL1, DSL i/o, and FIG 6 prescriptive formulae were programmed in each 40 ms and 640 ms release time. The output of the hearing aid on 32 phonemes were analyzed for speech measures like long term average speech spectra (LTASS), level difference between consonant and vowel and the root mean square (RMS) amplitude of phonemes. The data were compared between unaided with aided conditions. The results indicated that longer release time relatively affected all the speech measures in each prescriptive formula. However, the output recorded from 2cc coupler and KEMAR has its own drawback. The real ear coupler difference is not added to the output of the hearing aid. The ear canal resonance has not been subtracted from output of the hearing aid, as it obscured the resonance at the time of fitting the hearing aid with ear mold in the ear canal. It is for this reason, there is a need to know the acoustic changes to the hearing aid processed speech

signal at the level of ear canal using probe tube microphone measurement (PTM). This helps to understand the compression setting that best preserved the acoustic properties leading to best perception and the compression setting that most alters the acoustic properties resulting in poorer speech perception.

Evaluation of hearing aid output using probe microphone measurement. The probe tube microphone measurement (PTM) is used to objectively investigate the effect of amplification in response to the realistic stimuli i.e., speech in the ear canal (Bray & Nilsson, 2002). The PTM recordings are used because it consumes less time and does not involve active participation of patients. Additionally, recording reflects the acoustic effect of subjective factors such as pinna, ear canal, head and torso; and also the electro-acoustic effect of the hearing aid.

The PTM measurement is used to adjust hearing aid gain to match with the targets at each frequency prescribed by the fitting formula (Cunningham, Lao-davila, Eisenmenger, & Lazich, 2002). The prescriptive formulae are mathematical models developed for prescribing gain to the patient specific hearing loss (Dillon, 1999). Though gain in the hearing aid was set from prescriptive formula, there is still inadequate match to the target. Henning and Bentler (2005) studied compression dependent differences in hearing aid gain between speech and non speech input signals. Speech and non speech stimuli were presented at 65 dB HL to master hearing aid with independently varying the compression parameters. The result revealed a lesser gain to speech than non-speech stimuli in compression parameters of shorter release time and lesser compression ratio.

The reason could be that the compressor rapidly adjusts its gain in response to dynamic signal such as speech than non-speech. As the level of speech peaks was relatively well above the compression threshold of 50 dB HL, which causes saturation of the hearing aid circuit. However, in the verification stage of hearing aid fitting, the prescriptive formula used was based on normalizing loudness in each narrow band of frequency using non-speech stimuli and it is not appropriate for a speech as a input. Some of the nonlinear prescriptive formula such as FIG 6, IHAFF, DSL i/o utilized the principle of loudness normalization in each frequency using non-speech stimuli. Dillon (1999) reported that normalization of loudness at each frequency does not consider the low frequency emphasis of speech.

Non-speech stimuli are not par to speech. Pure tones are longer duration than brief speech components; there is no envelope change in an ongoing stimulus; and no crest factor in pure tone stimuli (Souza & Tremblay, 2006). Hence, the pure tone stimuli were replaced by composite signal (speech weighted noise), and digi speech (Souza & Tremblay, 2006). These stimuli mimic the speech characteristics such as shape, envelope, crest factor and bandwidth (Scollie & Seewald, 2002), but still contain significant discrepancies (Mueller, 1992; Dillon, 2001).

Instead of utilizing speech like stimuli in programming hearing aid, it is believed that use speech signals more closely assesses the performance in the real world. Dillon (1999) developed NAL-NL1 prescriptive formula with the consensus to provide a gain frequency response that maximizes speech intelligibility while keeping overall loudness

at a level no greater than that perceived by a normal hearing person listening to the same sound. The principle includes the idea of normalizing the overall loudness, not the loudness at each frequency. NAL-NL1 includes even lower cut frequency below 1000 Hz than the other procedures. This reflects the low frequency emphasis of speech. Speech intelligibility index methods were utilized to calculate the gain at different input levels. Scollie and Seewald (2002) reported that verification is often conducted using artificial test signals such as tones and noise, all though the targets are defined for speech inputs. Therefore, there is apparent mismatch between the stated goals of hearing aid selection strategies and the test signal used to verify. Hence, the speech stimuli should be used as a test signal to match the hearing aid gain, prescribed by the speech based rationale on prescriptive formulae. Unfortunately, matching the hearing aid gain to the prescribed target does not necessarily mean a better hearing aid fitting. Hence, the verified hearing aid should be tested under different types of speech stimuli and levels of signals that we are exposed to hear in real life.

Stelmachowicz, Kopan, Mace, Lewis, and Nittrouer (1995) fitted linear and non-linear hearing aids to three adolescent listeners with mild to moderate sensorineural hearing loss. Consonant-vowel (CV) and vowel-consonant (CV) speech stimuli were used as test stimuli. The output of a hearing aid in the ear canal was recorded using the PTM system. Spectrographic analyses were carried out for the recorded stimuli. The results revealed precipitous roll-off of high frequency response limited the consonant cues and also blurring of the boundary between the consonant and vowel. Thus, hearing aid may not preserve the temporal and spectral content of speech (van Tasell, 1993). Of

course, there is a need to consider the auditory ability of the listeners and hearing aid output in the ear canal.

Souza and Tremblay (2005) conducted a study to correlate the consonant error to acoustic analysis of amplified speech in mild to moderate sensorineural hearing loss subjects. A total of 66 recorded CV nonsense syllables were used as test stimuli. The results revealed place errors. The /ɖa/ stimulus was consistently misperceived as /ga/. This is because the aided burst spectrum of /ɖa/ recorded at the output of the hearing aid in the subject's ear is similar to the unprocessed burst spectrum of /ga/. Kewley-Port (1983) reported that for the identification of stop consonant sound in the initial position requires the spectrum of the burst as the primary cue for recognition. Additionally, the listeners mispercepived affricative consonant as fricative, when the input to hearing aid was affricative. These temporal distinction such as (i.e rise time and duration of noise is the primary cues to discriminate between fricative and affricative consonants (Howell & Rosen, 1983). The /zhi/ stimulus was consistently misperceived as /dgi/. This is because higher amplitude of spike in the onset of consonant /zhi/ recorded at the output of the hearing aid in the subject's ear is similar to the unprocessed amplitude of consonant onset /dgi/. The hearing aid output recorded in the ear canal should be analyzed in the spectral and temporal domain to understand the alteration made by the amplification characteristics.

To quantify the temporal contrast between the unaided and aided version of same speech stimuli Fortune, Woodruff, and Preves, (1994) developed Envelope difference

index (EDI). The EDI represents a precise quantification of the temporal contrast that exists between the unaided and aided waveforms. The output of EDI ranges from 0 (perfect correspondence between the envelopes) to 1 (no correspondence between the envelopes). Fortune, Woodruff, and Preves, (1994) reported a temporal contrast of 0.03 between the unaided and aided version of same signal indicate that the envelope is preserved. Souza, Jenstand, and Boike (2006) reported that with increased compression ratio and release time leads to level difference between consonant and vowel, resulted in increased EDI which consequently reduce the speech recognition. The EDI values of about 0.20 and above decrease the speech recognition (Jenstand & Souza, 2007). Temporal envelope conveys the information of manner but not the place cues (Rosen, 1992). However, temporal envelopes are indirectly conveying the voicing cues embedded in fine structure. If place information is not conveyed by envelope, then altering of envelope cues should not affect place cues. However, envelope amplitude reduced in one band resulted change in gross spectral shape (Blumstein, Issacs, & Mertus, 1982). Thus, there is a purpose to utilize the EDI and spectral (F_0, F_1 and F_2) measurement for analyzing speech after hearing aid processing.

As mentioned earlier, REM used for verification of the hearing aid gain for speech to match with the target gain prescribed by fitting formula is necessary. To program the hearing aid gain, to bring speech syllables well above the thresholds of an individual, results in the sound being merely audible. However, audibility does not necessarily entail perception. Correlating the acoustic analysis conducted to record the output of hearing aid and speech perception in the ear canal can solve the puzzle. That is,

though the acoustic feature was altered after amplification, the speech was being recognized correctly. This finding could probably be due to the redundancy of the speech. In other cases, where the acoustic cues was undistorted but listeners failed to recognize correctly. This is because of insufficient sensitivity in that area of the cochlea or concomitant changes at the higher auditory pathway. Hence, evoked potentials to speech stimuli should be recorded to validate the perception of speech that has been registered in the different levels of the auditory system.

In the present study, the hearing aid being set at preferred level by utilizing NAL NL-1 prescriptive formula with shorter release time of 30 ms, compression threshold at 55 dB SPL and a compression ratio ranges of 1.1 to 1.4 in both channels. In this setting, the hearing aid was programmed. Further, the hearing aid was optimized by presenting the ling's sound at 65 dB SPL. The target test stimulus was presented through loudspeaker and the output of hearing aid was recorded using probe microphone measurement. From the recorded output spectral and temporal analysis were carried out to know the extent of alteration caused by a hearing aid.

Physiological measures of auditory System

Electrophysiological responses were used to estimate the representation of spectral and temporal cues at the auditory system, but these responses were known less in poor and good hearing aid performers. The objective tests are used to analyze the hearing aid fitting by showing the representation of cues that have been registered at the levels of

the brainstem and cortex. Such a test provides subtle information to clinician in fine tuning the hearing aid and further directs the hearing aid designers to develop an effective strategy in utilizing digital signal technology to patch up the lost information in clients having altered physiology.

Brainstem Level. The Frequency Following Response (FFR) is recorded to investigate the integrity of neurons at the auditory brainstem level. FFR is a time-locked response to periodic aspects of the speech stimuli up to approximately 1 kHz (Kraus & Nicol, 2005). Encoding of fundamental frequency and F1 in some instances (Krishnan, 2002) can be measured using the FFR. In earlier studies of FFR, various stimuli such as clicks, tone pips and sinusoids were used to understand transient and sustained response (Kraus & Nicol, 2005). However, these stimuli were poor approximation to behaviorally relevant syllables that we encounter in our daily listening environment.

For perception of speech, the formant structures, frequency transition and acoustic onsets of speech cues were important the way the human auditory brainstem represents these acoustic cues of speech needs to be investigated. Krishnan (2002) conducted a study on brainstem (FFR) responses to three steady-state vowels. Each stimulus presented at 85 to 55 dB, in steps of 10 dB. The responses of FFR were compared with the spectral content of the vowel stimuli. The only difference among each of the vowel stimuli were the first two formant frequencies. His results indicated that at higher stimulus intensities, the FFR accurately represents F1 and F2; however, the increased representation of F1 was seen relative to F2. This finding supports the results of Sachs and Young, (1979) in

which vowel representation in the auditory nerve of animal was explored. These studies provide evidence that in both auditory nerve and brainstem level, mechanism of phase-locking encodes formant structure. Further, spectral visualization of the amplitude of harmonics closer to F1 and F2 formant frequencies as a function of intensity indicated higher amplitude seen at harmonics closer to that of formants and also the suppression of energy between harmonics centered at formants. A similar study was conducted on brainstem encoding of voiced consonant-vowel stop syllables (Johnson, Nicol, Zecker, Bradlow, Skoe, & Kraus, 2008). Three voiced stop consonant vowel (CV) syllables (/ba/, /ɖa/ , & /ga/) each with a duration of 170 ms and fundamental frequency (F0) of 100 Hz were utilized to record FFR from 22 normal children with age ranging from 8 to 12 years. The only difference between these stimuli was the F_2 and F_3 varied in the initial 40 ms of stimulus duration and later made constant. Since the F_2 and F_3 frequency ranges are well above the phase-locking capabilities of the brainstem, the data were analyzed using latency differences between responses. The earliest latencies were noticed in the order of stimulus /ga/ /ɖa/ and /ba/ due to the tonotopicity of the auditory system. Hence, it was speculated that separate specialized neurons for different formant frequencies were present at level of the brainstem.

van der Werff and Burns (2011) recorded auditory brainstem response for click stimulus and FFR to CV syllable on younger and older adults having normal hearing with thresholds of < 20 dB at 4000 to 8000 Hz. The result revealed the response strength and magnitude at onset and offset for both non speech and speech stimuli were significantly different in older adults than their younger counterparts. However, no difference was

noted between young and older adults in the response obtained at the transition and sustained portion of the stimulus.

In another experiment by Anderson, Parbery-Clark, Schwoch, and Kraus (2012) reported that the prolonged neural timing and reduced strength in the FFR was elicited to the transition portion of stimulus in older adults. It suggests that temporal precision is impaired in older adults at the sub-cortical level though the participants in these studies were free from hearing loss except at high frequencies (< 20 dB at 4000 to 8000 Hz). It consequently leads to a deleterious effect on understanding speech in the presence of noise.

Further, to probe the representation of speech at auditory brainstem from altered physiological mechanism caused by a complex interaction by aging and hearing loss Anderson, Parbery-Clark, Schwoch, Drehobl, and Kraus (2013) conducted a study. They recorded FFR to /ɖa/ stimulus on older adults having normal hearing and hearing impairment (sloping loss). The results revealed that the neural response amplitude of F0 (fundamental frequency) and H1 and H2 (harmonics) were higher in the aided condition than in the unaided condition, though the stimulus level was matched. An interesting finding was that the response magnitude (F0, H1 and H2) were reduced in older adults having normal hearing than their younger counterparts, in both the conditions (unaided and aided). This was speculated to be because of larger excitatory neural activity due to the broadened filters.

To summarize, the FFR represents the acoustic cues of speech syllables at brainstem level. However, from the literature it was noted that biologic aging, hearing loss and age-related hearing loss has serious impact on processing of speech cue to understand the message. In most of the studies, synthetic stimuli such a vowels (low /a/, high /i/ and mid /u/) and /ɖa/ without pre-voicing were used to record the FFR. These speech syllables are sensitive to assess the apical portion predominantly (till 4 kHz) and its representation at the sub cortical auditory level. However, the representation along the auditory pathway to high frequency stimulus is still questionable, as the older adults have difficulty in perceiving the fricatives (Bilger & Wang, 1976). Thus, there is a need to know how the spectral component of naturally produced speech (/ɖa/ and /si/) is represented at the brainstem level from altered physiological mechanism by an individual with biological aging, hearing loss and age-related hearing loss. Further, there is a need to determine how the speech cues are systematically represented at the cortical level, which has been routed through auditory brainstem.

Cortical level. The Cortical Auditory Evoked Potentials (CAEP) are an obligatory response, i e., the response varies with the physical characteristics of stimulus parameter. The CAEPs are evoked by rapidly changing acoustic components within speech-like syllables or speech stimulus. From literature, the Late Latency Response (LLR) to speech stimulus was often obtained from stimulus having shorter pre-transition period, especially voiced and voiceless stop consonants. Yet another potential from CAEP was acoustic change complex (ACC), elicited to longer pre-transition duration. Both LLR and ACC are sensitive to time varying acoustic cues at the level of the cortex.

Late Latency response. The late latency response is an electrophysiological approach utilized to investigate the temporal processing of the auditory system with high temporal precision of 1 ms at the cortical level (Hillyard, 1993). The components of LLR (P1, N1, and P2) are obligatory responses (Hyde, 1997), i.e., LLR changes their properties in relation to the physical characteristics of the stimulus. The amplitude of P1 is relatively small, thus the AEP morphology is mainly focused on the N1-P2 complex. The N1-P2 complex reflects neural synchrony of thalamo-cortical segment of the human auditory cortex. The LLR peaks of N1 in ~100 ms and P2 in ~200 ms occur after the stimulus onset (Naatanen & Picton, 1987). The N1 is suggested to reflect the sound detection function. It is sensitive to the onset of the sound, such as intensity and inter-stimulus interval. The P2 reflects the sound content properties such as acoustic or phonetic structures (Naatanen & Picton, 1987). The effect of age should be carefully considered, before attempting to reach conclusion in clinical populations, as the physiologic and metabolic changes take place in the auditory neuron throughout the life span. The impact of hearing loss at different age groups can be understood clearly if there is a knowledge on the temporal mechanism involved due to aging.

Goodin, Squires, Henderson, and Starr (1978) studied age-related variations in individuals with normal hearing. Forty-seven subjects ranging in the age from 6 to 76 years (in two groups - < 15 years of age and adults) were utilized. The LLR was recorded to tone bursts presented in an oddball paradigm at 60 dB SL. In adults, there was a systematic increase in latency and decrease in amplitude for each component of LLR (N1, P2 and N2) with age. They speculated that this result was due to a decreased rate of neural transmission, excitatory and inhibitory neurotransmitter, conduction velocity, and

cell and dendrite losses. In a similar study by Pfefferbaum, Ford, Roth, and Kopell (1980) investigated the effect of aging by considering 20 elderly (mean age =78.6 years) and young adults (mean age =22.5 years) subjects. The results revealed that there was no difference between elderly and young adults. This could be due to the stimulus presented at a higher level, which might have reached an asymptote. The latency of P2 was prolonged in elderly than young adults, and this difference was significant, due to the attention factor. In the slope of the regression line, it was found that latency of P2 (0.7 ms / year) was more prolonged than N1 (0.1 ms / year). Further, the amplitude of N1 and P2 was higher in older subjects than younger counterparts. This difference found significant only in the amplitude of N1. Though the study was aimed to assess the cortical neural integrity in older and younger groups, utilizing cross section design in the method would increase the sensitivity to assess the impact of aging.

Laffont, Bruneau, Roux, Agar, Minz, and Cathala **(**1989) recorded LLR from 30 adults in the age ranging from 20 to 80 years. These participants with normal hearing were divided into three groups (adults, middle age and older adults). Sound burst of four intensities (50, 60, 70, and 80 dB HL) was used. The effect of age and intensities on the responses of amplitude of P1-N1; N1-P2; and latencies of P1, N1 and P2 were analyzed. The result of each mean response was higher in amplitude and prolonged latency in older adults than middle age adults to four different intensities. Further, increased P1-N1 amplitude as a function of stimulus intensity was apparent in older adults. This could be due to the dopamine metabolism in older subjects. In another study Gordon, Lim, Li, Leslie, and Wright (1998) examined the effect of age on LLR. Fifty participants were

involved in the age range from 18 to 70 years. The LLR was obtained in conventional odd ball paradigm to tonal stimuli, presented at 80 dB SPL on equal number of participants in each decade. The latency of each component of LLR was prolonged with age. This difference in latency was significant except at N2. Further, there was no pattern noted in the amplitude of LLR. The mixed results in amplitudes were due to the variability in the method. This includes the sub-groups of age involved in the study, associated medical problem that was not controlled; higher level of stimulus presentation might have led to asymptote and probable chance of recruitment in the participants.

Although LLR is evoked by brief stimuli such as tone burst, the acoustic features of tone burst are far from complex speech stimuli. Tremblay, Piskosz, and Souza (2002) investigated the neural representation at the cortical level to speech cues in older adults. They hypothesized that the aging adversely affects the ability to process temporal cues. To study this hypothesis, the investigators recorded LLR on ten young adults with normal hearing (YNH) and ten older adults having normal hearing (ONH) to voiced and unvoiced syllables at 65 dB SPL in conventional odd ball paradigm. Stimuli were seven synthesized tokens, in which VOT varied every 10 ms step size, from /ba/-/pa/ continuum. The results of behavior threshold in differentiating the stimulus revealed that older adults performed poorly than younger adults in discriminating the 10 ms VOT contrast. The P2 latency was delayed in response to all the stimuli for older adults. Except at at 0 ms, the N1 latency was prolonged in older adults than their counter part. They concluded that aging individuals have difficulty in speech perception due to altered excitatory and inhibitory processes. Further, to confirm the physiological mechanism of

inhibitory and excitatory process, Tremblay, Billings, and Rohila (2004) studied the effects of age and stimulus presentation rate. They recorded LLR to tonal and speech stimulus at the presentation level of 74 dB SPL in slow, medium and fast rate of stimulus. The latency of each component of LLR was prolonged in ONH than in YNH, in both tonal and speech stimuli. However, more prolongation was noted at a faster rate in speech stimulus in ONH than YNH. They concluded refractory period might affect the synchronized neural activity underlying prolonged latencies of CAEPs recorded at a faster rate of presenting the speech stimulus. It was concluded from this study that though aging individuals had normal hearing sensitivity, they have difficulty in temporal resolution.

Apart from the temporal resolution difficulty, the aging individuals have degraded ability in frequency resolution. Harkrider, Plyler, and Hedrick (2005) investigated effects of age and spectral shaping on the behavioral and physiological representation of stop consonant stimuli. Synthesized /ba/, /ɖa/ and /ga/ voiced stop consonant vowel and replica of each stimulus with enhanced F2 formant frequency stimuli were utilized to record CAEPs in YNH and ONH participants at 82 dB SPL. The results revealed that in ONH, each component of CAEPs to enhanced stimuli was similar to those measured in YNH to original stimuli. The finding suggests that difficult in categorizing stop consonants based on F2 formant frequency, but the difference in findings of CAEPs from YNH and ONH participants was minimized when the F2 formant frequency was enhanced. Hence, it was concluded that aging has a degrading effect on temporal resolution and frequency resolution abilities. Now, the research question arises as to the

way in which speech is processed in studying the combined effect of aging and age-related hearing loss.

Tremblay, Piskosz, and Souza (2002) investigated the combined effect of aging and age-related hearing loss on ten participants in each group of YNH, ONH and older adults of hearing loss (OHI). Behavioral threshold and LLR to varied VOT stimuli (/pa/-/ba/) were obtained at 74 peSPL. The behavior threshold in differentiating the stimulus revealed OHI performed poorly than ONH. The findings in behavioural measure were depicted even in elecphysiological approach. The processing of N1 and P2 latency of LLR reduced as a function of increased VOT in OHI than ONH. The finding was discussed based on combined effect of aging and hearing loss. Marin, Kurtzberg, and Stapells (1999) and Oates, Kurtzberg, and Stapells (2002) studied the combined effect of aging and hearing loss and its concomitant change at cortical level.

In SNHL, the audibility is one among the confronting problems. Martin, Kurtzberg, and Stapells (1999) investigated the effect of decreased audibility on LLR by utilizing the normal hearing subjects. The high pass masking noise was presented to simulate hearing loss. The target test stimuli /ba/ and /ḍa/ were presented at 65 dBpeSPL in odd ball paradigm. The N1 latency prolonged and amplitude reduced with decreasing high pass masking noise and more pronounced effect was noted at 1 kHz high pass masking noise. They attributed that N1 reflects the presence of audible stimulus energy. The N1 peak diminished at decreased high pass masking. Further, inability to discriminate the phonemic boundary at reduced high pass masking noise obscured the formant frequency transition between the stimuli. However, the effect of SNHL and its

concomitant changes on central auditory pathway may not be accounted by merely reducing the audibility of target test stimuli by various high pass masking noise. The reduction of audibility produced a slowing of brain processes which was reflected by increased N1 latency.

Oates, Kurtzberg, and Stapells (2002) investigated the effects of SNHL on LLR. LLR was recorded to /ba/ and /ḍa/ speech stimuli presented in an odd ball paradigm at 65 and 80 dB SPL on subjects having different degrees of hearing loss. The N1, P2 and P3 latencies were prolonged and amplitudes reduced with respect to the magnitude of SNHL. At 65 dB SPL, the strength of LLR was stable in participants who had hearing loss less than 55 dB HL at frequencies of 1 kHz and 2 kHz. However, at 80 dB HL the response amplitude was stable till the hearing loss reached 65 to 75 dB HL. The signal strength of approximately +12 to +15 dB was required to record CAEP in different degree of hearing loss. Further, the latency of P3 was prolonged and amplitude reduced compared to N1 and P2 peaks. This suggested that there was an effect of reduced cognition in addition to the effect of loss of audibility.

Apart from assessing the speech processing in aging alone, combined effect of age and age-related hearing loss CAEPs has its own advantage. The CAEP recorded in the awake state correlates well with behavioral thresholds, and is more reliable. It assesses speech perception at cortical level and also is useful in measuring the efficacy of amplification both in infants and adults. Dun, Cater, and Dillon (2010) recorded CAEPs and obtained behavioral thresholds in children of 30 months age. It was noted that there

was a good correlation between LLR and audibility assessed behaviorally in children with SNHL. Pearce and Golding (2003) recorded LLR in awake infants in aided condition to /m/, /t/ and /g/ stimulus at 65 dB SPL. They were able to record peak P1 reliably in awake infants and wave shapes to each stimulus suggested speech perception. Agung, Purdy, McMahon, Dillon, Katsch, and Newall (2004) recorded CAEPs for Ling's syllables to objectively verify the hearing instruments in infants. They noted that the cortical response wave shape varied with different Ling's sound. Sharma Martin, Roland, Bauer, Sweeney, Gilley, and Dorman (2005) investigated the central auditory development in children with hearing impairment using CAEP. The P1 latency reduced with post hearing aid fitting. From the findings it was concluded that the hearing aid provides ample stimulation pertaining to normal development of the central auditory pathway. However, the audibility, spectral information and frequency response of the hearing aid was not controlled.

Hence, there is a growing interest in LLR as a measure of cortical function in individuals with hearing loss and hearing aid users. Further, to systematically investigate the combined effects of SNHL and prescribed personal hearing aids on CAEPs, Korczak, Kurtzberg, and Stapells (2005) recorded LLR at two different intensities in unaided and aided conditions. The N1component of LLR to /ḍa/ and /ba/ stimuli at 65 dB SPL and 80 dB SPL in oddball paradigm were recorded from fourteen adults having SNHL with moderate and severe to profound degree. At 65 dB SPL, the brain processing (latency) and response strength (amplitude) was better for aided than unaided condition, in both moderate and severe to profound SNHL, to each stimulus. This was due to the higher

signal level provided by hearing aid than the noise. However, at 80 dB SPL, variability of response was noted between conditions to each stimulus. They speculated that this could due to the hearing aid circuitry noise level. Hence, there is a necessary to measure the amplification characteristics on cortical response.

Prior to investigating the effect of hearing aid amplification on clinical population, it is first necessary to examine the basic science of amplification on acoustic speech stimuli. It is important to know how the amplifier of hearing aid preserves the acoustic cues. Billings, Tremblay, Souza, and Binns, (2007) recruited 13 individuals with normal hearing to eliminate the possible confounds that hearing impairment might contribute to the amplification and intensity effects. The CAEPs were recorded to seven levels of stimulus intensity from (30 to 90 dB peSPL). As expected the latency reduced and amplitude increased with increase in intensity. However, in aided condition to the same set of stimuli with 20 dB gain, response variability was noted. The results revealed that with 20 dB of hearing aid gain affects neural responses differently than 20 dB of stimulus intensity. The reason for this could be that the reduced SNR was stable in aided condition than unaided condition ; increased ambient noise when occluded the ear canal with hearing aid; hearing aid gain makes even the noise to be audible to the individuals with hearing impairment; higher input level activate the output limiting of hearing aid by compressing the signal well with in the dynamic range and at certain intensity (i.e., higher input intensity) there is no change in processing and strength of response (i.e., neural mechanism has reached asymptote). In order to study the amplification

characteristics alone, normal participants were utilized to investigate the effect of signal to noise ratio and also by varying the gain on CAEP. .

Billings, Tremblay, Stecker, and Tolin (2009) investigated the effect of SNR and absolute signal level on CAEPs. The CAEPs were investigated in 12 conditions; two levels (60 and 75 dB SPL) and 6 SNR (quiet, 20, 10, 0, -10, -20 dBA) conditions. The results revealed that there was no significant difference between signal levels. However, there was a significant difference between SNRs at each tonal stimulus. The result suggests that LLR primarily is sensitive to SNR, rather than absolute signal level. To improve the signal level, clinicians might encounter problems at the time of fitting a hearing aid assuming that increasing the signal level (adjusting the gain) will improve the morphology of the evoked response. In the similar line of experiment Billings, Tremblay, and Miller (2011) investigated the aided LLR in response to changes in hearing aid gain. The LLR was recorded both in unaided and aided conditions on nine individuals having normal hearing. In the aided condition, the input level was kept constant and the hearing aid gain was varied (0 to 30 dB, in steps of 10 dB). The SNR at ear canal level was measured in both unaided and aided conditions. The results revealed that aided LLR latencies were delayed relative to unaided conditions. This could be due to modification of the signal characteristics by the hearing aid by reducing SNR which in turn has an effect on the LLR.

Collectively, the previous research finding of CAEP assists the audiologist in the initial fitting of hearing aid, helps in fine tuning the hearing aid and also sensitive to the

experience-related plasticity associated with amplification. From the literature it was noted that target test stimulus used was synthetic and presented in odd ball paradigm. Although, the synthetic speech stimuli elicit a well defined response and also allow the researchers to control certain aspects of the stimulus, it is a far representative of everyday natural speech.

Acoustic change complex. Speech evoked CAEP are frequently used to study the neural detection of speech syllables at the cortical level. Pre-stimulus transition for speech stimulus more than 60 ms. (Ganapathy, Narne, Manjula, & Mohan, 2013) evokes multiple overlapping P1-N1-P2 complexes reflecting acoustic changes in the ongoing stimulus (Ostroff, Martin, & Boothroyd, 1998). This collection of overlapping AEPs within a single response for a stimulus is termed acoustic change complex (ACC) (Martin & Boothroyd, 1999).

In speech, the spectral and temporal cue change across time. Each acoustic cue conveys important information on speech perception. Synthetic non-speech stimuli are used to study the contribution of each acoustic cue, as particular cues can be varied in a controlled way. Martin and Boothroyd (1999) investigated periodicity change in an ongoing stimulus. They used synthetic stimuli such as noise-tone, tone- noise, tone - tone, noise- noise. In the first two stimuli, there is a change in the middle of ongoing stimulus. In the last stimulus, two copies of the noise/ tone was concatenated. These stimuli were used to study the difference in neural aggregate at cortical level for both time varying and non time varying stimuli. The response to tone only and noise only stimuli showed clear

N1/P2 complex. However, change in the middle of the ongoing stimuli shows multiple N1/P2 complexes. It was concluded that ACC is sensitive to change from a harmonic tonal complex to a noise band with the same spectral envelope. Thus, the stress and intonation cues conveyed by periodicity were detected at the cortical level using ACC.

Apart from periodicity changes, spectral and amplitude cues vary in the ongoing stimulus, which provide the place and voicing cues that are important for discrimination of speech syllables. Martin and Boothroyd (2000) investigated the neural detection at the cortical level to the changes in amplitude alone and the combination of amplitude and spectral change in the ongoing stimulus. They used two stimuli sets (/uu/ and /ui/) and each set varied with 11 amplitude changes. The results revealed that the multiple N1-P2 complex was evoked by amplitude increments of 2 dB or more and decrements of 3 dB; and second formant frequency change from perceived /u/ to /i/.

In additional, voice onset time (VOT) and duration cues rapidly change within speech and are important contributors for speech perception. To study the influence of temporal stimulus change Burger, Hoppe, Lohscheller, Eysholdt, and Dollinger (2009) utilized five monosyllabic words beginning with consonant, which were temporally separated by different VOTs and simple tone burst (1 kHz) of 300 ms duration including 5 ms rise and fall time of synthetic stimuli. The synthetic tone burst closely matched with each monosyllabic stimulus. The result revealed overlapping N1/P2 complexes in monosyllabic speech syllables even with short VOT. The response of each monosyllabic word was closely matched with synthetic tone burst. Further, the clear N1/P2 and visually

detectable complex were noticed for monosyllables beginning with voiceless consonant. This is because of longer VOT. Hence, the pre-transition stimulus duration is essential to avoid the overlap of N1/P2 complex.

Ganapathy, Narne, Manjula, and Mohan (2012) investigated the minimum pre-transition duration to elicit an ACC using synthetic tone complex. Tone complex was created by pure tone of 1 kHz followed by 2 kHz with a total duration of 350 ms The pre-stimulus duration alone (1 kHz) was varied systematically from 50 ms to 150 ms in 10 ms steps keeping the total stimulus duration of 350 ms The finding suggests that minimum pre-stimulus duration required to evoke multiple N1/P2 complexes to speech stimulus was 100 ms. From this it was confirmed that the neural detection in response to time varying acoustic cue can reliably be studied using ACC. Thus, ACC is clinically utilized in assessing intensity discrimination (Martin & Boothroyd, 2000) and frequency discrimination (Martin, 2007). However, in the above mentioned experiments, synthetic speech stimuli were utilized to study the spectral and temporal cues of speech represented at the cortical level using ACC. The synthetic speech stimuli elicit a well defined complex response (Martin & Boothroyd, 1999) and also allow the researchers to control certain aspects of the stimulus. However, it is a far representative of everyday natural speech (Tremblay, Friesen, Martin, & Wright, 2003).

Ostroff, Martin, and Boothroyd (1998) investigated cortical responses to naturally produced speech syllable /sei/ decomposed to reflect the contributions of the acoustic events contained in the constituent phonemes /s/ and /ei/. The finding suggests that

cortical regions activated from /s/ to /si/ stimulation merely overlap to the response of constituent phoneme. The ACC recorded on individuals having normal hearing using naturally produced /see/ and /shee/ speech syllables were reliable. It contains first N1-P2 complex (1^{st} trough and peak) corresponding to a change from silence to frication; a second N1-P2 complex (2^{nd} trough and peak) corresponding to the transition from frication to the onset of vowel (Tremblay, Friesen, Martin, & Wright, 2003). Hence, naturally produced speech stimulus can reliably be utilized to study the neural representation of speech at the cortical level in adults having normal hearing.

In peripheral hearing loss, the individual suffers from inaudibility (elevated threshold), reduced frequency selectivity, and normal or near normal temporal resolution depending on the degree of hearing loss. Hearing aid is one of the rehabilitative options to overcome these difficulties faced by individuals having hearing impairment. The hearing aid alters temporal parameters (Stelmachowicz, Kopan, Mace, Lewis and Nittrouer (1995), which are inherent in speech; and these, were represented neurally in the central auditory system. Therefore, it is important to study the effect of hearing loss and amplification on neural response pattern separately to understand the benefit of hearing aid received by individuals with hearing impairment.

Tremblay, Billings, Friesen, and Souza (2006) investigated the effect of hearing aid characteristics alone on the neural response pattern in cortical response in seven normal hearing individuals. The CAEPs were recorded in unaided and aided conditions to naturally produced speech syllables (/see/ and /she/). Each stimulus was presented at 64

dB peSPL. The gain of the hearing aid was 12 to 15 dB at 1 kHz, 17 to 19 dB gain at 2 kHz, and 24 to 26 dB at 3 kHz for an input of 60 dB SPL. The results revealed that consonant vowel boundary preserved by the hearing aid was detected neurally. This resulted in different neural response patterns for /see/ and /she/ stimulus. Further, the hearing aid with mild gain at the high frequencies altered the amplitude of aided waveform minimally relative to the unaided waveform. The subtle change between unaided and aided response was due to the presentation level of stimulus. In unaided condition, the response reach near level asymptote, where as in aided condition the response reach an asymptote due to the presentation level and gain of the hearing aid. However, many of the parameters were uncontrolled to generalize the findings like, gain of the hearing aid, presentation of input level used and method of presentation of stimuli.

Hemanth (2008) conducted a study on the effect of amplified speech on cortical response. The compression threshold of the hearing aid was kept at 75 dB SPL and the gain of the hearing aid was kept at 30 dB in each frequency. The output of the hearing aid was recorded from 2 cc coupler at 40 dB HL (below saturation condition) and at 90 dB HL (Saturation condition) using /si/ stimulus. The recorded output was presented at 50 dBnHL (non-asymptote) and 90 dBnHL (asymptote) respectively through ER 3A insert earphone. The results revealed that the onset and offset response of ACC had negligible effect on saturated and unsaturated conditions; and non-asymptote and asymptote conditions. At the transition portion of the response, clear detectable and increased amplitude was noticed in unsaturated stimulus condition than saturated condition. Hence, the ACC can be recorded reliably in individuals wearing with hearing aids. It would be

interesting to know the way the amplified speech is represented in impaired auditory pathway.

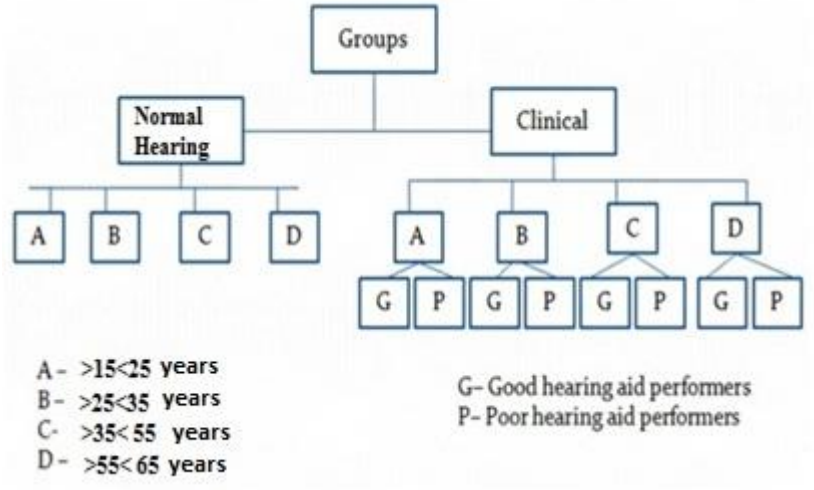
Tremblay, Billings, Friesen, and Souza (2006) investigated the neural representation of speech in the impaired auditory system in which hearing aid had been fitted to know the benefit that they receive from their amplification. Seven mild to severe sensorineural individuals were participated in the study. Two naturally produced speech /see/ and /shee/ were presented through the loud speaker. The ACC was recorded at 70 dBpeSPL for two naturally produced speech stimuli. The results revealed that CV transition appears to be earlier in the latency of 2N1 component of ACC for /shee/ than /see/ stimulus. The findings suggest that preserved speech cues after hearing aid processing was sufficient for the neurons to detect time varying cues in the impaired auditory system. The NAL-R prescriptive formula were formulated using 1/3 rd gain rule with respect to hearing loss in each octave frequency (0.25 kHz to 8 kHz) and takes overall loudness normalization into consideration. In real ear condition, the frequency gain response for each octave frequency was matched with the target prescriptive gain. However, for recording the cortical potentials, speech stimulus was used to study the hearing aid benefit. It was noted that there was a discrepancy between matching the gain to target prescription and the stimulus used to record the ACC. Hence, there is a need to optimize the hearing aid output to the speech stimulus and its representation at the cortical level. From the literature, it is well documented that LLR and ACC are a sensitive tools to detect the subtle change in an ongoing speech stimulus at the cortical level.

In the present study the output of the hearing aid at the ear canal was measured to know the extent of spectral and temporal alterations. Spectral information was measured using spectrogram and spectra. Temporal information was obtained from envelop difference index. This will throw light on processing parameter of hearing aid. Once after knowing the output of hearing aid, it is interesting to know its representation at brainstem and cortical level by predisposing factors such as aging and hearing loss. These two factors definitely altered the signal as we know from the review. The FFR, LLR and ACC were utilized as they which closely represent the inherent speech cues present in the stimulus. It was well reported in literature that among individuals with the same type, degree and configuration of hearing loss some individuals with hearing impairment benefit more than others who reject hearing aids. Thus, it would be interesting to know processing of acoustic cues after amplification. In addition, the research question arises on to what extent the hearing aid alleviates the problem faced by individuals with hearing impairment. This was studied by comparing the aided response obtained at different levels of auditory pathway from individuals with hearing impairment with the unaided response obtained from age matched normal individuals. If difference in encoding present even after fitted with hearing aid, than another research question arises on how does the amplified speech encoded in good hearing aid performers (GHP) differed from poor hearing aid performers (PHP).This was studied by comparing the response obtained from GHP and PHP at brainstem and cortical levels. To solve these research questions, the present study was taken up.

CHAPTER - 3 METHOD

The main aim of the study was to investigate the representation of amplified speech along the auditory pathway in individuals with sensorineural hearing loss. This study consists of clinical and normal hearing groups. Each group was further divided into four sub-groups, based on age. Each participant of clinical sub-group was either classified into good and poor hearing aid performers based on the acceptable noise level (ANL). In Phase 1, the participants were assigned to either normal hearing or clinical groups on the basis of the audiological evaluations. In Phase 2, the change in the signal at the output of the hearing aid was measured using probe tube microphone system at the ear canal of each participant in the clinical group. In Phase 3, the representation of speech syllables was studied at the brain stem level using FFR and at cortical level using LLR and ACC, in both clinical and normal hearing groups.

The study was carried out using a explorative, cross sectional, purposive stratified, one shot, pre-test post-test only clinical group with comparative research design. Figure 3.1 below provides a schematic representation of the relationship between the independent, dependent and control variables used in the study.



Independent variables

1. Groups (Clinical and normal hearing) and sub-groups ('A', 'B', 'C', 'D')
2. Good and poor hearing aid performers
3. Speech stimuli /da/ and /si/

Dependent variables

1. At the level of the ear canal–
   Spectral : F_0, onset of transition (F_1 and F_2), offset of transition (F_1 and F_2) and spectra in both unaided and aided conditions for each stimulus.
   Temopral – EDI for each stimulus.
2. At the level of auditory brainstem: Slope (V-A), F_0, F_0 energy, and F1 energy to each stimulus
3. At the level of the auditory cortex: Slope (N1-P2) for /da/ stimulus, latency and amplitude of N1 and P2 (Onset of consonant) and 2N1 and 2P2 (Onset of vowel) for /si/ stimulus.

Control variables

1. Test environment
2. Hearing ability of participants (degree of hearing loss)
3. Digital BTE hearing aid

Figure 3.1. Schematic representation of grouping of participants and independent, dependent and control variables in the study.

Participants

In total, 100 participants were included in the study. They were classified into two groups, i.e., clinical group and normal hearing group. The clinical group comprised of individuals with sensorineural hearing impairment.

Clinical group. Sixty clients registered at All India Institute of Speech and Hearing, Mysore, were recruited for the study after obtaining a written informed consent (format enclosed in Appendix I). The following criteria were used in the process of recruiting the participants. The participants of wider age range from 15-65 years were sub-grouped into four. The each sub-group was assigned into 'A', 'B', 'C' or 'D'. The demographic data of each participant in clinical group are tabulated in Table 3.1.

1. Age was ranging from 15 to 65 years. The participants were sub-grouped into four categories based on age of the participants. Group 'A' (N=6) comprised of participants of >15 to < 25 years (mean age = 19 years; range – 16 to 21 years); Group 'B' (N=7) comprised of participants; > 25 to < 35 years (mean age = 29.4 years; range – 26 to 32 years); Group 'C' (N=19) comprised of participants of > 35 to < 55 years (mean age = 49 years; range – 44 to 53 years); and Group 'D' (N=28) comprised of participants of > 55 to < 65 years (mean age = 61 years; range 59- 63 years).

2. Ear canal of the test ear was free from cerumen, debris, foreign body or any infection.

3. The test ear had sensorineural hearing loss. The pure tone average (PTA) obtained at frequencies 0.5 kHz, 1 kHz and 2 kHz was between 26 and 55 dB HL. The test

ear had a flat configuration of audiogram. Flat audiogram configuration being operationally defined as the difference between the lowest and highest air-conduction thresholds from 0.25 kHz to 8 kHz being less than 20 dB (Pittman & Stelmachowicz, 2003). The pure tone thresholds across frequencies for air-conduction (0.25 kHz to 8 kHz) and bone-conduction (0.25 kHz to 4 kHz) in each clinical sub-group is shown in Figure 3.2. The three frequency pure tone average in four sub-groups was between 40 dB HL and 55 dB HL. The mean and standard deviation of pure tone average (PTA) was 54.7±5.11 in sub-group 'A'; 49.77±6.22 in sub-group 'B'; 49.53±6.1 in sub-group 'C' and 48.97±6.63 in sub-group 'D'. Furthermore, the threshold at each frequency from the participants of clinical sub-group 'A' met the assumption of normal distribution from Kolmogorov-Smirnov test normality test ($p > 0.05$). Similar results were noted in the other clinical sub-groups. Further, the threshold at each frequency between sub-groups were homogeneous in the Levene's homogeneity test ($p>0.05$). Hence, MANOVA was performed to evaluate difference in threshold (both air-conduction and bone-conduction) between sub-groups at each frequency. The results revealed that there was no significant difference in threshold at each frequency between sub-groups.

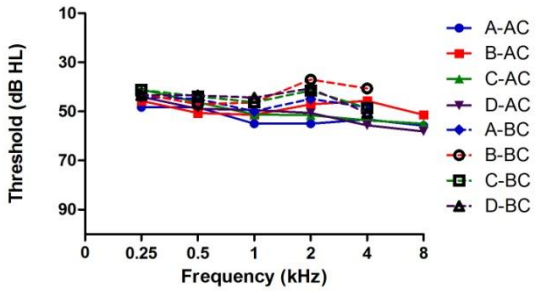


Figure 3.2. The mean pure tone thresholds across frequencies for air-conduction (AC) (0.25 kHz to 8 kHz) and bone-conduction (--- BC) (0.25 kHz to 4 kHz) in each clinical sub-group ('A', 'B', 'C', & 'D').

4. Speech Identification Scores (SIS) at 40 dB SL (re: Speech Reception Threshold, SRT) was greater than or equal to 75%. The SIS (Max. Score being 25) in each clinical sub-group is shown in Figure 3.3. The mean and standard deviation of SIS was 21.30 ±1.86 in sub-group 'A'; 20.57±1.98 in sub-group 'B'; 22.15 ±1.95 in sub-group 'C'; and 22.33 ± 2.17 sub-group 'D'. The SIS from the participants of each clinical sub-group met the assumption of normal distribution from Kolmogorov-Smirnov normality test ($p > 0.05$) and homogeneous between groups in the Levene's test of homogeneity ($p>0.05$). Hence, the ANOVA was performed. The results revealed that there was no significant difference in SIS between groups.

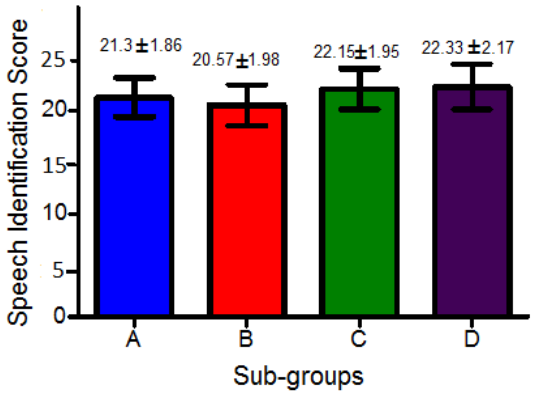


Figure 3.3. Mean and standard deviation of Speech Identification Score (Max. = 25) in each clinical sub-group.

5. Each participant had normal middle ear function as indicated by the middle ear analyzer. The middle ear peak pressure ranged from + 50 da Pa to -100 daPa, and the admittance ranged from 0.5 ml to 1.75 ml with the probe frequency of 0.226 kHz. The acoustic reflexes were present, at least in 0.5 kHz and 1 kHz.

6. The latency difference of V peak of auditory brainstem responses was less than 0.8 ms, between the repetition rates of 11.1/sec. and 90.1/sec., at 90 dBnHL (Don, Allen, & Starr, 1977).

7. All of them were naïve hearing aid users.

8. The clinical sub-groups were further classified into good and poor hearing aid performers using acceptable noise level (ANL). The participants with an ANL score of <7 dB were considered as good performers; those with a score of >13 dB were considered as poor performers; and those with an ANL score ranging between 8 and 12 were average performers (Nabelek, Freyaldenhoven, Tampas, Burchfield, & Muenchen, 2006). Participants with an ANL between 7 and 13 were not considered in the present study.

9. All the participants were native speakers of Kannada. They had acquired hearing loss with adequate speech and language.

10. They had no complaint of psychological or cognitive or neurological problems

Table. 3.1

Demographic data of clinical sub-groups

SN	*Age (yrs)*	*Gender*	*Ear*	*Duration of HL (yrs)*	*PTA dB HL*	*SIS (Max. 25)*	*V peak latency difference at two repetition rates*	*ANL*
Sub-group 'A' (N=6; Mean = 19 yrs , SD= ±3.28)								
1	15.00	F	R	10.00	58.30	20.00	0.38	GHP
2	18.00	M	L	1.00	45.00	21.00	0.44	GHP
3	18.00	F	R	2.00	56.60	20.00	0.35	GHP
4	19.00	M	R	7.00	48.30	21.00	0.46	GHP
5	19.00	M	L	1.00	53.30	25.00	0.27	GHP
6	25.00	F	R	2.00	55.00	21.00	0.46	PHP
Sub-group 'B' (N=7; M = 29.4 yrs, SD= ± 3.50)								
7	27.00	M	L	0.17	50.00	18.00	0.65	PHP
8	27.00	F	R	0.17	51.60	18.00	0.55	PHP
9	27.00	M	R	6.00	51.60	21.00	0.25	PHP
10	32.00	M	R	2.00	53.30	20.00	0.41	GHP
11	32.00	M	R	0.50	55.30	22.00	0.33	PHP
12	35.00	M	R	1.50	51.60	23.00	0.56	GHP
13	26.00	F	R	20.00	55.00	22.00	0.30	GHP
Sub-group 'C' (N=19; Mean = 49 yrs, SD= ± 5.83)								
14	37.00	M	L	2.00	51.60	21.00	0.34	PHP
15	40.00	F	L	2.00	50.00	20.00	0.30	GHP

16	41.00	M	L	1.00	50.00	20.00	0.27	PHP
17	42.00	M	L	10.0	50.00	25.00	0.37	PHP
18	43.00	M	L	4.00	55.00	19.00	0.25	GHP
19	47.00	F	R	4.00	55.60	23.00	0.38	GHP
20	47.00	M	R	3.00	46.60	19.00	0.37	PHP
21	49.00	M	R	4.00	53.30	20.00	0.38	GHP
22	50.00	F	R	1.50	53.30	23.00	0.30	PHP
23	50.00	M	L	1.00	45.00	25.00	0.55	GHP
24	53.00	M	L	3.00	45.00	21.00	0.22	PHP
25	54.00	F	R	0.08	51.60	24.00	0.39	PHP
26	54.00	M	R	3.00	48.30	24.00	0.28	GHP
27	54.00	M	L	0.08	41.00	23.00	0.30	GHP
28	54.00	M	L	1.00	41.60	23.00	0.57	GHP
29	55.00	F	L	4.00	55.00	21.00	0.24	PHP
30	55.00	F	R	0.33	53.30	23.00	0.50	GHP
31	55.00	F	L	0.33	50.00	23.00	0.58	GHP
32	51.00	M	L	1.00	45.00	24.00	0.50	GHP
Sub-group ‘D’ (N=28; Mean= 61yrs , SD= ± 2.60)								
33	56.00	F	L	0.17	46.60	23.00	0.24	GHP
34	58.00	M	R	1.00	45.00	24.00	0.34	GHP
35	58.00	M	L	1.00	48.30	24.00	0.32	GHP
36	58.00	M	R	0.08	45.00	24.00	0.24	GHP
37	58.00	M	L	0.08	45.00	24.00	0.34	GHP
38	60.00	F	R	1.00	41.60	21.00	0.32	GHP
39	60.00	F	L	1.00	53.30	21.00	0.22	GHP
40	60.00	F	R	4.00	45.00	24.00	0.54	PHP
41	60.00	M	R	1.00	55.00	18.00	0.43	PHP
42	60.00	M	L	1.00	43.30	25.00	0.55	GHP
43	60.00	M	R	1.00	46.30	24.00	0.37	PHP
44	60.00	M	L	1.00	46.30	24.00	0.23	PHP
45	60.00	M	R	5.00	58.30	19.00	0.34	PHP
46	61.00	F	L	1.50	55.00	23.00	0.44	GHP
47	61.00	F	R	1.50	58.30	24.00	0.36	GHP
48	61.00	M	R	2.00	41.60	24.00	0.70	PHP
49	61.00	M	R	6.00	55.00	21.00	0.42	PHP
50	62.00	M	R	1.00	55.00	19.00	0.26	GHP
51	62.00	M	L	4.00	51.60	25.00	0.28	PHP
52	62.00	F	R	1.00	50.00	24.00	0.34	PHP
53	64.00	M	L	1.00	45.00	19.00	0.23	PHP
54	64.00	F	L	2.00	55.30	22.00	0.28	GHP
55	65.00	M	L	1.00	51.60	21.50	0.28	PHP
56	65.00	M	R	3.00	55.00	24.00	0.32	GHP
57	65.00	M	R	1.00	46.60	21.50	0.30	GHP
58	65.00	M	R	3.00	43.30	20.00	0.35	GHP
59	65.00	M	R	1.00	50.00	18.50	0.68	GHP
60	65.00	F	R	1.00	56.60	24.00	0.45	PHP

Note : HL = Hearing loss, PTA = Pure tone audiometry, SIS= Speech identification scores, ANL= Acceptable noise level, F= Female, M= Male, R= Right ear, L= Left ear, GHP= good hearing aid performers, PHP= Poor hearing aid performers.

Normal hearing group. A total of 40 adults with normal hearing were included in this group. The participants of normal hearing group were sub-grouped based on age range. The age range of participants was >15 to < 25 years (mean age = 20 years; range 18- 22 years) in Group 'A' (N=10); >25 to <35 years (mean age = 30 years; range 27 – 33 years) in Group 'B' (N=10); > 35 to < 55 years (mean age = 46 years; range 40- 52 years) in Group 'C' (N=10); and > 55 to < 65 years (mean age = 62 years; range 58- 64 years) in Group 'D' (N=10). There were ten participants in each of these sub-groups. The mean pure tone average (PTA) was 10 dB HL in sub-group 'A'; 6.6 dB HL in sub-group 'B'; 11.6 dB HL in sub-group 'C' and 16.3 dB HL in sub-group 'D'. All the participants had normal middle ear status with 'A' type tympanogram with measurable ipsi-lateral and contra-lateral reflexes at 0.5 kHz, 1 kHz, 2 kHz and 4 kHz. All the participants were native speakers of Kannada. All the participants had adequate speech and language skills. None of the participants had any complaint of neurological, psychological, cognitive or otological problems. Written informed consent was obtained from each participant (Appendix II).

Test environment

All the tests were conducted in air-conditioned, sound treated single or double room suite with noise levels within permissible limits (ANSI, S3.1-1991).

Instrumentation

The following instruments were used to record the stimuli and collect the data.

1. A calibrated clinical audiometer (Madsen OB922, version 2.64) with headset having TDH 39 earphones enclosed in MX-41/AR supra-aural ear cushions to estimate the air-conduction thresholds, SRT and SIS; and Radio Ear B-71 bone vibrator to estimate the bone-conduction thresholds. To estimate the ANL, Martin Audio loudspeaker was used to deliver the Kannada passage and speech noise to determine MCL and BNL.
2. A calibrated Grason-Stadler TympStar (version 2) middle ear analyzer was used to evaluate the status of the middle ear.
3. A personal computer-based Neuroscan 4.4 (Stim 2-version 4.4; and Scan 2-version 4.4) instrument was used to present the /ɖa/ and /si/ stimulus separately through a calibrated loud speaker (DB technology), and to record the Frequency Following Response (FFR), Late Latency Response (LLR) and the Acoustic Change Complex (ACC). Silver chloride (AgCl) electrodes were used to record the FFR, LLR and ACC in both unaided and aided conditions.
4. Fonix 7000 hearing aid test system (version 1.63 from M/s. Frye Electronics, USA) was used to measure electroacoustic characteristics of the test hearing aid in order to confirm the working of the hearing aid according to the specifications given by the manufacturer. This instrument was also used for probe tube microphone measurement and to optimize the hearing aid gain.
5. In order to program the test hearing aid, NOAH software (version 3) and hearing aid specific software installed in the personal computer were utilized. A HiPro was connected to this computer for providing an interface between the computer and the hearing aid to be programmed.

6. Adobe Audition software (version-3) and a unidirectional microphone (Ahuja, AUD-101XLR) with the frequency response ranging from 0.05 kHz to 10 kHz was used to record the naturally produced speech syllables on to a computer. The recorded speech stimuli were scaled and normalized to obtain the evoked potentials, viz., FFR, LLR and ACC.
7. Praat software (version5.1.29) was used to note the fundamental frequency (F_0), formant transition duration and extent of first formant (F_1) and second formant (F_2) transitions in each of the speech stimulus (/ḍa/ and /si/). This software was also used to record and analyse the unaided and aided stimuli at the ear canal of each participant.
8. Matrix laboratory (MATLAB) (7.9.0. 529. R2009b version) was used as platform to run the 'm-file' of Envelope difference Index (EDI) (Fortune, Woodruff, & Preves, 1994). The EDI quantifies the temporal envelope contrasts between unaided and aided speech stimulus (/ḍa/ and /si/) recorded at the ear canal of each participant.
    a. m-file of autocorrelation was used to analyze the FFR recorded at the auditory brainstem level in terms of F_0 in both unaided and aided conditions to each stimulus (/ḍa/ and /si/)
    b. 'Brainstem Toolbox' (Skoe & Kraus, 2010) was used to compute F_0 energy and F_1 energy.
9. Each test ear of the participant was fitted with a monaural eight-band, two-channel digital Behind-The-Ear (BTE) hearing aid coupled with custom made soft earmould in the test ear. This was used to obtain spectral and temporal measurement at ear canal, aided FFR at brainstem level, LLR and ACC at cortical level. The compression circuit was switched 'on', and the omni-directional

microphone was enabled. The volume control wheel was disabled so that the participant could not manipulate the amount of gain provided by the hearing aid during the data collection. No other signal processing algorithms of the hearing aid (i.e., noise reduction and feedback suppression circuit) were active during testing. The test hearing aid had a fitting range from mild to moderately severe degree of hearing loss.

Stimulus preparation

Three male speakers whose mother tongue was Kannada were chosen to utter the two test stimuli. Sridhar (1990) defines Kannada as one of the four major literary languages of the Dravidian family, spoken by over 30 million people, primarily in the state of Karnataka (formerly Mysore), South India. Two speech syllables uttered with a normal vocal effort were utilized as test stimuli. The test stimuli included a voiced retroflex plosive with a low back vowel, i.e., /ɖa/; and an unvoiced alveolar fricative consonant with high front vowel, i.e., /si/.

A total of six consonant-vowel combinations, i.e., /ɖa/ and /si/ stimuli uttered by three male speakers, were recorded using the Adobe Audition software (version-3) installed in the personal computer via the recording microphone (Ahuja, AUD-101XLR) placed at a distance of 10 cm from the lips of the speaker (Winholtz & Titze, 1997). The recorded stimuli were digitized using a 32-bit processor at 44,100 Hz sampling frequency. A goodness test was performed in order to select one set (/ɖa/ and /si/) of the test stimuli, in which ten listeners with normal hearing rated for the naturalness of these stimuli. Two stimuli /ɖa/ and /si/ produced by the same speaker,

which were rated as being natural by 8 of the 10 listeners with normal hearing, were selected as the test stimuli. The duration of /ɗa/ and /si/ stimuli was 94 ms and 301 ms respectively. For the syllable /ɗa/, the voice onset time (VOT) was 18 ms, the burst duration was 5 ms, transition duration was 37 ms, and vowel duration was 34 ms. The voice onset time of /ɗa/ stimulus was edited to have 18 ms. The middle portion of VOT was cropped using pitch pulse method (Boersma & Weenink, 2014). This was done to elicit the synchronous onset response at the auditory brainstem level. The syllable /si/ had the fricative duration of 159.3 ms, with a transition duration of 47.1 ms, and vowel duration of 94.6 ms.

The stimuli were converted from '.wav' to '.avg' format. The '.avg' format of each stimulus was band pass filtered from 0.10 kHz to 1.5 kHz using Neuroscan (Scan 2-version 4.4). The rationale behind converting the '.wav' to '.avg' format is because the naturally produced stimulus was being presented to elicit FFR, but the band pass filter was used for recording FFR was 0.10 kHz to 1.5 kHz. In addition, the functional relationship between the acoustic structure of speech and the brain stem response to speech can be established. The stimulus.avg waveform and spectrogram of both the stimuli are depicted in Figure 3.4. The F_0, F_1 and F_2 in the onset and end of transition of original and filtered versions of both /ɗa/ of /si/ stimuli were measured using Praat software (version -5.1.29). This is tabulated in Table 3.2.

Further, three female speakers were chosen to read out a Kannada passage (Sairam, 2002) in normal vocal effort. The recorded Kannada passage by these three speakers was digitized using a 32-bit processor at 44.1 kHz sampling frequency in

Adobe Audition software (version-3). A goodness test was performed in order to select one of the three Kannada passages. For this, ten listeners with normal hearing rated the recorded passage for naturalness. The passage that was rated as being natural was selected to determine the ANL.

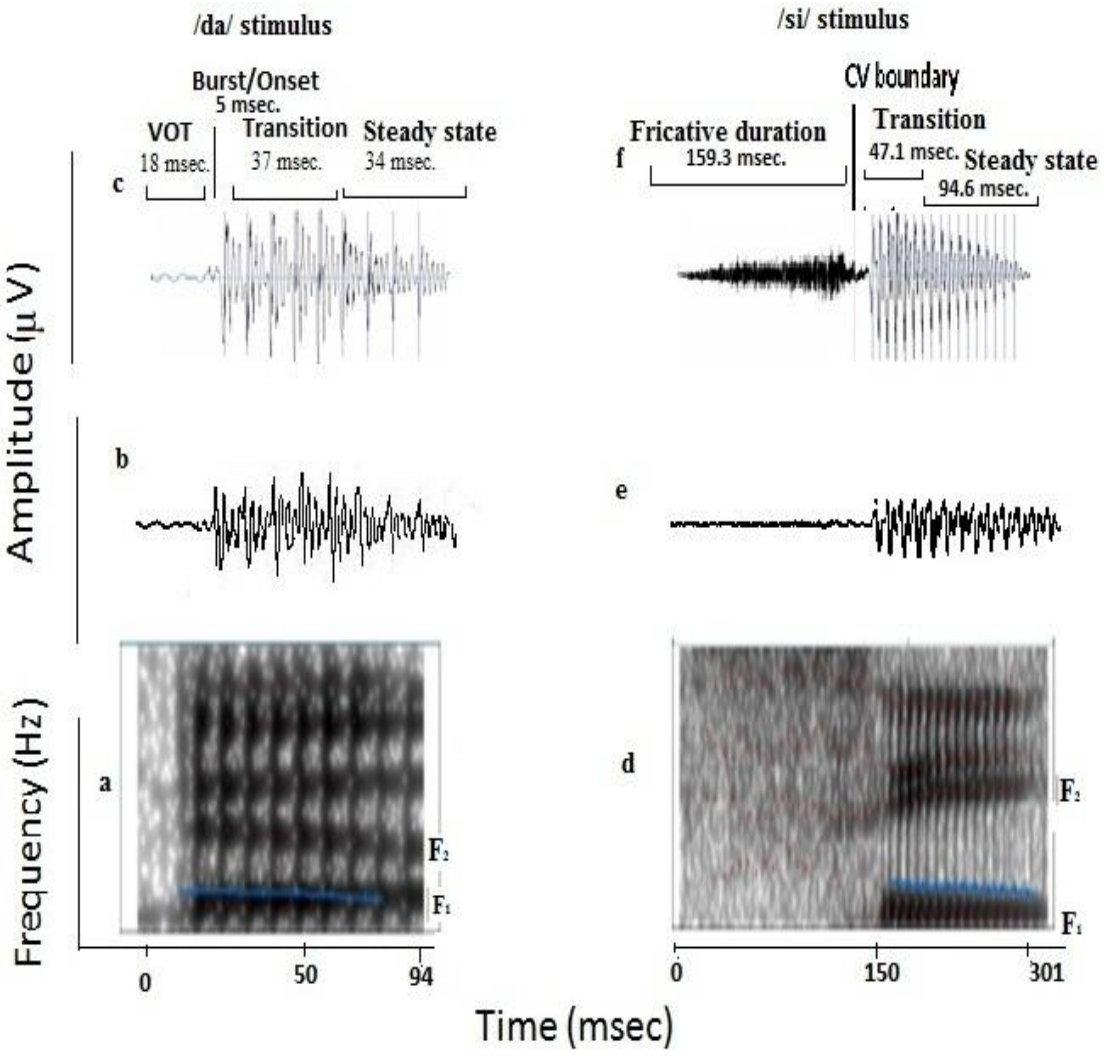


Figure 3.4. a) and d) are the spectrogram of /ɗa/ and /si/ stimuli. The blue solid line in both stimuli indicates the F_0, which has a falling pattern. The F_1 of /ɗa/ stimulus is in raising pattern and F_2 is in falling pattern. The F_1 of /si/ stimulus is in falling pattern and F_2 is in raising pattern.
c) and f) are the waveforms of /ɗa/ and /si/ stimuli. For syllable /ɗa/, the voice onset time was 18 ms, the burst duration was 5 ms, the transition duration was 37 ms and vowel duration of 34 ms. For the syllable /si/, the fricative duration was 159.3 ms, transition duration was 47.1 ms and vowel duration was 94.6ms
b) and e) are stimulus.avg waveforms of the speech stimuli /ɗa/ and /si/.

Table.3.2

Fundamental frequency (F_0 in Hz) and the two formant frequencies (F_1 and F_2 in Hz.) at the transition of original and filtered version of /ɗa/ and /si/ stimuli

Stimuli		*F_0(Hz)*	*F_1(Hz)*		*F_2(Hz)*	
			Onset of formant transition	*Onset of steady state*	*Onset of formant transition*	*Onset of Steady State*
Original version	/ɗa/	136	520	556	1822	1678
	/si/	146	345	309	2268	2452
Filtered version	/ɗa/	136	517	556		
	/si/	146	345	309		

Note: F_2 was not there for the filtered version, as the band pass filter used was 0.10 kHz to 1.5 kHz.

Justification of Stimuli

Dubno and Levitt (1981) have opined that the Consonant Vowel (CV) speech syllables are less likely to be confused compared to the Vowel Consonant (VC) combinations. Wang and Bilger (1973) and de Gelder and Vroomen, (1998) reported that the stops and fricatives in initial position are more vulnerable to errors compared to other classes of speech syllables. In addition, the voiced stop consonants showed more errors than the voiceless. It could be because the power spectrum of /b/ is relatively less than other stop voiceless consonants (Sanders, 1971). Yet another reason could be older adults unable to fire synchronously to the voice onset time (VOT) of voiced consonants, as it has lower amplitude and more likely chance of perceiving it as unvoiced consonants (Tremblay, Piskosz, & Souza, 2003).

In the present study, a stop consonant with a vowel combination and a sibilant consonant with a vowel combination were included as stimuli, in order to investigate the representation of these stimuli at different levels of the auditory system. Among stop consonants, retroflex /ɗa/ has been found to be more complex for recognition (Dorman, Marton, Hannley, & Lindholm, 1985) due to the abruptness in frequency change. In addition, stimulus /ɗa/ has rapid temporal changes and complex spectral distributions which are inherent in speech (Cunningham, Nicol, Zecker, & Kraus, 2001). Walley and Carrell (1983) reported that formant frequency is the primary cue for the identification of stop consonant in different vowel contexts such as /a/, /i/ and /u/. However, testing with multiple vowel contexts consumes time which is impractical in clinical and research applications. In order to discuss the findings of the present study in relation to that of literature (Johnson, Nicol, & Kraus, 2005; Russo, Nicol, Masacchia, & Kraus, 2004; Hemanth & Manjula, 2012), the vowel context /a/ was included in the study. Yet another reason for selecting /ɗ/ combined with vowel context /a/ is because of closely spaced frequencies of first two formants F1 and F2 in the onset of transition compared to other vowels (Liberman, 1957). This closely spaced spectral content of information might be difficult for older adults to perceive.

In the second stimulus i.e., /si/, the sibilant /s/ was included because of its high frequency content. The vowel /i/ was included as the energy was present up to 6 kHz, compared to /u/ and /a/ contexts (Boothroyd & Medwetsky, 1992). Whalen (1991) reported that vowel formant transition contributes for the perception of /s/ in individuals with normal hearing. However, individuals with hearing impairment relay more on fricative noise rather than on vowel formant transition (Zeng & Turner, 1990) and require wider bandwidth (Stelmachowicz, Pittman, Hoover, & Lewis,

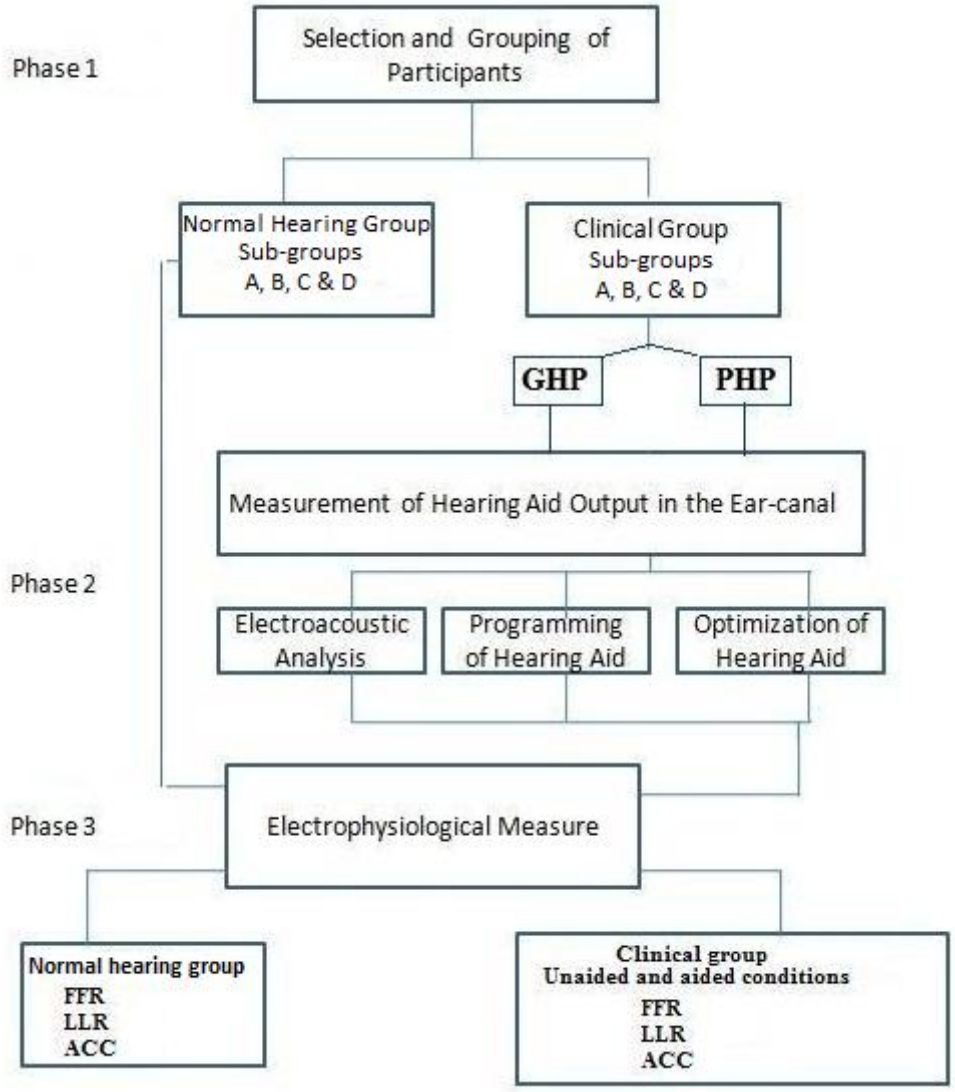
1995). Unfortunately, due to hearing loss the fricative noise and / or formant transition become inaudible and hence difficult to perceive (Dubno & Dirks, 1982).

To convey the information of fricative noise to the individuals with hearing impairment, the hearing aid output transducer should have wider bandwidth. If higher output is desirable in the high frequency region of a hearing device, there is a higher chance of distortion and acoustic feedback. This is due to the limit in the frequency response of the transducer used in hearing devices. This might have an effect on the perception of speech syllables such as /si/ in individuals with hearing impairment. Thus, in the present study, the CV speech syllables, in which both voiced and unvoiced consonant combined with vowel, were utilized as target test stimuli. The stimuli were produced naturally to determine how the time varying acoustic cues in speech syllables are represented at ear canal and the available acoustic cues are tuned into neural code at the brainstem and cortical levels. In addition, the available acoustic cues at different levels of auditory pathway help to understand the complete circuit in which acoustic cues are neurally represented.

Procedure

The study was carried out in three phases. Phase 1 was carried out for the selection of participants; and also to classify good and poor hearing aid users. Phase 2 was carried out for measuring the hearing aid output in the ear canal and Phase 3 was to investigate the representation of speech stimuli at the brainstem and cortical levels of the auditory system. The Phases 1 and 3 were administered for both clinical group and normal hearing group, whereas Phase 2 was administered only on participants

from the clinical group. Block diagram of sequence of phases in the study is shown in Figure 3.5.



Note: GHP - Good Hearing aid Performers; PHP - Poor Hearing aid Performers; FFR - Frequency Following Response; LLR - Late Latency Response; and ACC - Acoustic Change Complex.

Figure 3.5. Sequence of the three phases in the study.

Phase 1: Selection and grouping of participants. This study consists of two groups viz., clinical group and normal hearing group. Each group was further divided into four sub-groups, based on age. Each participant of clinical sub-group was either classified into good (ANL<7) and poor (ANL>13) hearing aid performers based on

the acceptable noise level (ANL). Each participant underwent a routine hearing evaluation which included pure tone hearing assessment at 0.25 kHz, 0.5 kHz, 1 kHz, 2 kHz, 4 kHz and 8 kHz; speech identification test and immittance evaluation.

1. Air-conduction and bone-conduction thresholds were determined using modified Hughson-Westlake procedure using a +5 dB and -10 dB step-size (Carhart & Jerger, 1959). If indicated, the non-test ear was effectively masked with narrow band noise (Liden, Nilsson, & Anderson, 1959).
2. The speech identification test was administered using the recorded speech material (Yathiraj & Vijayalaksmi, 2005) presented through the auxiliary input of the audiometer at a level of 40 dB SL (re: speech reception threshold, SRT). Each participant was instructed to repeat the word heard. If indicated, the non-test ear was masked (Konkle & Berry, 1983). The total numbers of correctly identified words were noted down to calculate the speech identification scores (SIS). The resultant score was retained and not converted to percentage.
3. Tympanometry was carried out using a 226 Hz probe tone and a change in air pressure rate of 200 daPa /s.
4. Ipsilateral and contralateral acoustic reflex thresholds were measured at 0.5 kHz, 1 kHz and 2 kHz by varying the intensity of stimulus in 5 dB-steps to observe changes in acoustic admittance.

In addition to the above tests, a detailed diagnostic hearing evaluation was administered on the participants of the clinical group. The protocol included -

1. Auditory brain stem response was recorded to clicks at two repetition rates, 11.1/sec and 90.1/sec, at 90 dBnHL. All the participants had a difference of less

than 0.8 ms in V peak latency between the waveforms obtained at the two repetition rates. This was used to rule out retro cochlear pathology (RCP).

2. Acceptable noise level (ANL) evaluated the reaction of the listener to background noise while listening to speech. For the measurement of ANL, the method given by Nabelek, Tucker, and Letowski (1991) was adopted. The participant was made to sit comfortably on a chair in front of the loudspeaker of the audiometer that was located at 1 m distance and 45^0 Azimuth from the participant.

Instructions given for establishing the most comfortable level (MCL) of the participant was "You will listen to a story through the loudspeaker. The loudness of the story will be varied. First, the intensity will be turned up until it is too loud and then turned down until it is too soft. Then the level is adjusted. You have to indicate the level at which the loudness of the story is most comfortable for you". The recorded Kannada passage was routed though the auxiliary input of the audiometer to the loudspeaker at the level of SRT, which had been determined at the time of audiological assessment. Gradually, the level was adjusted in 5 dB-steps up to the level of Most Comfortable Level (MCL) and then in smaller steps size of +1 and -2 dB, until the MCL of the participant was established reliably. These steps were repeated again and the average of the MCL obtained from the two measures was taken as the MCL for the participant.

After the MCL was established, a speech noise was introduced at 30 dB HL. The level of the speech noise was increased in 5 dB-steps initially, and then in 2 dB-steps, to a point at which the participant was willing to accept the noise without becoming tired or fatigued while listening to and following the passage. The maximum level at which he/she could accept or put up with the noise without

becoming tired was considered as the Background Noise Level (BNL). The level of the speech noise was adjusted until the participant was able to 'put-up-with' the noise while following the story. These steps were repeated again and the average of the BNL obtained from the two measures was taken as the BNL for the participant.

Table 3.3 gives the values of mean and standard deviation of the most comfortable level (MCL), background noise level (BNL) and acceptable noise level (ANL) in good and poor hearing aid performers of each sub-group. It was noticed that in each sub-group, the mean MCL was obtained at a lower level in good hearing aid performer (GHP) than a poor hearing aid performer (PHP). The mean BNL obtained was higher in GHP than in PHP. Hence, the ANL score obtained was higher in PHP. In sub-group 'A' of PHP, the mean ANL was 14 dB as the MCL was 67 dB HL and the BNL was 53 dB HL (i.e., MCL-BNL=ANL).

Table 3.3

The values of most comfortable level (MCL), background noise level (BNL) &acceptable noise level (ANL) in good and poor hearing aid performers in each sub-group

Sub-groups	*Hearing aid performers*	*MCL (dB HL)*	*BNL(dB HL)*	*ANL(dB HL)*
		Mean±SD	*Mean±SD*	*Mean±SD*
'A'	GHP (N=5)	65.60±1.90	60.80±2.48	3.00±1.54
	PHP (N=1)	-	-	-
'B'	GHP (N=3)	60.00±4.00	54.00±4.00	3.00±1.62
	PHP (N=4)	69.70±2.50	53.25±3.68	16.45±0.95
'C'	GHP(N=11)	61.18±6.12	56.36±5.86	3.45±1.57
	PHP(N=8)	69.00±7.46	54.21± 6.71	14.75±1.75
'D'	GHP(N=16)	67.75±6.51	60.93±5.39	3.56±1.54
	PHP(N=12)	69.75±9.16	57.41±7.10	14.58±0.99

Note: 1. GHP = Good hearing aid performers; PHP = Poor hearing aid performers;

2. As the N was 1 for PHP in sub-group 'A', statistics was not conducted. The MCL, BNL and ANL of this participant were 67 dB HL, 53 dB HL and 14 dB HL, respectively.

The ANL quantifies the acceptable level of background noise and is calculated as the difference between MCL (dB HL) and BNL (dB HL) (Nabelek, 2005). Based on the scores of ANL, each participant of clinical group was classified as good (<7) and poor (>13) performers with the hearing aid. Participants with an ANL between 8 and 12 were not considered in the present study. The value of the MCL, BNL and ANL between sub-groups in GHP/PHP met the assumption of normal distribution from Kolmogorov-Smirnov normality test ($p > 0.05$) and Levene's homogeneity test ($p>0.05$). To check if there was any significant difference in the MCL between sub-groups, ANOVA was carried out separately in GHP and PHP. The results revealed that there was no significant difference in MCL between sub-groups of GHP and PHP. Similar results were noted in BNL and ANL.

Phase 2: Measurement of the output of the hearing aid in the ear canal.

Participants from Clinical group alone were involved in Phase II. Prior to the measurement of the output of the hearing aid in the ear canal, electro- acoustic characteristics of hearing aid were measured to confirm the working status of the test hearing aid that was programmed for maximum output. The test hearing aid was then programmed according to the hearing loss of the participant. The NAL-NL1 prescriptive procedure was used along with an acclimatization level 2. Further, the hearing aid was optimized to match with the target gain during insertion gain measurement (IGM) and then the aided SIS was obtained to evaluate the benefit from the hearing aid.

Electroacoustic analysis of testing hearing aid. Electroacoustic measurement was carried out to verify the working condition of test hearing aid, to see if it confirms to the manufacturer's specification. Fonix 7000 was used for the purpose. The calibrated system was leveled. The test microphone was connected to one end of the 2 cc coupler (HA-2). The test BTE hearing was connected to the other end of the coupler through an adapter. The hearing aid was given a constant power supply of appropriate voltage using the battery substitution pill. For the digi speech stimulus presented at 65 dB SPL, the frequency range of the hearing aid extended from 0.21 kHz to 6.5 kHz. The peak full-on gain was 58 dB and high-frequency average full-on gain was 49 dB. The equivalent input noise was 10 dB SPL. Further, the attack and release time were 10 ms. and 30 ms. respectively for an input of 2 kHz tone .Thus, it was ensured that the test hearing aid conformed to the manufacturer's specifications. The functioning of the hearing aid was ensured at the beginning of the data collection and repeated every three months till the completion of data collection. The same instrument (Fonix 7000) was used to carry out the probe tube microphone measurements. Figure 3.6 depicts the electroacoustic measurement.

Figure 3.6. The output of hearing aid, being picked up by the microphone through the coupler and adapter. This is fed to Fonix 7000 hearing aid analyzer for electroacoustic measurement (Reprinted with permission from Frye electronics, Inc.).

Programming of test hearing aid using NAL-NL1 prescriptive procedure.

Each participant in the clinical group was fitted with the digital BTE hearing aid using a custom made soft ear mould. The hearing was connected to the personal computer through the hardware interface, HiPro (Hearing Instrument Programmer). The NOAH software and the hearing aid programming software were utilized for programming the hearing aid, using the following steps.

1. NOAH software (version 3) was selected and the participant's demographic data were entered.
2. Audiometric pure tone thresholds (at octave intervals from 0.25 kHz to 8 kHz for air- conduction and from 0.25 kHz to 4 kHz for bone-conduction) of the test ear of each participant were fed into the NOAH software using the audiogram module. This data were saved.
3. The hearing aid was connected to the HiPro which in turn was connected to a computer in which the NOAH and hearing aid specific software (s) are installed.
4. After the hearing aid was detected through the hearing aid programming software, the option of 'first fit' was selected for programming. The hearing aid was programmed using NAL-NL1 prescriptive formula at an acclimatization level of 2.
5. The volume control and other algorithms (noise reduction, feedback management) in the hearing aid were disabled.

Optimization of hearing aid gain using insertion gain measurement and audibility of Ling's six syllables. Each participant was made to sit comfortably on a chair. The loud speaker of the Fonix 7000 hearing aid test system was positioned 12 inches away from the test ear of the participant at an angle of 45^0 Azimuth. The

standard loud speaker distance and angle from the test ear in the measurement of real ear measurement is depicted in Figure 3.7. Initially, the hearing threshold of the test ear was fed into the audiogram module of the Fonix 7000 Real Ear Measurement (REM) device. The uncomfortable loudness level was predicted by the device for each test frequency (0.25 kHz to 8 kHz). The NAL-NL 1 fitting rule was selected to obtain the target gain required for each participant.

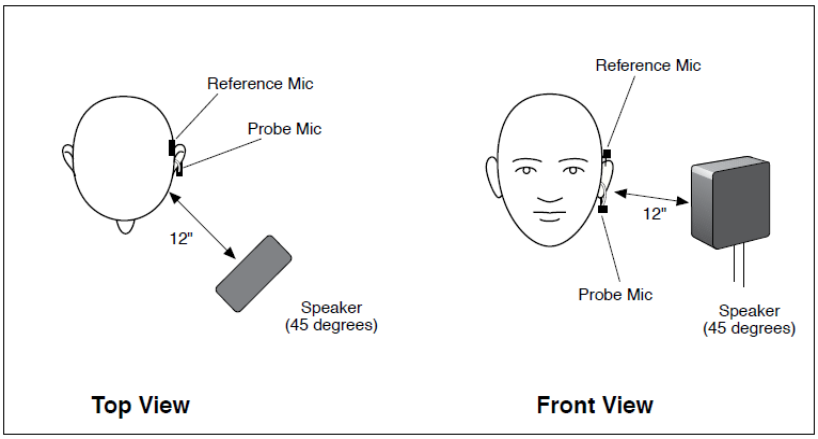


Figure 3.7. Insertion gain measured at the ear canal using Digi speech delivered through the loudspeaker, which was kept at the distance of 12 inches with 45^{0} Azimuth from the participant (Reprinted with permission from Frye electronics, Inc.).

The probe tube was detached from the integrated probe microphone of Fonix 7000 set and placed on a flat surface along side of custom made soft ear mould of the participant. The probe tube was marked with the tube extending atleast 5 mm past the canal length and attached back to the integrated probe microphone set. The probe tube marking is depicted in Figure 3.8.

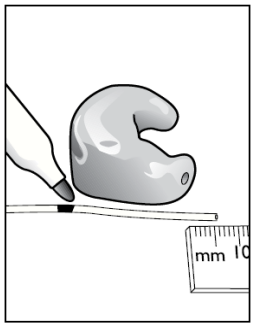


Figure 3.8. Custom soft ear mould placed next to the probe tube, with the tube extending at least 5 mm past the canal. A marking was made on the probe tube at the end of ear mould. (Reprinted with permission from Frye electronics, Inc.).

The integrated probe microphone set of Fonix 7000 system consisted of the reference microphone and the probe tube microphone. This unit was placed on test ear of participant. The calibration of the system was confirmed. Levelling of the sound field was carried out to ensure calibrated sound pressure level delivered from the loud speaker. The earhook slider was moved up or down for optimal positioning of the probe tube, that was inserted in the ear canal, such that the marking was visible at the inter tragal notch.

The Real Ear Unaided Response (REUR) was measured for digi speech at 65 dB SPL. As mentioned earlier, the length of the probe tube that was inserted into the ear canal was such that the marking was at the inter tragal notch for measuring the REUR. The hearing aid with custom soft ear mould was fitted to the test ear of participant to measure the Real Ear Aided Response (REAR) using the stimulus that was used for recording the REUR. The REUR was subtracted from the REAR to obtain the Real Ear Insertion Gain (REIG) by the system. The real ear measurement was carried out to verify that the gain of hearing aid matched with the target gain. Further, to optimize the hearing aid gain, the Ling's six syllables were presented at 65 dB SPL through the audiometer in a sound field. The gain and the frequency shaping

of the hearing aid were manipulated through fine tuning option for the audibility of Ling's six syllables.

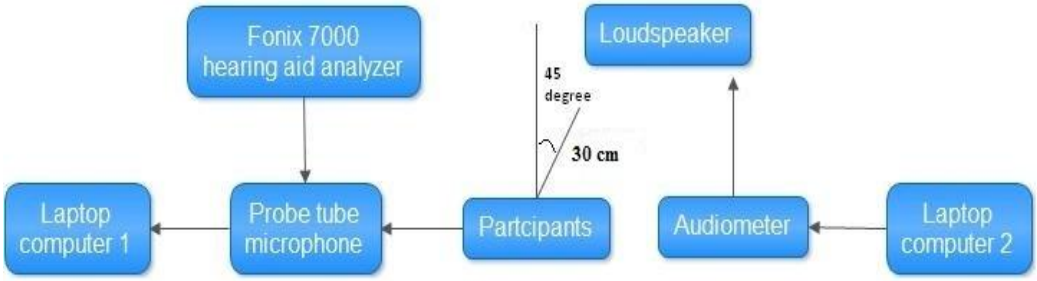


Figure 3.9. Instrumentation used to record CV syllables in the ear canal of the participant.

Further, to optimize the hearing aid gain, Ling's (1976, 1989) six syllables were presented at a calibrated level of 65 dB SPL through the audiometer in a sound field. The gain and the frequency response of the hearing aid were manipulated for the audibility of Ling's six syllables, through fine tuning option. After optimizing the hearing aid gain during insertion gain measurement (IGM), the output of the hearing aid for /ɗa/ and /si/ stimuli at the ear canal of each participant was measured, in both unaided and aided conditions. The output of the laptop computer 2 was connected to the auxiliary input of audiometer to present the recorded CV tokens /ɗa/ and /si/ stimuli at 65 dB SPL. The speech output from the audiometer was delivered through loudspeaker. The probe tube microphone in the ear canal pick up the speech stimulus and the calculated spectral energies at approximately half-octave bands from 0.25 kHz to 8 kHz were displayed in the Fonix 7000 instrument. The levels as a function of frequency from 0.25 kHz to 8 kHz, in octaves, were noted down for each stimulus, in the unaided and aided conditions. Further, the output of these stimuli (/ɗa/ and /si/ stimuli, in both unaided and aided conditions) was subsequently digitally recorded using a laptop computer 1 installed with Praat software (version 5.1.29), using a

sampling frequency of 44.1 kHz with 16 bit resolution. Before recording the hearing aid output at the ear canal, it was ensured that the level of each signal was varied in the audiometer so that the intensity measured at the test ear was 65 dB SPL, as measured by the sound level meter (Larson & Davis, Model 831). This gives a more realistic approximation of the natural condition than the response measured using pure tones of different frequencies. Figure 3.9 depicts the block diagram to record CV stimuli in the ear canal of the participant. The two CV stimuli thus recorded from the probe tube microphone, in unaided and aided conditions, were subjected to spectral and temporal analysis. Inorder to obtain spectral information, the fundamental frequency (F0) and the first two formant frequencies (F1 and F2) were measured at the onset and end of formant transition, in both conditions, for each token. These components were utilized to evaluate the extent of spectral alteration between unaided and aided conditions to each stimulus. Envelope Difference Index (EDI) was measured for obtaining information on the extent of temporal alteration between the unaided and aided conditions for each stimulus. EDI is a technique which quantifies the temporal contrast between any two envelopes of stimuli (Fortune, Woodruff, & Preves, 1994). The EDI value ranges from 0 to 1, 0 indicating that there is no difference between the two stimuli being compared and 1 indicating that the two stimuli being compared are entirely different.

Phase 3: Measurement of the representation of amplified speech at the brainstem and cortical levels using electrophysiological measure. For recording the FFR, LLR and ACC from each partcipant, a new session was created by entering and saving the participant information in the participant's demographics section of the Neuroscan instrument. Each participant was seated comfortably on a reclining chair. The electrode sites were prepared using skin preparing gel. Disc type silver coated

electrodes were placed with conduction gel. The non-test ear was masked with narrow band noise of 50 dB SPL, if indicated (Burkard & Hecox, 1987). While recording the FFR, responses were recorded from Cz to nose, with the forehead as ground. However, for recording the potentials of LLR and ACC, the non-inverting / active electrode (+) was placed on the Cz, Fpz, C3, & C4, the ground electrode was on the mastoid of non-test ear and the inverting / reference electrode was placed on the nose. It was ensured that intra electrode impedance was less than 5 k Ohms and inter electrode impedance was less than 2 k Ohms.

The participant was instructed to ignore the signals and watch a movie that was muted and played through a battery-operated laptop. This instruction was made to reduce the movement of the eyeball and blink. He/she was also asked to minimize the head movement. A five-minute break between recording conditions was given. The /ḍa/ and /si/ stimuli were delivered separately through the loud speaker of the Stim 2 of Neuroscan. The FFR, LLR and ACC responses were recorded after ensuring a stable EEG recording, from each participant. The entire procedure of recording FFR, LLR and ACC were repeated after fitting the hearing aid with custom soft shell ear mould to the test ear of each participant in the clinical group. The stimuli were presented at 65 dB SPL for both unaided and aided conditions. Stimulus and recording parameters for FFR, LLR and ACC potentials were as described in Table 3.4. The block diagram of sequence of phases in the study is represented in Figure 3.9. The rationale for using these parameters also provided, whenever relevant.

Table.3.4

Stimulus and recording parameters of FFR at the brainstem level; and LLR & ACC at the cortical levels of the auditory pathway

Parameters	*FFR*		*LLR*	*ACC*	*Rationale*
Stimulus	/ɗa/	For /i/ portion of /si/	/ɗa/	/si/	As given earlier in the Justification of stimulus
Duration	94 ms	301 ms	94 ms	301 ms	Optimal duration of stimulus in which recording could be completed in lesser time without affecting the response of interest (Skoe, & Kraus, 2010; Rosburg, Haueisen, & Sauer, 2002).
Polarity	Condensation and rarefaction		Alternating		Adding both condensation and rarefaction polarities in FFR, minimizes the stimulus artifacts and removes the cochlear microphonics. Additionally, accentuates the low frequency component of the response including phase locking to the amplitude envelope (Russo, Nicol, Musacchia, & Kraus 2004; Aiken & Picton, 2006) Alternating polarity used in LLR and ACC minimizes the stimulus artifacts (Campbell, Kerlin, Bishop, & Miller, 2012).
Inter- stimulus interval	/ɗa/ = 93 ms /si/ = 113 ms		700 ms		In FFR, to avoid the overlap of preceding response trail with the successive response trail; and to preserve the perception of complex syllables (Skoe & Kraus, 2010). A slow rate of signal presentation is essential, due to long refractory time of cortical neurons for recording the LLR and ACC (Tremblay, Billings, & Rohila, 2004).
Number of Sweeps	1000 sweeps from each polarity		250		In FFR, to obtain robust and reliable response (Skoe & Kraus, 2010). In LLR and ACC, overall SNR increases quickly at the first few sweeps and then begins to plateau (Tremblay, Billings, & Rohila, 2004). Thus, fewer sweeps were used.
Transducer	Magnetically shielded loud speaker				Since the aided responses were measured

Montage	Vertical montage: - Active: Cz - Reference: Nose - Ground: Forehead		- Active: C3, Cz, C4, Fpz - Reference: nose - Ground: non-test ear mastoid	The FFR recorded from Cz to nose accentuates more rostral (i.e., lateral lemniscus/ inferior colliculus) components rather than more peripheral contributions (Galbraith, Threadgill, Hemsley, Salour, Songdej, & Ton, 2000). Simultaneous recordings from multiple scalp at cortical level are essential for understanding scalp field generated in the sensory pathways and the nature of artifact.
Pre-stimulus Epoch	-30 ms	-30 ms	-100 ms	To accurately record the baseline activity when there is no stimulus
Post-stimulusEpoch	100 ms	340 ms	700 ms	An analysis epoch long enough to encompass the acoustic content of speech stimulus recorded at brainstem and cortical levels
Baseline correction				Average amplitude in the baseline time window is removed from each point of post baseline to obtain only the neural code with respect to stimulus
Filter setting	0.10kHz to 1 kHz		0.0001 kHz to 0.03 kHz	Low pass filter (1 kHz) - neurons of auditory brainstem phase lock to approximately 1kHz; and resolving harmonics of cochlea (Shackleton & Carlyon, 1994) High pass filter (0.10kHz) - the fundamental frequency of both the stimuli were well above 0.10 kHz i.e., F0 of /da/ being 133 Hz and /i/ of /si/ being 138 Hz) The cortical response consists of low frequency energy within the spectrum of EEG
Signal averaging				The signal averaging improves the signal-to-noise ratio (Hood, 1998). Further, in epoched files, if there were any high amplitude responses, they were not included while being averaged to avoid the eye blink response
Artifact rejection	+/- 37µV		+/- 70 µV	To remove three types of artifacts (non-biological, muscular, & stimulus), which exceed typical neural response size
Number of recording	Twice		Twice	To determine response replicability

Response analyses

The data collected from the procedure were analyzed for spectral and temporal parameters of speech syllables at the ear canal of each participant. Further, the representation of speech syllables was investigated at the brainstem and cortical levels of the auditory system in the group with normal hearing and clinical group.

Analyses of spectral and temporal aspects of speech syllables at the ear canal. The unaided and aided speech stimuli (/ɗa/ and /si/) recorded using the probe tube microphone of the Fonix 7000 at the ear canal, were analyzed for spectral and temporal contents. The recorded waveforms of the speech stimuli were opened in Praat software (Version-5.1.29) for spectral analysis. The transition duration was identified visually in each recorded speech syllable. The F_0 and the frequencies of first two formants F_1 and F_2 were noted down at the onset and offset of formant transition of /ɗa/. This was noted in the unaided and aided conditions. The transition duration is the time difference between the onset and steady state of the frequency of second formant of the following vowel (Santhosh, 2007). The onset of transition was defined as the first glottal pulse following the release of the stop consonant. The end of F_2 transition was defined as the first glottal pulse of the steady state of vowel (Santosh, 2007). The F_0 at the onset of transition, and the frequencies of the first two formants at the onset and offset of formant transition were noted down. Additionally, the levels across octave frequencies from 0.25 kHz to 8 kHz were measured for /ɗa/ and /si/, in unaided and aided conditions, to know the representation of energy across frequencies.

For temporal analysis, the envelope difference index (EDI) (Fortune, Woodruff, & Preves, 1994) was utilized to determine the extent to which the hearing aid altered the natural temporal characteristic of each speech syllable (/ʤa/ and /si/). The EDI represents a precise quantification of the temporal contrast that exists between the two waveforms, i.e., the unaided and aided waveforms in this context. The EDI ranges from 0 (i.e., perfect correspondence between the envelopes) to 1 (i.e., no correspondence between the envelopes). In each case, the EDI represented a comparison of signal envelopes of syllable (/ʤa/ or /si/) for unaided and aided versions of the same syllable. The stimulus was rectified, filtered with digitally low-pass filtered (Butterworth 6^{th} order filter with a 50 Hz cut-off), and down sampled (sampling frequency of 6 kHz). Further, the mean amplitude was calculated from the down sampled envelope. Each sampled data point in the envelope was scaled to the mean amplitude by dividing every value by the mean amplitude (Jenstad & Souza, 2007). This provided a common reference for comparing the two envelopes to obtain the EDI. The same steps were followed for the aided syllable. The unaided and aided versions of /ʤa/ stimulus are depicted in Figure 3.10. The EDI was calculated using the equation suggested by Fortune, Woodruff, and Preves (1994). The equation is given below.

$$\text{EDI} = \sum_{n=1}^{N} |\text{Env1n} - \text{Env2n}|)/2\text{N},$$

Where ‘Env1n’ was the aided waveform of a given syllable, and ‘Env2n’ was the unaided waveform of the syllable; and N = number of sample points in the waveforms.

The EDI was computed using the MATLAB code for /ɗa/ and /si/ stimuli. The value of EDI was EDI = 0.31 for /ɗa/ and 0.32 for /si/.

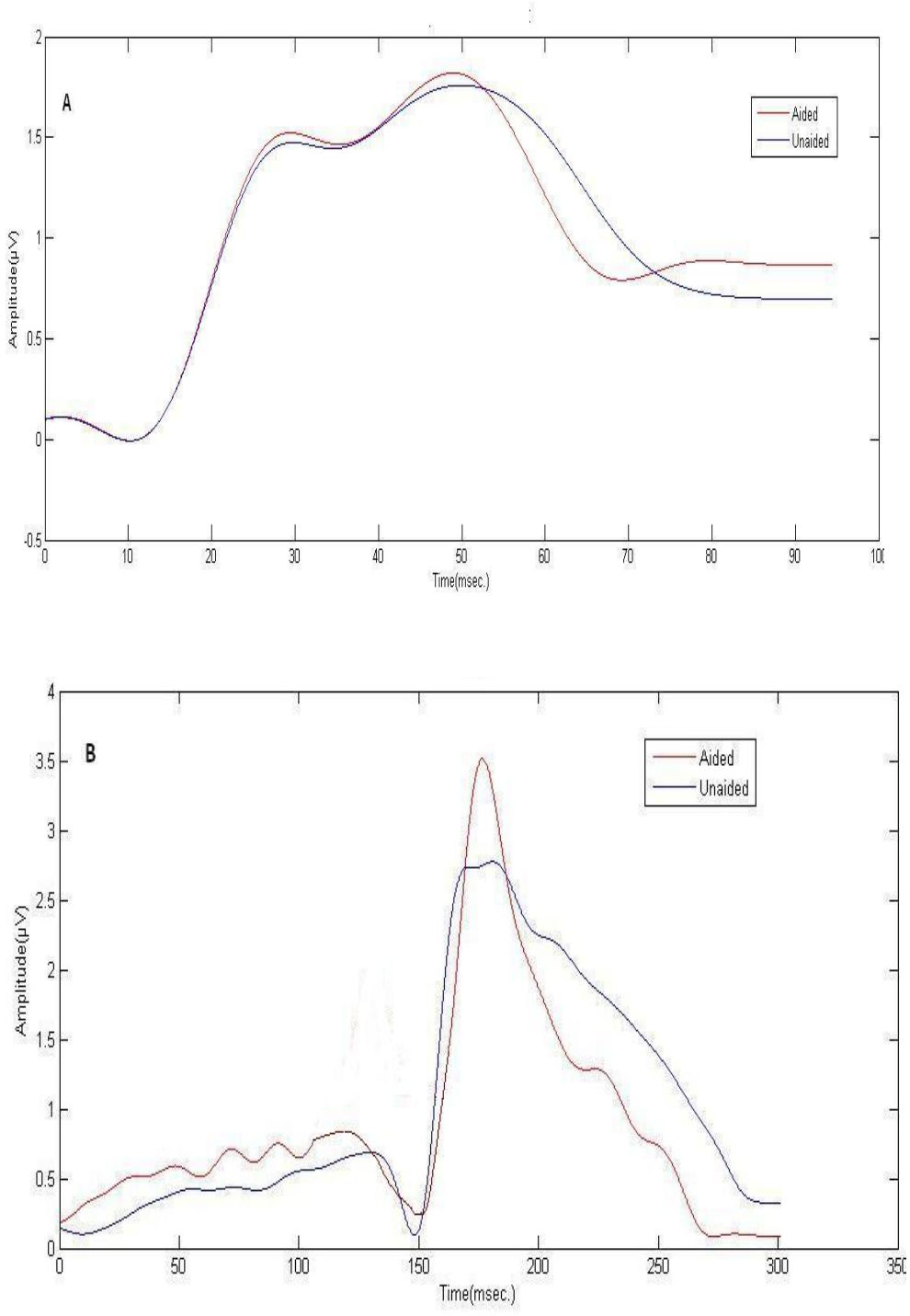


Figure 3.10. The EDI of the unaided and aided versions (A) for /ɗa/ and (B) for/si/ stimuli.

Analyses of representation of speech syllables at the level of auditory brainstem. The FFR to /ɗa/ stimulus was recorded at the level of auditory brainstem in both rarefaction and condensation polarities. The responses obtained from both the polarities were added. In the group with normal hearing, the responses to /ɗa/ and /si/ stimuli were recorded at 65 dB SPL in unaided condition alone. However, in the clinical group, the responses to /ɗa/ and /si/ stimuli were recorded at 65 dB SPL, in both unaided and aided conditions. The responses were absent in unaided condition for both /ɗa/ and /si/ stimuli, due to hearing loss.

The brain stem response was analyzed for slope of V-A, and F_0, F_0 energy and F_1 energy in the aided condition of clinical group and unaided condition of normal hearing group. Before analysis, the recording for each stimulus was repeated to check the reliability. The test-retest reliability was performed using intra-class correlation for the responses elicited for each stimulus obtained from clinical group and group with normal hearing. The result revealed that correlation value of test-retest reliability was 0.98 for response elicited to /da/ stimulus in both clinical and normal hearing groups and 0.97 in clinical group and 0.96 in normal hearing group for response elicited to /si/ stimulus. The slope of V-A was calculated for the transient response to /ɗ/ of /ɗa/ syllable. The responses of F_0, F_0 energy and F_1 energy were analyzed in the transition portion from /ɗ/ to /a/ in /ɗa/ stimulus. It must be noted that herein after the notation /ɗa/ was utilized instead of /ɗ/ of /ɗa/. In another stimulus /si/, except for slope, a similar analysis was carried out to obtain F_0, F_0 energy and F_1 energy from transition portion of /i/ in /si/ syllable. Here in after referred to as /si/ instead of /i/ of /si/ with reference to the brain stem responses.

The response to the transient portion of the speech stimulus /ɗa/ included a positive peak V, analogous to the peak V elicited by click stimuli, followed immediately by a negative trough A (Russo, Nicol, Musacchia, & Kraus, 2004). The latencies of V and A were measured at the centre, if the waveform contained a single peak. If the waveform had a double peak of unequal amplitudes, then the centre of the largest peak was considered.(Tremblay, Friesen, Martin, & Wright, 2003).Thus, the latency (ms) and amplitude (µV) of the discrete V and A were measured. The slope of V-A was calculated from discrete amplitude differences between the V and A, which was divided by latency differences (i.e., duration) of the V to A (Wible, Nicol, & Kraus, 2005). The slope represents sharpness of the neural response (Anderson, Parbery-Clark, White-Schwoch, Drehobl, & Kraus, 2013) The discrete marking of V and A is shown in Figure 3.11. The slope of V-A (in µV/ms) was calculated by the equation given below.

$$\text{Slope V} - \text{A} = \frac{\text{peak amplitude of V} - \text{peak amplitude of A}}{\text{latency of V} - \text{latency of A}}$$

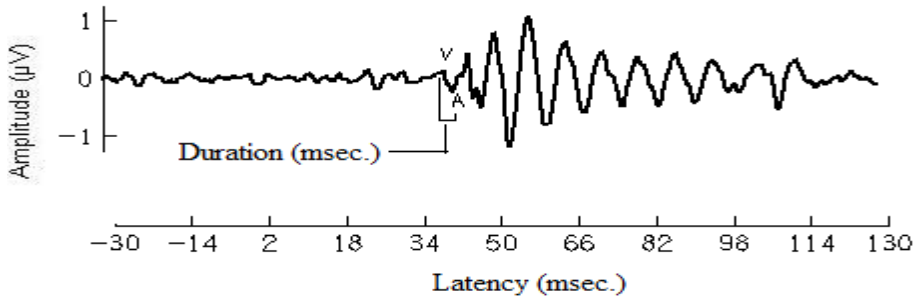


Figure 3.11. Transient response of FFR (V and A) obtained from burst portion of /da/ stimulus in aided condition.

The autocorrelation method was utilized to obtain the F_0 in the FFR corresponding to the transition duration (37 ms) of /ɗa/ stimulus (Figure 3.12). For

this, the '.avg' file of 'Cz' channel was selected. Later, the range of transition duration to which autocorrelation was to be done was specified. The range of transition duration was between the latency of transient response of 'V' to which 37 ms was added. A default algorithm of the autocorrelation, m-code, was utilized to calculate the F_0. Further, the 'Brainstem Tool box' (Kraus & Nicol, 2005) was utilized to find out the F_0 energy and F_1 energy in the formant transition within the same range of transition duration.

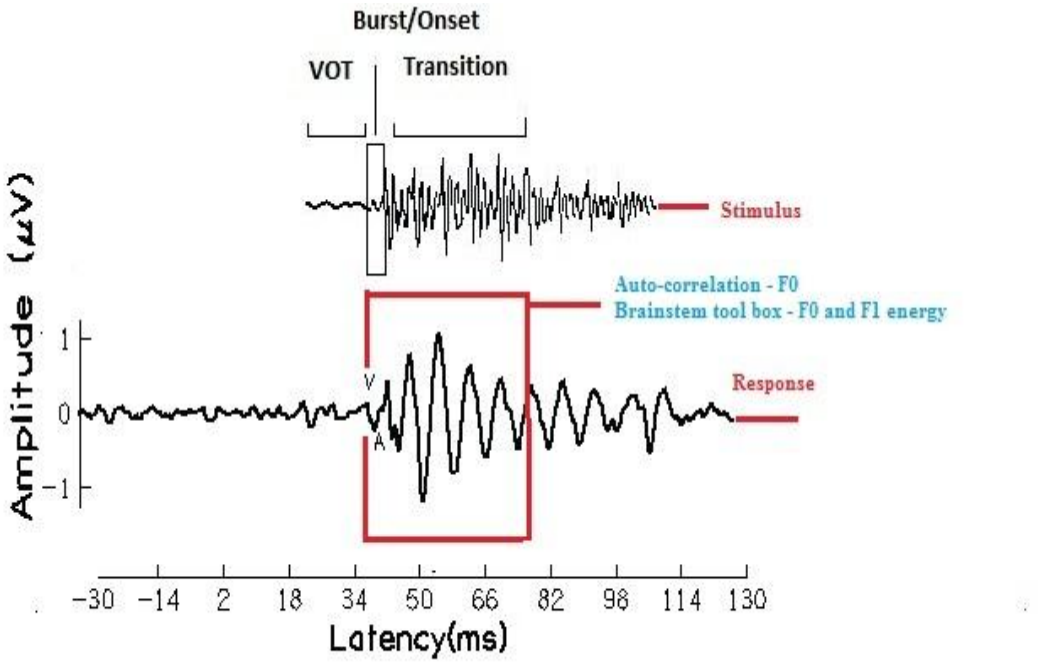


Figure 3.12. Transition response of FFR obtained from /ɖa/ stimulus.

The autocorrelation method was utilized to determine frequency of F_0 in the FFR corresponding to the range of transition duration of /si/ stimulus (47.1ms). Before performing auto correlation, the transient response was detected in the waveform of FFR elicited to /si/ stimulus (Hemanth & Manjula, 2012). The latency of transient response a^1 corresponds to the onset of formant transition /i/ of /si/. To the transient response, 47.1ms was added, such that it corresponded to the offset of

formant transition/ onset of steady state. The range of time in ms correspond to initiation of transient response a^1 and off set of formant transition (a^1 + 47.1ms) was specified in the algorithm of autocorrelation to obtain frequency of F0 in the FFR elicited to /si/ stimulus. Similarly, in 'Brainstem stem Toolbox' (Kraus & Nicol, 2005), the latencies corresponding to the initiation of transient response a^1 and off set of formant transition (a^1 + 47.1ms) were fed to obtain the energy of F_0 and F_1 in the FFR recorded to /si/ stimulus (Figure 3.13).

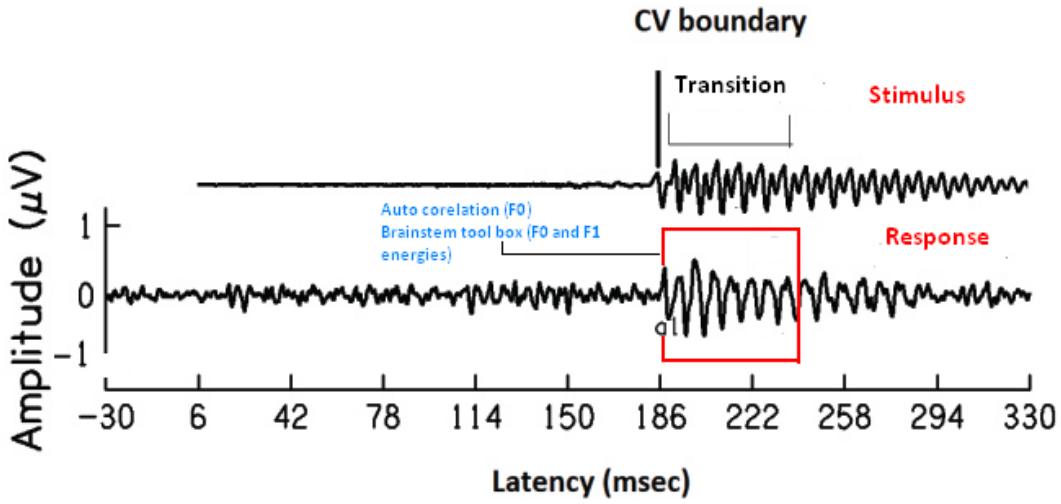
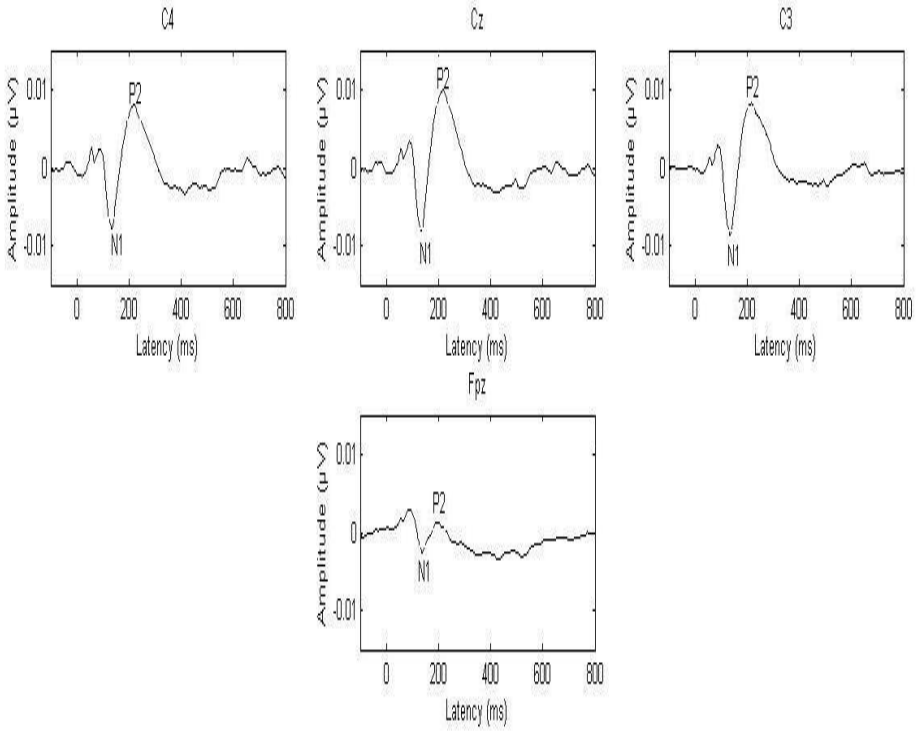


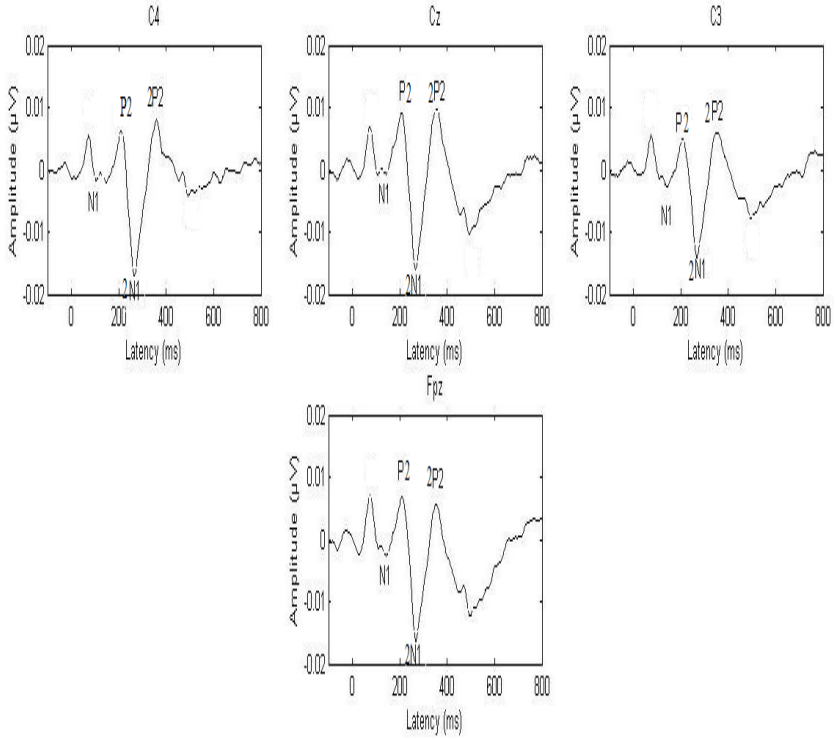
Figure 3.13. Transition response of FFR obtained from /si/ stimulus in aided condition.

Analyses of representation of speech syllables at the level of auditory cortex.

At the cortical level, the response was recorded from four electrode sites 'C3', 'Cz', 'C4' & 'Fpz'. The electrode site from which higher amplitude was recorded was utilized to investigate the representation of speech syllables. The waveforms of LLR and ACC from four electrode sites are depicted in Figure 3.14. The mean and standard deviation of the amplitudes of N1 and P2 of LLR components are tabulated in Table 3.5. and the ACC are tabulated in Table 3.6.



LLR



ACC

Figure 3.14. LLR and ACC recorded at four electode sites from normal hearing group.

Table. 3.5

Mean (M) and Standard Deviation (SD) of the amplitudes (µV) of N1 and P2 component of LLR from four electrode sites.

	Amplitude (µV) of LLR components	
Electrode sites (N=40)	*N1 M±SD*	*P2 M± SD*
C3	-2.59±1.42	2.53±1.23
Cz	-2.70±1.46	3.04±1.40
C4	-2.33±1.66	2.58±1.31
Fpz	-2.50±1.60	2.72±1.50

Table. 3.6

Mean (M) and Standard Deviation (SD) of the amplitudes (µV) of ACC from four electrode sites.

			Amplitude (µV) of ACC components	
Electrode sites (N=40)	*N1 M±SD*	*P2 M±SD*	*2N1 M±/SD*	*2P2 M±SD*
C3	1.13±0.77	1.13±0.90	-3.07±0.29	1.88±0.22
Cz	-1.30±0.92	1.43±1.22	-3.28±0.36	2.02±0.26
C4	-0.97±0.82	1.08±0.81	-2.79±0.27	1.52±0.18
Fpz	-1.06±0.77	1.37±1.04	-3.15±0.32	1.83±0.33

From Tables 3.6 and 3.7, it was noted that the mean amplitudes was higher in electrode site ‘Cz’ compared to the other electrode sites in each components of LLR and ACC. The amplitude of each component of LLR and ACC recorded from four electrode sites were analyzed separately using MANOVA. The results of MANOVA

revealed no significant difference across electrode sites in each component of LLR and ACC.

In each component of LLR and ACC, maximum amplitude was noted at 'Cz' electrode site. The finding of the present study is in accordance with that reported by Vaughan and Ritter (1970). The reason speculated by them was that the resistance offered at vertex 'Cz' is least compared to that offered by other electrode sites. The above reason was substantiated by Ohm's law, as the current flow (Ionic exchange) is higher or least resistance is offered (cerebrospinal fluid, skull and scalp), there is higher voltage of potential generated from the source and this is recorded by the electrode (Berger & Scherg, 1994).

In the normal hearing group, the LLR and ACC were recorded from /ḍa/ and /si/ stimulus respectively in unaided condition alone. In clinical group, the LLR and ACC were recorded in both unaided and aided conditions. The responses were absent in the unaided condition in clinical group.

Each epoched file in the Fpz channel was visually inspected for higher amplitude. The purpose was to remove the eye blink activity (Picton et al., 2000). Those epochs were rejected in all the four channels. After artifact rejection, a minimum of at least 200 sweeps were included for averaging. The epoched file from each channel is as shown in Figure 3.15.

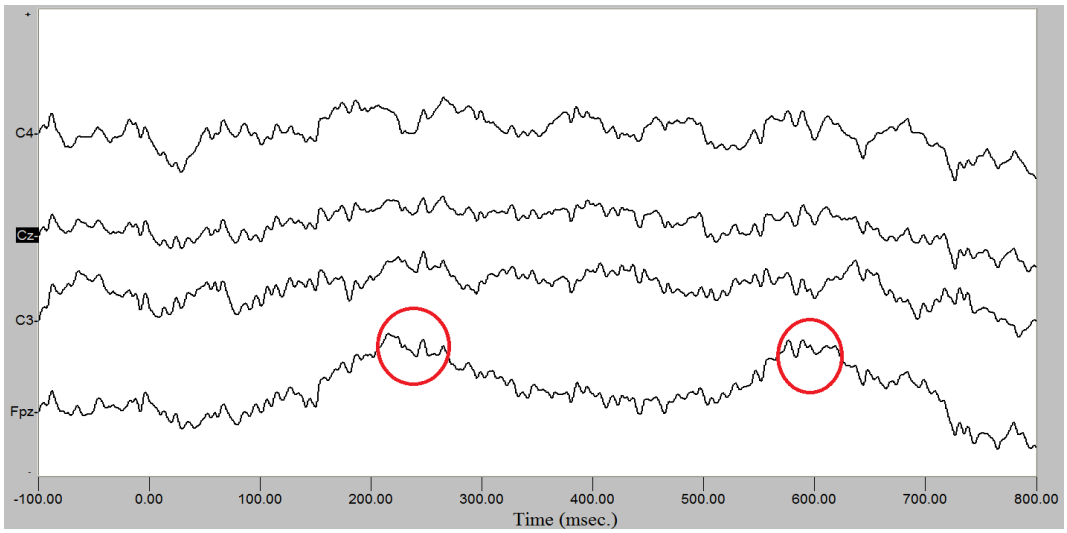


*Figure 3.15.*The aided epoch file recorded from clinical group to /ɖa/ stimulus. The higher amplitude of epoch in Fpz channel is marked in circles. Those epochs with higher amplitude were rejected in all the four channels.

The LLR waveform was analyzed for latency and amplitude. The peaks and troughs were typically measured at the centre. If the waveform contained double peak/trough of equal amplitudes, the latency was measured at the mid-point between the two. When the peaks/troughs were unequal in amplitude, the latency was measured at the centre of the higher peak (Tremblay, Friesen, Martin, & Wright, 2003). Before analysis of the response, intra-class correlation was performed to check the test re-test reliability. The results revealed correlation values appeared 0.99 clinical group and 0.98 in normal hearing group. The latency (ms) and amplitude (µV) of the discrete N1 and P2 were measured. The slope was calculated from the amplitude difference between N1 and P2, which was divided by latency differences (i.e., duration) of the N1 to P2 (Wible, Nicol, & Kraus, 2005). The discrete marking of N1 and P2 is shown in Figure 3.16. The slope of N1-P2 (in µV/ms) was calculated by equation below.

$$\text{Slope N1} - \text{P2} = \frac{\text{peak amplitude of N1} - \text{peak amplitude of P2}}{\text{latency of N1} - \text{latency of P2}}$$

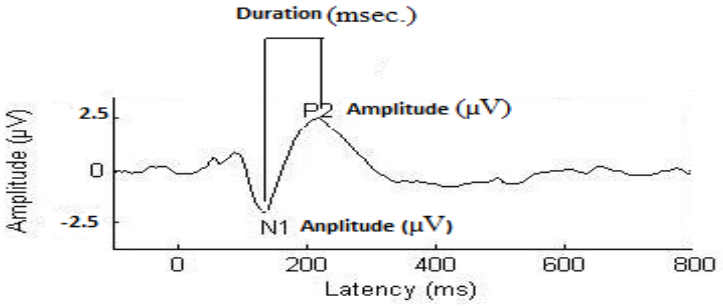


Figure 3.16. Late latency response (LLR) evoked for /ɗa/ stimulus in aided condition.

Further, at the cortical level, acoustic change complex was obtained for /si/ stimulus. The acoustic change complex was analyzed in terms of latencies (ms) and amplitudes (μV) at the onset of consonant (N1-P2) and onset of vowel (2N1-2P2) portions of the response corresponding to /si/ stimulus. The intra-class correlation was performed to check the test re-test reliability. The results revealed correlation values appeared 0.98 in both clinical and normal hearing groups. The marking of peaks in the ACC was adopted from Tremblay, Friesen, Martin, and Wright (2003). The discrete peaks and troughs were marked on the ACC waveform as shown in the Figure 3.17.

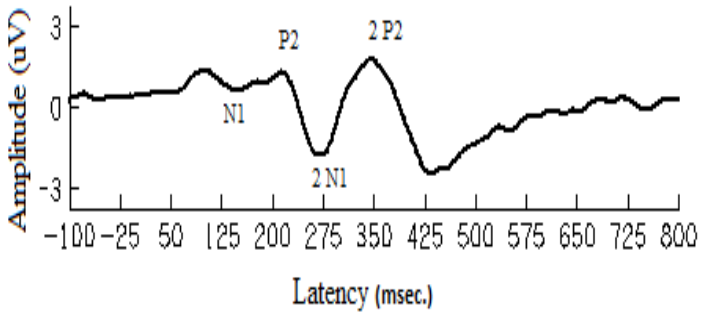


Figure 3.17. The onset of consonant (i.e., N1-P2) and the onset of vowel (i.e., 2N1-2P2) of ACC to /si/ stimulus, in aided condition.

The P1 and the N1, being the first positive and negative peaks, corresponded to the onset response. This reflects the change in acoustic energy from silent portion

to the onset of frication. The 2P1and 2N1, the second positive and negative peaks, correspond to the transition response. This reflects the transitional change in acoustic energy from consonant portion to the onset portion of the vowel.

Thus, the following measures for each test ear were tabulated:

1. At the ear canal of the participants in the clinical group: Spectral (frequency and intensity) and temporal (EDI) parameters were measured for each stimulus.
2. At the auditory brainstem level (FFR) in normal hearing and clinical groups : Slope of V-A, F0, F0 energy and F1 energy were measured for each stimulus.
3. At the auditory cortical level in normal hearing and clinical groups : Slope of N1-P2 (calculated from N1 and P2 components of LLR for /ḍa/ stimulus) and latency and amplitude of ACC (i.e., N1 and P2 – onset of consonant; 2N1 and 2P2 – onset of vowel).

Statistical Analyses

The following statistical analyses were performed on the data:

1. Descriptive analysis was performed for each dependent variable.
2. Kolmogorov-Smirnov normality test was done to know the distribution of data within each sub-group, and Levene's homogeneity test was carried out to know the distribution of data across sub-groups.
3. Kruskal-Wallis / ANOVA / MANOVA test was carried out to know the main effect of age.
4. Mann-Whitney *U* test was performed to know the sub-groups might have caused significant difference.
5. In MANOVA, if significant difference was noted then post-hoc Duncan test was used.

6. Paired samples t-test was utilized to evaluate the difference between unaided and aided conditions for each spectral parameter.
7. Independent samples t-test was utilized to evaluate statistical significant difference between
    a. aided responses from clinical group and unaided response from group with normal hearing at brain stem and cortical level responses of the auditory pathway.
    b. the responses obtained from good and poor hearing aid performers at brain stem and cortical level responses of the auditory pathway.

CHAPTER - 4 RESULTS

The main aim of the study was to investigate the representation of amplified speech along different levels of the auditory pathway in individuals with sensorineural hearing loss. In order to address this, the objectives were to a) measure the output of hearing aid at the ear canal; b) to compare the representation of speech syllables at the auditory brainstem level between clinical group and the group with normal hearing c) to compare the representation of speech syllables at the auditory cortical level between clinical group and normal hearing group, and d) to study the brainstem responses; cortical responses in good and poor hearing aid performers. To evaluate these objectives, the study was conducted in three phases for collection of data. The data were subjected to statistical analyses using the Statistical Package for Social Sciences (SPSS for Windows version 17.0).

The data obtained from the ear canal, brain-stem and cortical levels were tabulated and analyzed using descriptive statistics. The normality test was performed, when indicated, in order to know the distribution of data and to decide about the choice of the statistical tests. Based on this, non-parametric / parametric statistical analyses were carried out to evaluate the significant differences between the variables / conditions studied. The post-hoc test was used whenever required.

At the ear canal level, the spectral and temporal parameter of speech syllables, in unaided and aided conditions

At the ear canal, the spectral and temporal measures of each stimulus (/ḍa/ and /si/) were analysed, in unaided and aided conditions. The spectral measure included the fundamental frequency (F_0) and the frequencies of first two formants (F_1 and F_2) at the onset and offset of transition in each stimulus. In additional, the spectral energy at frequencies from 0.25 kHz to 8 kHz, in octaves, was measured in the aided and unaided conditions for /ḍa/ and /si/ stimuli.

The temporal measure included the Envelope Difference Index (EDI). Details of the statistical analyses in order to investigate the extent to which the hearing aid preserved the spectral and temporal contents of the stimuli at the level of ear canal are given below.

1. Spectral measures, F_0, F_1, F_2:
    a. F_0, the frequencies of first two formants (F_1 and F_2) at the onset and offset of the transition and spectral energies (from 0.25 to 8 kHz, in octaves), in the unaided and aided conditions, across clinical sub-groups were analysed using descriptive statistics. *Figure 4.3.* Illustrates statistical tests performed on data of spectral parameters in different sub-groups
    b. Kruskal-Wallis test was carried out to know the difference between sub-groups in F_0, formant frequencies (F_1 and F_2) at the onset and offset of transition, and spectral energy at octaves frequencies from 0.25 kHz to 8 kHz in the ear canal, for both the stimuli, in unaided and aided conditions.
    c. Kolmogorov-Smirnov normality test was carried out to know the distribution of the combined unaided (F_0, frequencies of first two formants at the onset and offset of transition) and combined aided (F_0, frequencies of first two formants at the onset and offset of transition) data.
    d. Paired samples t-test was utilized to evaluate the difference between unaided and aided conditions for each spectral parameter.
2. Temporal measure, EDI :
    a. The envelope difference index (EDI) obtained, from each sub-group to both stimuli, was analysed using descriptive statistics.
    b. Kruskal-Wallis test was carried out to know the difference in EDI of each stimulus between sub-groups.

Effect of hearing aid processing on spectral parameter of speech stimuli. The mean value of F_0 for /ɖa/ in unaided and aided conditions is depicted in Figure 4.1. There was only a slight variation in the mean F_0 in the sub-groups, in unaided and aided conditions.

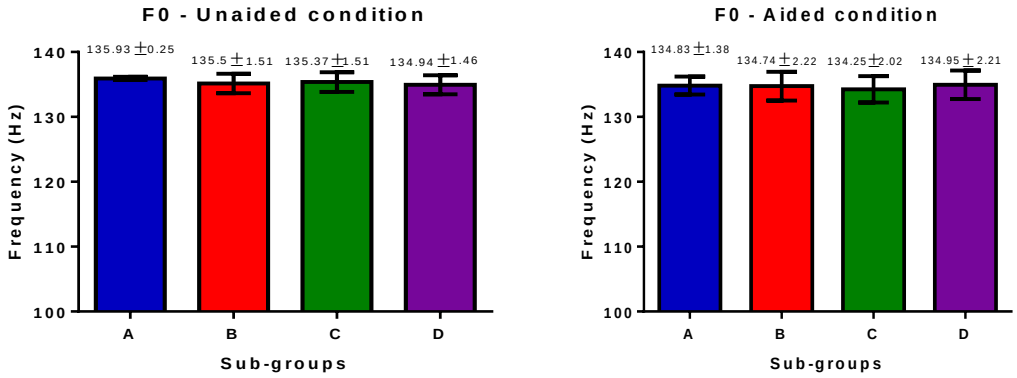


*Figure 4.1.*Mean and standard deviation of F_0 for /ɖa/ stimulus, in the unaided and aided conditions.

The mean values of the frequencies of first two formants (F_1 and F_2) at the onset and offset of transition for /ɖa/ in the unaided and aided conditions are tabulated in Table 4.1. There was only a slight variation in the mean values of the frequencies of first two formants (F_1 and F_2) at the onset and offset of transition for /ɖa/ in the sub-groups, in unaided and aided conditions.

Table.4.1

Mean and standard deviation of the frequencies (in Hz) of first two formants, F_1 and F_2, at the onset and offset of transition for /ḍa/ stimulus obtained from sub-groups, in the unaided and aided conditions

Sub-groups (No. of participants)	*Spectral parameters*	*Unaided condition*		*Aided condition*	
		Onset of transition M±SD	*Offset of transition M±SD*	*Onset of transition M±SD*	*Offset of transition M±SD*
Sub-group 'A' (N=6)	F_1	523.26±13.56	564.33±25.69	529.68±33.95	557.28±42.91
	F_2	1832.50±17.96	1680.33±14.00	1827.33±14.90	1669.83±25.37
Sub-group 'B' (N=7)	F_1	518.41±13.20	561.00±17.73	533.78±18.66	568.28±23.29
	F_2	1828.85±20.44	1681.42±45.73	1823.00±21.66	1673.00±6.35
Sub-group 'C' (N=19)	F_1	525.51±22.44	553.28±28.23	519.38±27.61	562.54±34.48
	F_2	1830.68±49.49	1676.63±26.09	1829.78±11.89	1670.94±41.86
Sub-group 'D' (N=28)	F_1	521.42±28.43	554.76±17.11	520.29±20.54	556.39±27.20
	F_2	1821.90±31.74	1672.53±70.16	1821.36±45.47	1680.35±30.38

Figure 4.2. depicts the mean value of F_0 for /si/ in the unaided and aided conditions. There was only a slight variation in mean F_0 in the sub-groups, in the unaided and aided conditions.

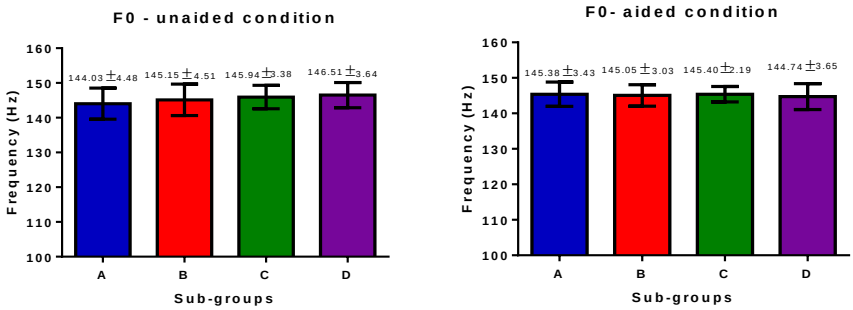


Figure 4.2. Mean and standard deviation of F_0 for /si/ stimulus in unaided and aided conditions.

Table 4.2.

Mean and standard deviation of the frequencies (in Hz) of first two formants F_1 and F_2 at the onset and offset of transition for /si/ stimulus obtained from sub-groups, in the unaided and aided conditions

Sub-groups (No. of participants)	*Spectral parameters*	*Unaided condition*		*Aided condition*	
		Onset of transition M±SD	*Offset of transition M±SD*	*Onset of transition M±SD*	*Offset of transition M±SD*
Sub-group 'A' (N=6)	F_1	344.68±11.48	312.30±8.69	347.57±7.76	314.88±14.49
	F_2	2255.61±71.35	2423.00±77.79	2227.89±62.17	2459.50±32.94
Sub-group 'B' (N=7)	F_1	348.29±4.85	306.24±16.84	351.08±16.58	137.40±12.24
	F_2	2264.57±50.51	245.71±31.07	2238.28±30.92	2458.57±51.58
Sub-group 'C' (N=19)	F_1	350.30±9.76	312.42±14.73	350.61±9.44	305.37±14.78
	F_2	2265.97±18.72	2461.14±17.92	2263.99±29.91	2449.18±52.66
Sub-group 'D' (N=28)	F_1	347.50±5.44	305.71±13.55	349.66±6.337	307.11±16.92
	F_2	2258.51±28.52	2249.95±45.36	2264.13±23.76	2456.05±30.48

The mean values of the frequencies of first two formants (F_1 and F_2) at the onset and offset of transition for /si/, in the unaided and aided conditions, are tabulated in Table 4.2. There was only a slight variation in the mean of the frequencies of first two formants (F_1 and F_2), at the onset and offset of transition, for /si/ in the sub-groups, in unaided and aided conditions.

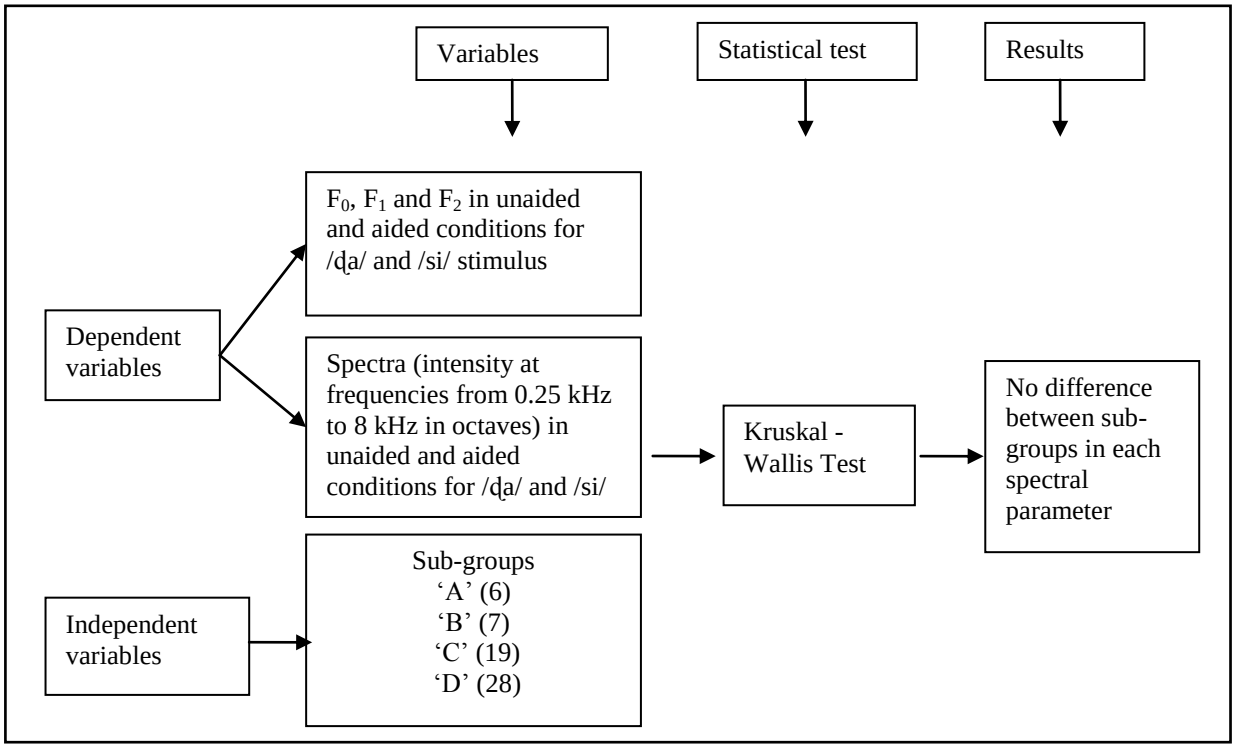


Figure 4.3. Illustrates statistical tests performed on data of spectral parameters in different sub-groups.

Further, Kruskal-Wallis test was carried out to know the significant difference between sub-groups in each spectral parameter of /ɖa/ and /si/ stimuli in unaided and aided conditions. The results indicated that there was no difference between age on the F_0 of each stimulus (/ɖa/ and /si/), in both unaided and aided conditions. Similar findings were noted in the F_1 and F_2 formant frequencies at the onset and offset of transition between the sub-groups. The chi-square and significant value of Kruskal-Wallis test for the spectral parameters of each stimulus, in the unaided and aided conditions, are tabulated in Tables 4.3. and 4.4.

Table 4.3

Chi-square (χ^2) and p-value of Kruskal-Wallis test on F_0 of /ḍa/ and /si/ stimuli between sub-groups, unaided and aided conditions

	/ḍa/ stimulus		*/si/ stimulus*	
Conditions	*χ^2(3)*	*p-value*	*χ^2(3)*	*p-value*
Unaided	34.55	0.207	1.55	0.670
Aided	1.34	0.719	1.10	0.777

Table 4.4

Chi-square (χ^2) and p-value of Kruskal-Wallis test on F_1 and F_2 *for /ḍa/ and /si/ stimuli, between sub-groups, in the unaided and aided conditions*

Conditions	*Spectral parameters*	*/ḍa/*				*/si/*			
		Onset of transition		*Offset of transition*		*Onset of transition*		*Offset of transition*	
		χ^2(3)	*p-value*	*χ^2(3)*	*p-value*	*X^2(3)*	*p-value*	*X^2(3)*	*p-value*
Unaided	F_1	0.79	0.850	1.84	0.605	4.14	0.247	2.90	0.406
	F_2	2.33	0.507	1.91	0.590	1.60	0.659	0.41	0.937
Aided	F_1	2.34	0.504	1.23	0.724	0.76	0.857	4.59	0.204
	F_2	3.15	0.363	1.71	0.634	5.27	0.153	0.94	0.814

As noted earlier, there was no significant difference in each of the spectral parameters (F_0, F_1 and F_2) between the sub-groups. Hence, data for each spectral parameter in sub-groups were combined. Further, the combined data of F_0 and the frequencies of first two formants (F_1 and F_2) in the onset and offset of transition, in unaided and aided conditions, were subjected to normality test. For /ḍa/ stimulus, it can be observed that the F_1 followed a rising pattern and F_2 followed a falling pattern of transition, in unaided and aided conditions (Figure 4.4). For /si/ stimulus, in Figure (4.5), it can be noted that the F_1 followed a falling pattern and F_2 followed a rising pattern of transition, in unaided and aided conditions, in the combined group.

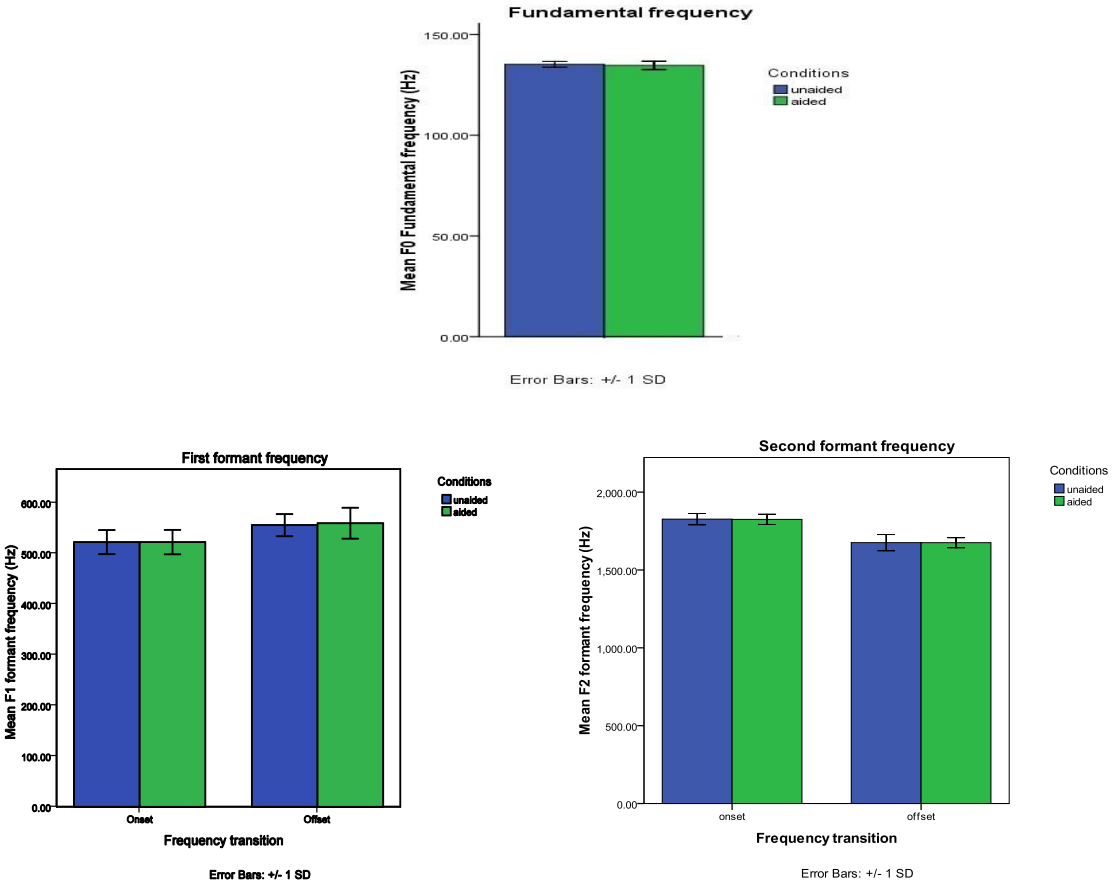
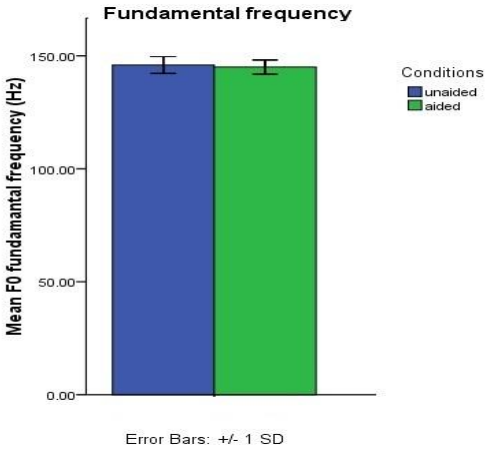


Figure 4.4. Mean and standard deviation of F_0 and frequencies of first two formants (F_1 and F_2) at onset and offset of transition for /ɖa/ stimulus in unaided and aided conditions.



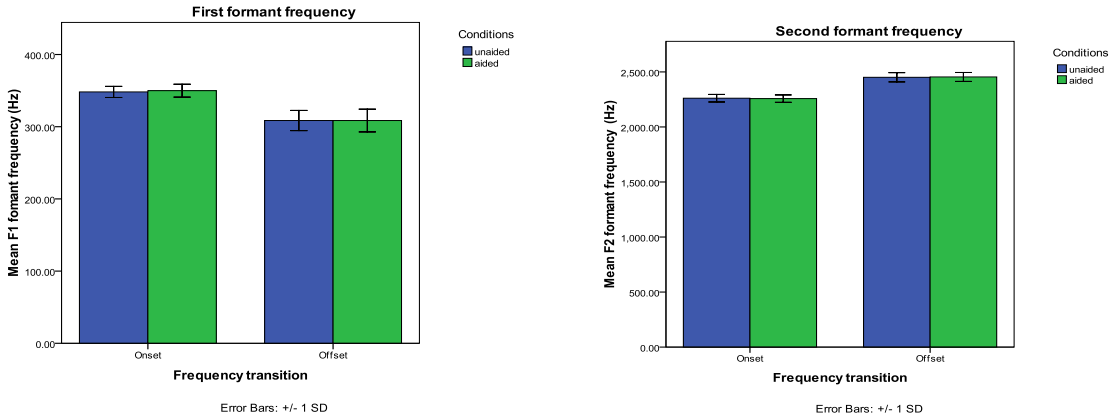


Figure 4.5. Mean and standard deviation of spectral parameters in unaided and aided conditions for /si/ stimulus.

Figure 4.6. Illustrates statistical tests performed on data of spectral parameters in unaided and aided conditions. The result of Kolmogorov-Smirnov normality test revealed that in each spectral parameter there was no significant difference ($p>0.05$) in each unaided and aided conditions indicating that the data were normally distributed. Thus, paired samples t-test was utilized in order to know the difference between unaided and aided conditions in each spectral parameter. The result revealed that there was no significant difference between unaided and aided conditions on F_0 for /ɖa/ stimulus ($t= 1.69$, $p = 0.09$) and for /si/ stimulus ($t=1.35$, $p = 0.18$). The t-value and p-value of paired samples t-test on the frequencies of first two formants (F_1 and F_2) at the onset and offset of transition for each stimulus are tabulated in Table 4.5. The result revealed that there was no significant difference between the unaided and aided conditions in the spectral parameters of F_1 and F_2 formant frequencies at the onset and offset of transition for each stimulus. Thus, it was noted that there was no significant difference in spectral parameters studied (F_0, F_1 and F_2) between the unaided and aided conditions.

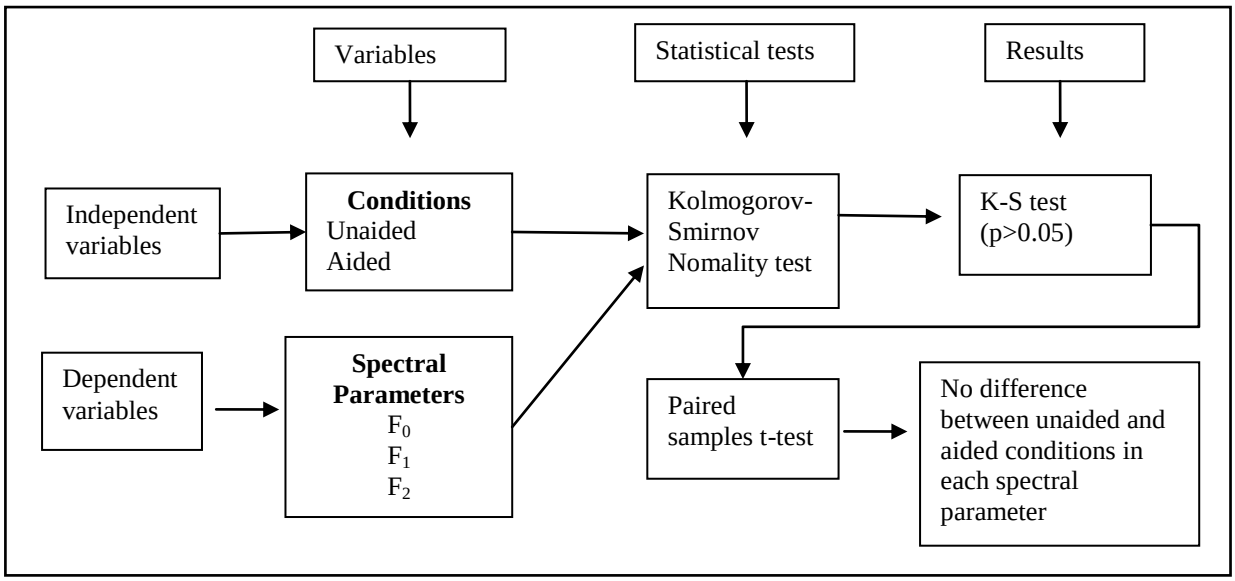


Figure 4.6 Illustrates statistical tests performed on data of spectral parameters in unaided and aided conditions.

Table 4.5

The t-value and p- value of paired samples t-test on F_1 and F_2 at the onset and offset of transition, between the unaided and aided conditions, to each stimulus

Spectral parameters		*/ɖa/ stimulus*		*/si/ stimulus*	
		t-value	*p-value*	*t-value*	*p-value*
Onset of transition	F_1	0.008	0.993	-1.09	0.278
	F_2	0.30	0.762	0.57	0.566
Offset of transition	F_1	-0.77	0.442	0.007	0.994
	F_2	0.04	0.967	-0.45	0.650

In addition, spectral energy at frequencies from 0.25 to 8 kHz (in octaves) in sub-groups, in the unaided and aided conditions, for both the stimuli was analyzed, to see the representation of energy across frequencies at the ear canal in sub-groups.

Table 4.6

Mean (M) and standard deviation (SD) of spectral energy (in dBSPL) at different octave frequencies to /ɖa/ stimulus in sub-groups, in the unaided and aided conditions

Conditions	Sub-groups (N)	Spectral energy (dB SPL) for /ɖa/ stimulus at ear canal					
		0.25 kHz Mean ±SD	0.5 kHz Mean± SD	1 kHz Mean± SD	2 kHz Mean± SD	4 kHz Mean± SD	8 kHz Mean± SD
Unaided	'A'(6)	61.33±7.71	63.50±6.59	61.16±6.21	69.16±11.87	63.33±10.68	49.50±7.55
	'B'(7)	54.00±8.48	56.28±4.71	54.42±7.48	62.28±10.45	59.57±12.39	48.28±5.40
	'C'(19)	54.00±6.20	55.42±8.62	54.89±8.89	59.31±7.14	60.52±8.28	46.84±7.11
	'D'(28)	59.00±7.44	60.39±8.07	59.10±8.14	61.89±8.95	59.39±9.70	46.67±6.22
Aided	'A'(6)	76.83±7.49	79.66±10.03	83.16±11.08	96.50±10.50	79.16±15.89	59.16±10.81
	'B'(7)	76.14±10.09	83.71±10.37	92.57±8.69	95.71±11.48	84.14±4.28	59.42 ±7.41
	'C'(19)	74.31±7.47	80.31±7.91	85.47±10.63	91.15±9.45	81.94±9.00	59.21 ±6.44
	'D'(28)	76.03±9.67	86.03±10.78	89.21±7.44	94.96±10.86	82.60±10.43	63.10 ±8.89

Table. 4.7

Mean (M) and standard deviation (SD) of spectral energy (in dBSPL) at different octave frequencies in sub-groups, in the unaided and aided conditions, to /si/stimulus

Conditions	Sub-groups (N)	Spectral energy (dB SPL) for /si/ stimulus at ear canal					
		0.25 kHz Mean ±SD	0.5 kHz Mean± SD	1 kHz Mean± SD	2 kHz Mean± SD	4 kHz Mean± SD	8 kHz Mean± SD
Unaided	'A'(6)	61.00±2.09	53.00±8.79	57.16±7.67	60.00±4.49	57.00±11.47	47.50±4.88
	'B'(7)	56.71±5.79	54.85±5.78	51.14±2.79	58.71±7.54	53.57±5.31	44.71±7.95
	'C'(19)	57.78±6.61	58.73±7.73	58.31±6.90	60.10±9.17	60.63±6.86	45.63±7.78
	'D'(28)	57.21±6.13	58.32±5.95	57.57±7.22	58.07±7.01	57.64±8.30	44.46±5.75
Aided	'A'(6)	74.50±7.39	77.83±3.06	78.83±4.79	86.16±6.40	78.50±9.18	58.33 ±7.39
	'B'(7)	72.71±10.78	75.14±8.21	76.42±9.67	88.57±7.74	80.14±10.02	59.28±11.98
	'C'(19)	73.31±9.15	77.52±8.86	78.94±10.46	87.94±10.88	83.36±14.04	59.42±8.65
	'D'(28)	72.00±7.44	76.71±8.60	80.28±8.36	87.10±8.48	80.46±11.37	57.75±7.78

From Tables 4.6 and 4.7 it can be noted that there was an increase in the spectral energy at octave frequencies from 0.5 kHz to 8 kHz in the aided condition compared to the unaided condition in all the sub-groups, for both stimuli. The data of spectral energy at each frequency from 0.5 kHz to 8 kHz (in octaves) obtained from sub-groups, in unaided and aided conditions, for both stimuli were subjected to Kruskal-Wallis test. The result revealed that there was no significant difference between sub-groups in the spectral energy at each octave frequency, in both the unaided and aided conditions, for /ɖa/ and /si/ stimuli.

Table 4.8

Chi-square χ^2 (3) and p-value of Kruskal-Wallis test on spectral energy between sub-groups, in the unaided and aided conditions, for /ɖa/ and /si/ stimuli

Stimulus	Conditions	0.25 kHz		0.5 kHz		1 kHz		2 kHz		4 kHz		8 kHz	
		χ^2	p-value	χ^2	p-value	χ^2	p-value	χ^2	p-value	χ^2	p-value	χ^2	p-value
/ɖa/ stimulus	Unaided	0.83	0.29	2.50	0.06	1.73	0.17	1.86	0.11	0.29	0.83	0.38	0.76
	Aided	0.20	0.89	1.58	0.20	1.81	0.15	0.72	0.54	1.81	0.15	0.72	0.54
/si/ stimulus	Unaided	0.76	0.51	0.88	0.45	2.02	0.12	0.30	0.82	11.46	0.23	0.39	0.76
	Aided	0.18	0.90	0.17	0.91	0.36	0.77	0.10	0.95	0.36	0.78	0.16	0.96

The chi-square and significant value of Kruskal-Wallis test for the spectral energy in the unaided and aided conditions for each stimulus are tabulated in Table 4.8. The spectral energy data at each frequency from sub-groups were combined. Descriptive analysis was carried out separately in the unaided and aided conditions. For /ɖa/ stimulus (Figures 4.7), as expected, it can be noted that the energy in aided condition was higher than in the unaided condition across frequencies (0.25 kHz to 8 kHz). At extreme low frequency (0.25 kHz) and extreme high frequencies (4 kHz and 8 kHz) the difference in energy between unaided and aided conditions is relatively minimal than at other frequencies (0.5 kHz, 1 kHz and 2 kHz).

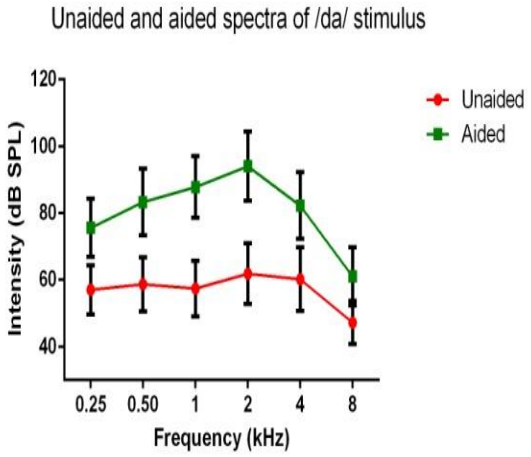


Figure 4.7. Mean and standard deviation of intensity of /ɖa/ stimulus as a function of frequency in unaided and aided conditions.

For /si/ stimulus too (Figures 4.8), it can be noted that the energy was higher in the aided condition than in the unaided conditions across frequencies (0.25 kHz to 8 kHz). At extreme low frequencies (0.25 kHz) and extreme high frequency (8 kHz) the difference in energy (i.e., from unaided and aided conditions) is relatively minimal compared to other frequencies (1 kHz, 2 kHz and 4 kHz).

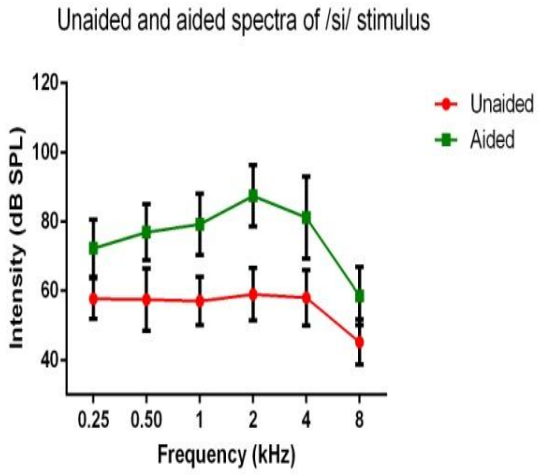


Figure 4.8. Mean and standard deviation of intensity of /si/ stimulus as a function of frequency in unaided and aided conditions.

Effect of hearing aid processing on temporal parameter of speech stimuli. The mean (M) and standard deviation (SD) of EDI for /ɖa/ and /si/ stimuli in each sub-group are tabulated in Table 4.9. It may be recalled that higher the values of EDI, greater the difference between the aided and unaided conditions and vice versa. It was observed that the mean EDI of /ɖa/ is higher in sub- group 'D' followed by sub-group 'C', sub-group 'B' and sub-group 'A'. For /si/ stimulus, the mean EDI shows a similar pattern as that for /ɖa/.

Table.4.9

Mean (M) and standard deviation (SD) of EDI between unaided and aided conditions to /ḍa/ and /si/ stimuli in each sub-group

Temporal parameter	*Sub-groups (No. of participants)*	*/ḍa/ M± SD*	*/si/ M±SD*
Envelope difference index (EDI)	*'A' (6)*	0.31±0.04	0.32±0.03
	'B' (7)	0.32±0.06	0.33±0.06
	'C' (19)	0.34±0.05	0.34±0.04
	'D' (28)	0.36±0.06	0.36±0.06

Figure 4.9 Illustrates statistical test performed on data of envelope difference index. To evaluate the difference in the EDI of each stimulus between sub-groups, Kruskal-Wallis test was carried out. The results indicated that there was no significant difference in EDI between different age groups, for each stimulus. Thus, the data on EDI for /ḍa/ and /si/ from different sub-groups were combined. The mean EDI in the combined group for /ḍa/ was 0.355 (SD = ±0.06) and that for /si/ was 0.352 (SD = ±0.05).

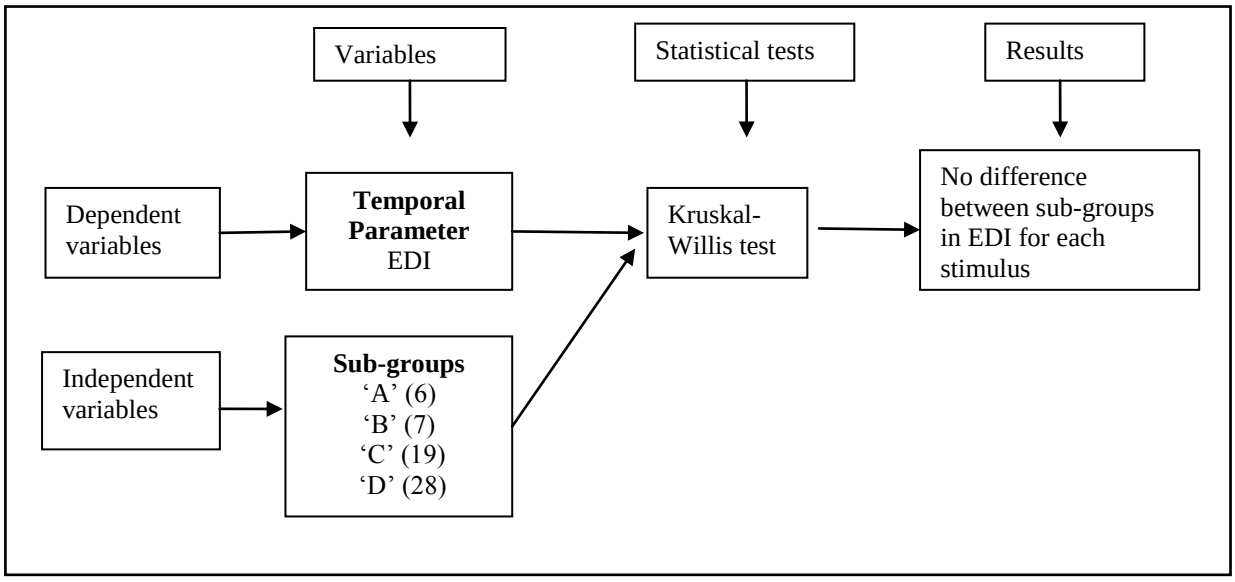


Figure 4. 9. Illustrates statistical test performed on data of envelope difference index.

At brainstem level, the representation of speech syllables in clinical group and group with normal hearing

The data from aided response obtained at brainstem level of the auditory pathway in clinical sub-groups were combined, if there was no significant difference. Likewise, the data from unaided response obtained in sub-groups with normal hearing were combined if there was no significant difference. Further, the aided response from clinical sub-groups was compared with unaided response from sub-groups with normal hearing at brainstem level of auditory pathway. This was done to investigate if the aided responses approximated the response from a normal auditory pathway.

Comparison of aided slope of V-A from clinical group with unaided slope of V-A from group having normal hearing. The slope of V-A in the brainstem responses was analyzed in the clinical sub-groups. In the clinical group, the responses at brainstem level were absent in the unaided condition. The responses obtained from the aided condition were analyzed at the brainstem level of the auditory pathway. The components of V and A were recorded for the transient portion of /ɖa/ stimulus. The slope of V-A was calculated from latencies and amplitudes of V and A. The following statistics were carried out.

1. Descriptive analysis was done for slope of V-A for /ɖa/ stimulus. Illustrates statistical tests performed on data of slope of V-A on sub-groups of clinical group is depicted in Figure 4.11.
2. Kruskal-Wallis test was performed to see if there were significant differences between sub-groups in the mean values of the slope of V-A corresponding to the transient portion of /ɖa/ stimulus.

The mean and standard deviation of slope of V-A for transient portion of /ɖa/ stimulus in each clinical sub-group are tabulated in Tables 4.10. The grand average waveform of FFR

in aided condition obtained from four clinical sub-groups is depicted in Figure 4.10. The slope of V-A was shallower with respect to age. Kruskal-Wallis test was performed to see if there were any significant differences between sub-groups in the mean values of the slope of V-A. The results indicated that there was no significant difference in the slope of V-A between age.

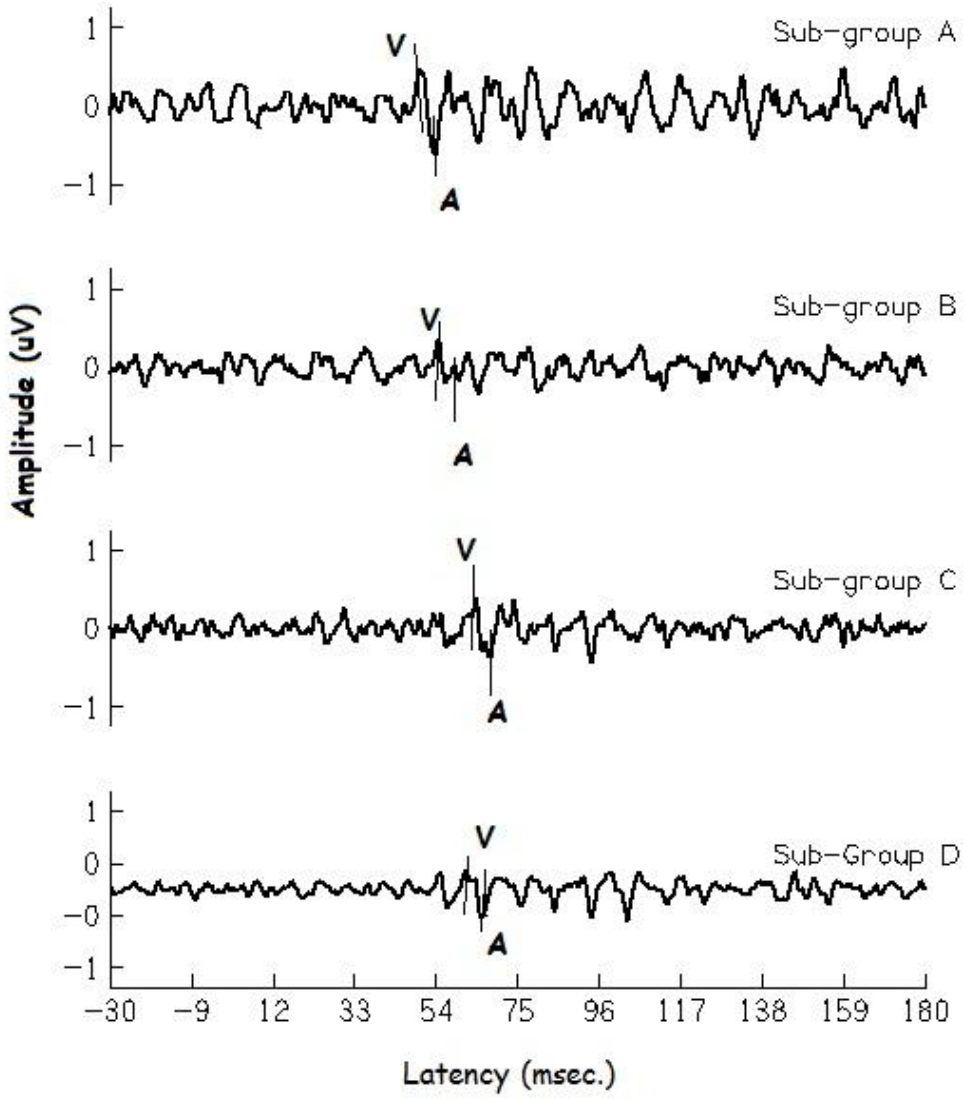


Figure 4.10. Grand average waveform of FFR for /ɖa/ stimulus from four clinical sub-groups. Peaks V and A registered at the auditory brainstem level for transient portion of /ɖa/ stimulus.

Table 4.10

Mean (M), standard deviation (SD), χ^2 (3) and p-value of Kruskal-Wallis test on slope of V-A (in µV/ms*), to /ḍa/ stimulus in clinical sub- groups*

Brainstem response	*Sub-groups (N)*	*M±SD*	*χ^2-value*	*p-value*
Slope of V-A	'A' (6)	-0.12±0.07	5.88	0.118
	'B' (7)	-0.18±0.07		
	'C' (19)	-0.22±0.08		
	'D' (28)	-0.27±0.10		

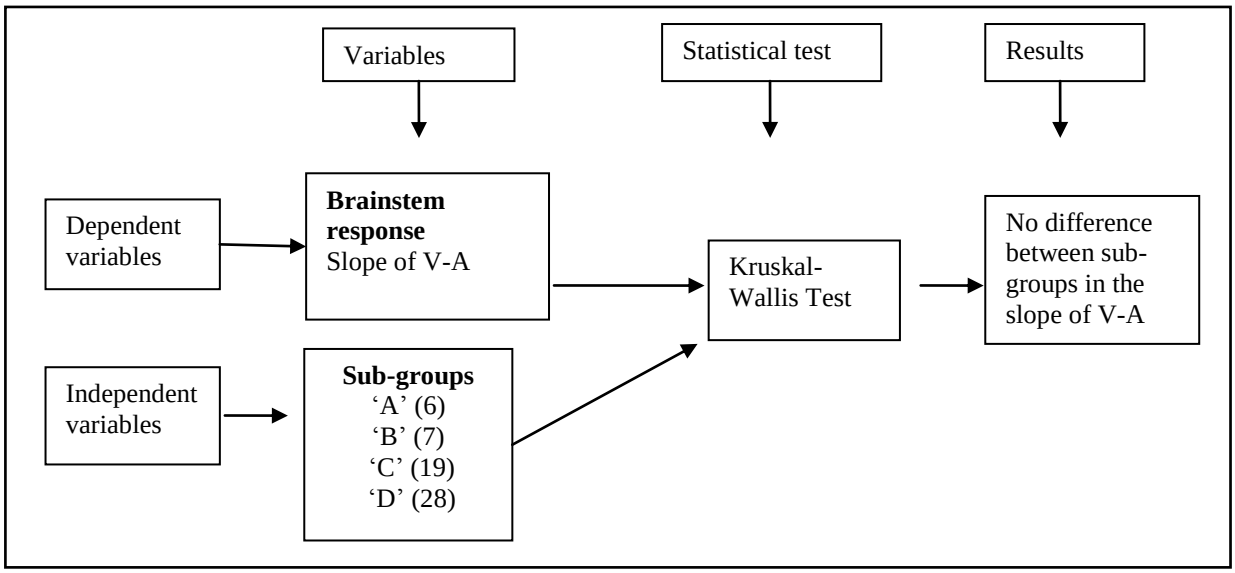


Figure 4.11. Illustrates statistical test performed on data of slope of V-A obtained from clinical sub-groups.

Further, the slope of V-A obtained from participants of group with normal hearing was subjected to statistical analyses. This included -

1. Descriptive analysis of slope of V-A for /ḍa/ stimulus obtained from four different sub-groups with normal hearing.
2. Kolmogorov-Smirnov normality test was performed to know the distribution of data within each sub-group and Levene's homogeneity test was carried out to know the distribution of each data across sub-groups with normal hearing.

3. ANOVA was performed to evaluate the significant difference across the sub-groups with normal hearing in the slope of V-A. Illustrates statistical tests performed on data of slope of V-A on sub-groups of normal hearing group is depicted in Figure 4.13.

The mean and standard deviation of the slope of V-A for /ɖa/ stimulus are tabulated in Table 4.11. The grand average waveform of FFR obtained from normal hearing sub-groups is represented in Figure 4.12. The slope of V-A was shallower with respect to age. Further, slope of V-A for /ɖa/ stimulus between the sub-groups met the assumption of normal distribution on Kolmogorov-Smirnov normality test ($p > 0.05$) and homogeneity on Levene's test ($F < 2$). Hence, ANOVA was conducted on the data of slope of V-A. The result revealed shallower slope of V-A as a function of age, such that this difference did not reach significant difference between sub-groups.

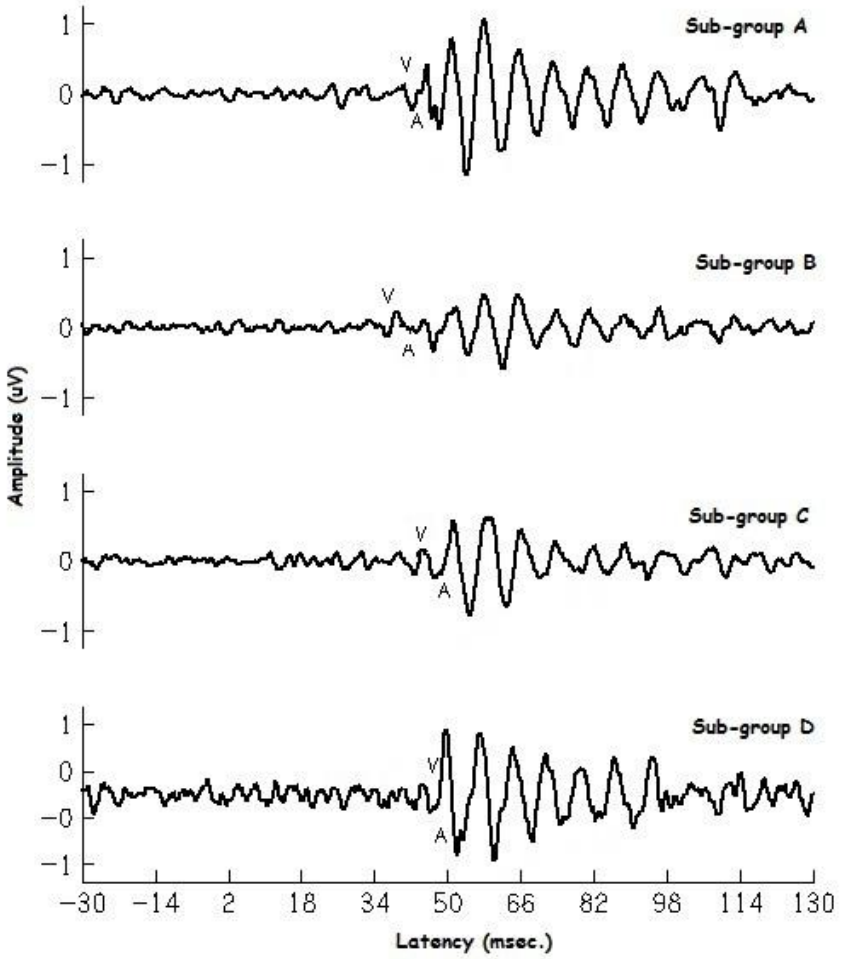


*Figure 4.12.*Grand average waveform of FFR obtained for /ɖa/ stimulus. The discrete peaks of V and A registered at the auditory brainstem level for transient portion of stimulus /ɖa/.

Table 4.11

*Mean (M) and standard deviation (SD) of slope of V-A (*µV/ms) *in sub-groups with normal hearing*

Brainstem Responses	*Sub-groups (N=10)*	*M± SD*
Slope of V-A for /ɖa/ stimulus	'A'	-0.18±0.08
	'B'	-0.20±0.06
	'C'	-0.23±0.06
	'D'	-0.25±0.07

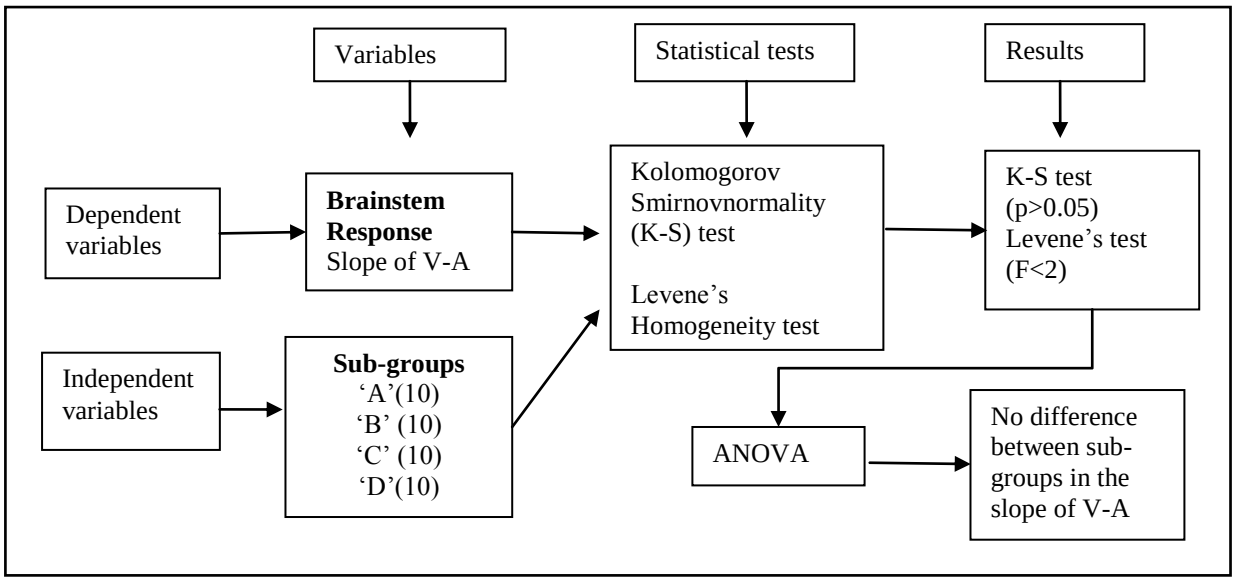


Figure 4.13. Illustrates statistical tests performed on data of slope of V-A obtained from normal hearing sub-groups.

In the clinical group, as there was no significant difference in the slope of V-A, the data from different sub-groups were combined. Similar result was noted in the group with normal hearing. The mean slope of V-A was steeper in the group with normal hearing than clinical group (Figure 4.14).

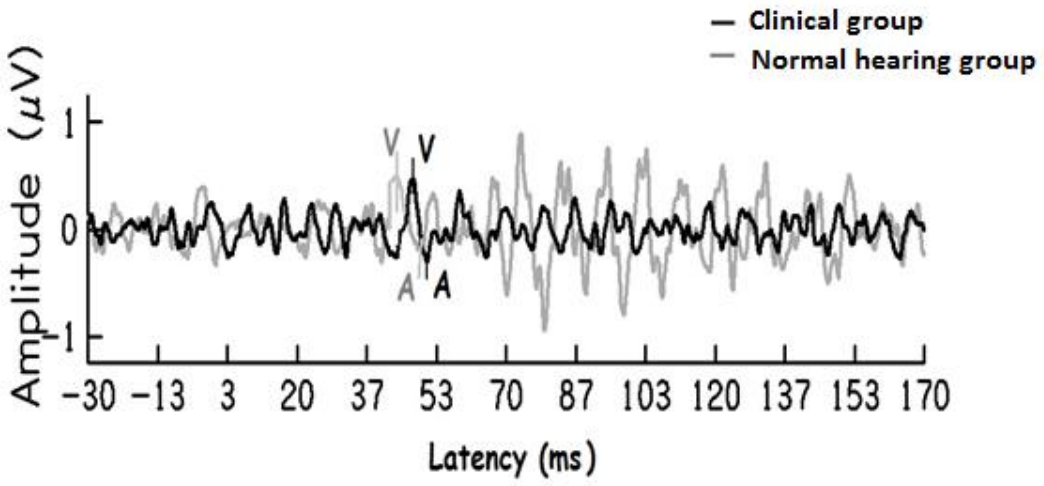


Figure 4.14. Grand average waveform of FFR for /ɖa/ stimulus from clinical group and normal hearing group are represented. The latency of V and A was prolonged and amplitude was reduced in clinical group than normal hearing group.

Figure 4.15 Illustrate the statistical tests performed on data of slope of V-A obtained from normal hearing group and clinical sub-group. The Kolmogorov-Smirnov test was done to know the distribution of slope of V-A within clinical group and normal hearing group. The Levene's homogeneity test was conducted to see the distribution of slope of V-A in clinical and normal hearing groups. The result revealed that the slope of V-A was normally distributed ($p>0.05$), both in the clinical group and in group with normal hearing. Further, the slope of V-A was homogenous, with variance less than 2, between clinical and normal hearing groups. Thus, parametric statistical test, i.e., independent samples t-test, was performed to know if there was any significant difference between clinical and normal hearing groups in the slope of V-A.

Table 4.12

*The mean (M), standard deviation (SD), t-value and p-values on independent samples t-test on slope of V-A (*µV/ms*) in normal hearing and clinical and groups*

	Slope of V-A		
Groups (N)	*M ±SD*	*t-value*	*p-value*
Normal hearing (40)	-0.20±0.12	0.35	0.726
Clinical (60)	-0.21±0.18		

The mean (M), standard deviation (SD), t-value, and p-value of independent samples t-test on slope of V-A are tabulated in Table 4.12. The result revealed that though the slope of V-A was found to be steeper in the group with normal hearing compared to that in clinical group, there was no significant difference in the slope of V-A between the two groups.

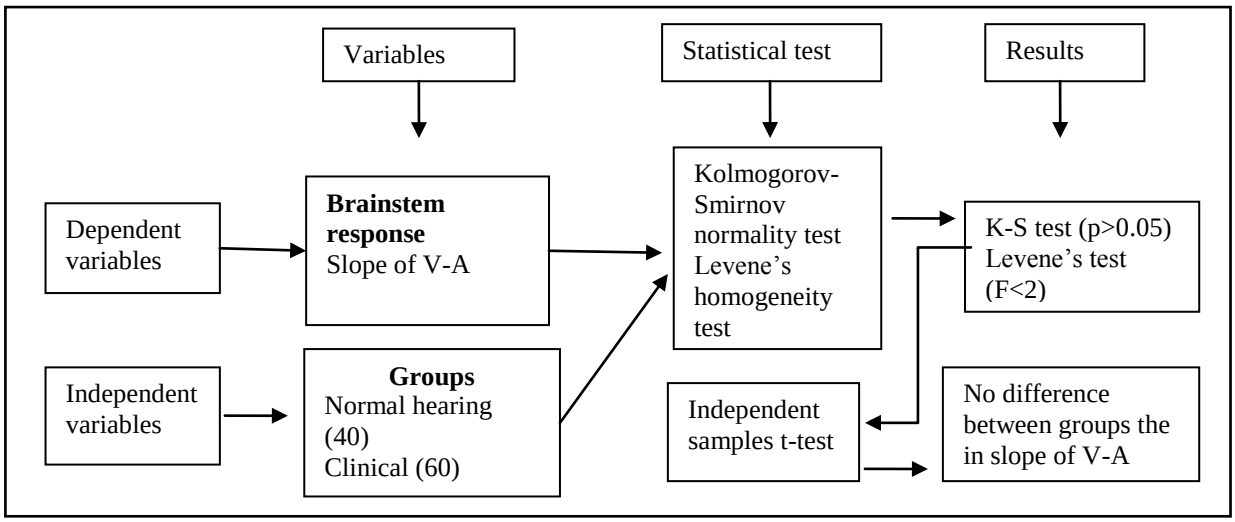


Figure 4.15. Illustrates statistical tests performed on data of slope of V-A obtained from normal gearing group and clinical sub-group.

Comparison of FFR in terms of aided F_0, F_0 energy and F_1 energy in clinical group with unaided F_0, F_0 energy and F_1 energy in group having normal hearing. In brainstem responses i.e., the data on F_0, F_0 energy and F_1 energy were tabulated for the clinical sub-groups. In the unaided condition, the responses at brainstem level were absent. The FFR response corresponding to the transition portion of stimuli /ɖa/ and /si/ obtained from the aided condition were analyzed at brainstem level of auditory pathway. The following statistics were performed to study the auditory brainstem response to each of the two stimuli in the clinical group (Figure 4.17).

1. Descriptive analysis was done for F_0, F_0 energy and F_1 energy of FFR to /ɖa/ and /si/ stimuli obtained from each sub-group.
2. Kruskal-Wallis test was performed to see if there were significant differences between sub-groups in the mean values of the F_0, F_0 energy and F_1 energy obtained from FFR corresponding to the transition portion of /ɖa/ and /si/ stimuli.
3. Mann-Whitney *U* test was conducted when indicated.

The mean and standard deviation of F_0, F_0 energy and F_1 energy of FFR for each stimulus in each clinical sub-group are tabulated in Tables 4.13 and 4.14. Figure 4.16 represents the mean and standard deviation of F_0, F_0 energy and F_1 energy of FFR for each stimulus in each sub-group. It was found that the mean F_0 encoding of both the stimuli reduced with age in each clinical sub-group. The F_0 energy and F_1 energy to each stimulus also reduced with age.

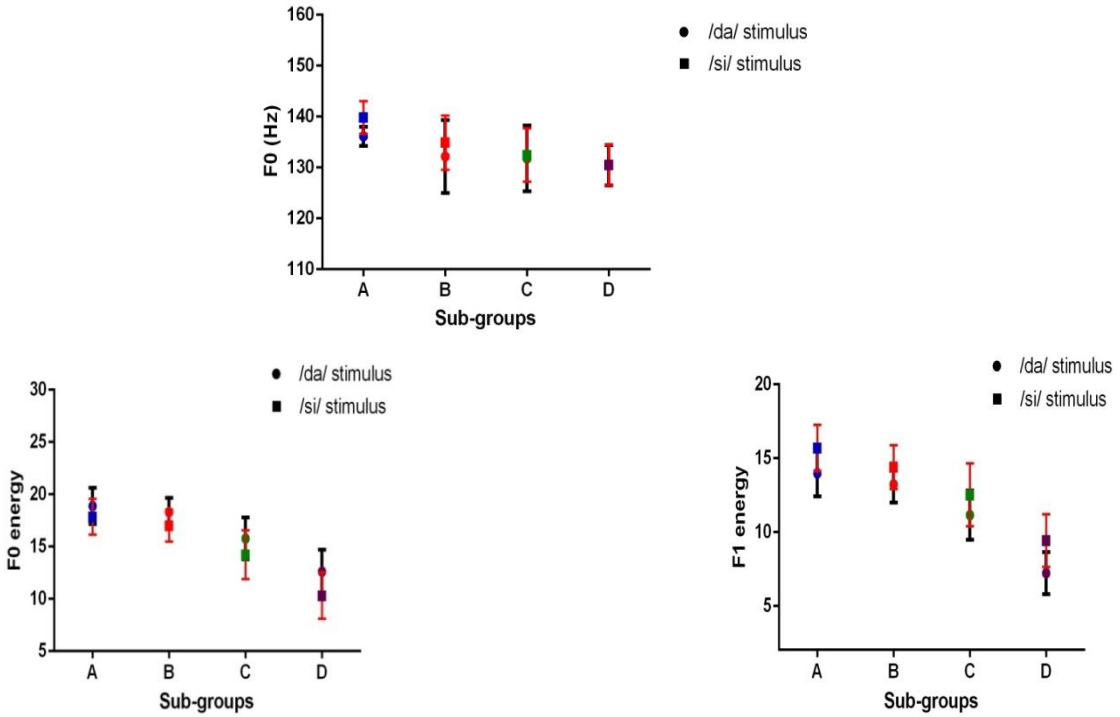


Figure 4.16. Mean and standard deviation of F_0, F_0 energy and F_1 energy, obtained from FFR for each stimulus in different clinical sub-groups.

Table 4.13

Mean (M), standard deviation (SD), χ^2 (3) and p-value of Kruskal-Wallis test on F_0 (in Hz), F_0 energy and F_1 energy to /ḍa/ stimulus in clinical sub-groups

Brainstem response	*Sub-groups (N)*	*M± SD*	*χ^2-value*	*p-value*
F_0	*'A' (6)*	136.14±1.90	8.10	0.041*
	'B' (7)	132.18±7.16		
	'C' (19)	131.76±6.48		
	'D' (28)	130.46±3.96		
F_0 energy	*'A' (6)*	18.88±1.73	38.16	0.000***
	'B' (7)	18.31±1.37		
	'C' (19)	15.79±2.01		
	'D' (28)	12.61±2.10		
F_1 energy	*'A' (6)*	13.97±1.56	44.79	0.000***
	'B' (7)	13.23±1.24		
	'C' (19)	11.14±1.67		
	'D' (28)	7.21±1.43		

Note: * =p <0.05; ***= p<0.001

Table 4.14

Mean (M), standard deviation (SD), χ^2(3) and p- values of Kruskal-Wallis test on F_0 (in Hz), F_0 energy and F_1 energy to /si/ stimulus in clinical sub-groups

Brainstem Response	*Sub-groups (N)*	*M± SD*	*χ^2-value*	*p-value*
F_0	*'A' (6)*	139.83±3.12	11.29	0.010*
	'B' (7)	134.88±5.35		
	'C' (19)	132.43±5.25		
	'D' (28)	130.48±3.97		
F_0 energy	*'A' (6)*	17.85±1.71	39.01	0.000***
	'B' (7)	16.99±1.52		
	'C' (19)	14.22±2.34		
	'D' (28)	10.30±2.20		
F_1 energy	*'A' (6)*	15.69±1.56	37.62	0.000***
	'B' (7)	14.38±1.48		
	'C' (19)	12.50±2.14		
	'D' (28)	9.40±1.79		

Note: *=p<0.05; ***=p<0.001

Kruskal-Wallis test was performed to see if there were any significant differences between sub-groups in the mean values of the F_0, F_0 energy and F_1 energy in each stimulus. The results indicated that there was a significant reduction as a function of age in the encoding of F_0 [χ^2 (3) =8.10, p = 0.041], F_0 energy [χ^2 (3) =38.16, p = 0.000] and F_1 energy [χ^2 (3) =44.79, p = 0.000] to /ɖa/ stimulus. Likewise, for /si/ stimulus, there was a significant reduction in the encoding of F0 [χ^2 (3) =11.29, p = 0.010], F_0 energy [χ^2 (3) =39.01, p = 0.000] and F_1 energy [χ^2 (3) =37.62, p = 0.000] between sub-groups, as a function of age. In order to know the sub-groups that might have accounted for significant difference in F_0, F_0 energy and F_1 energy to each stimulus, Mann-Whitney *U* test was carried out separately. The /U/ value and p-value of Mann-Whitney *U* test for F_0, F_0 energy and F_1 energy to /ɖa/ stimulus are tabulated in Table 4.15.

Table 4.15

/U/-value and p-value of Mann-Whitney U test for F_0 (in Hz), F_0 energy and F_1 energy of FFR to /ɖa/ stimulus from clinical sub-groups

	F_0		*F_0 energy*		*F_1 energy*	
Sub-groups	*/U/-value*	*p-value*	*/U/-value*	*p-value*	*/U/-value*	*p-value*
'A' vs. 'B'	-0.78	0.430	-0.71	0.243	-1.00	0.316
'A' vs. 'C'	-1.91	0.560	-2.86	0.007**	-2.64	0.008**
'A' vs. 'D'	-2.97	0.003**	-3.80	0.000***	-3.80	0.000***
'B' vs. 'C'	-0.28	0.772	-2.66	0.018*	-2.34	0.015*
'B' vs. 'D'	-2.43	0.006**	-4.04	0.000***	-4.04	0.000***
'C' vs. 'D'	-2.36	0.004**	-4.41	0.000***	-5.65	0.000***

Note: * = p<0.05; ** = p<0.01; *** = p<0.001

The results of Mann-Whitney *U* test revealed that the clinical sub-groups 'A', 'B' and 'C' were significantly different from sub-group 'D' in F_0 component obtained from FFR for /da/ stimulus. Further, Mann-Whitney *U* test was performed on F_0 energy obtained from FFR. The results revealed that there was no significant difference in F_0 energy between the clinical sub-groups 'A' and 'B'. However, sub-groups of 'A' and 'B' were significantly different

from sub-group ‘C’ and sub-group ‘D’ in F_0 energy. In addition, sub-group ‘C’ was significantly different from sub-group ‘D’ in F_0 energy. Similar results were found for F_1 energy obtained from FFR between the sub-groups.

Table 4.16

/U/-value and p-value of of Mann-Whitney U test for F_0 (in Hz), F_0 energy and F_1 energy of FFR to /si/ stimulus from clinical sub-groups

	F_0		*F_0 energy*		*F_1 energy*	
Sub-groups	*/U/-value*	*p-value*	*/U/-value*	*p-value*	*/U/-value*	*p-value*
‘A’ vs. ‘B’	-0.88	0.630	-9.30	0.352	-1.50	0.132
‘A’ vs. ‘C’	-0.77	0.560	-2.80	0.005**	-2.86	0.004**
‘A’ vs. ‘D’	-3.23	0.010*	-3.79	0.000***	-3.79	0.000***
‘B’ vs. ‘C’	-0.40	0.685	-2.45	0.041*	-2.88	0.040*
‘B’ vs. ‘D’	-2.89	0.004**	-4.04	0.000***	-4.04	0.000***
‘C’ vs. ‘D’	-2.52	0.006**	-4.56	0.000***	-4.42	0.000***

Note: * = p<0.05; ** = p<0.01; *** =p<0.001

Table 4.16 gives the /U/-value and p-value of of Mann-Whitney *U* test for F_0, F_0energy and F_1 energy of FFR to /si/ stimulus. The results of Mann-Whitney *U test* on clinical sub-groups for F_0, F_0 energy and F_1 energy to /si/ stimulus were similar to that of each component of FFR to /ɖa/ stimulus.

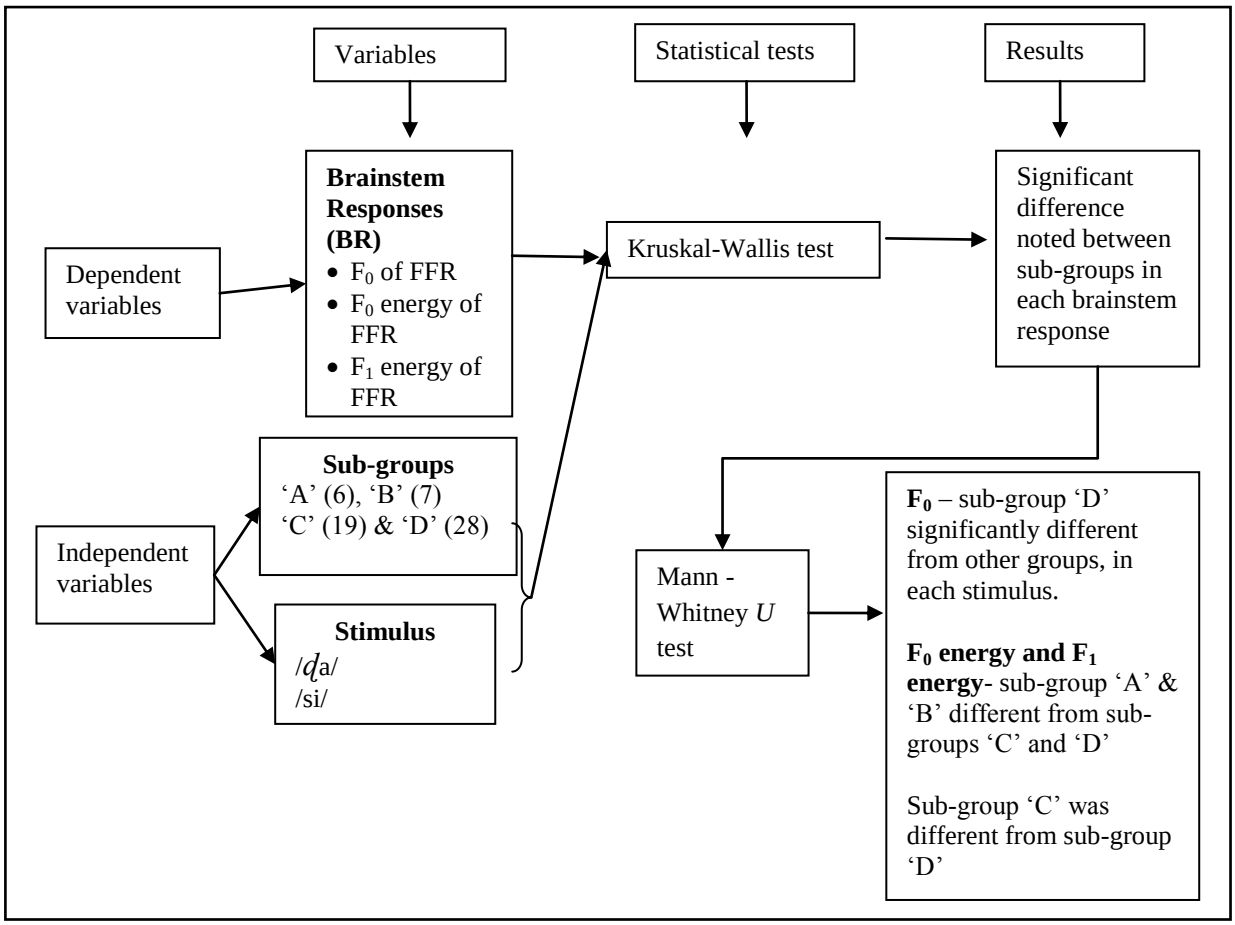


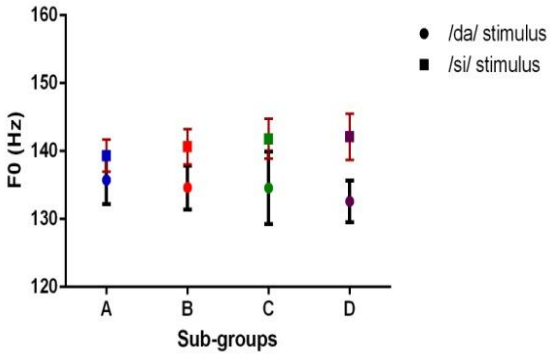
Figure 4.17. Illustrates statistical tests performed on data of F_0, F_0 energy and F_1 energy of FFR obtained from clinical sub-groups.

Further, in the transition portion for each stimulus the F_0, F_0 energy and F_1 energy were obtained from FFR in each participant of group with normal hearing. The following statistical analyses were performed.

1. Descriptive analysis of F_0, F_0 energy and F_1 energy for each stimulus were obtained from four different sub-groups with normal hearing. *Figure 4.19* illustrate statistical tests performed on data of F_0, F_0 energy and F_1 energy of FFR obtained from normal hearing sub-groups
2. Kolmogorov-Smirnov normality test was administered to know the distribution of data within each sub-group and Levene's homogeneity test were carried out to know the distribution of each data across sub-groups with normal hearing.

3. MANOVA was performed to see if there were significant differences between the sub-groups with normal hearing in the means of the F_0, F_0 energy and F_1 energy of FFR to each stimulus.
4. Post-hoc Duncan test was used when indicated.

The mean and standard deviation of the F_0, F_0 energy and F_1 energy of FFR for each stimulus at brainstem level in each sub-group are tabulated in Table 4.17. Figure 4.18 represents the mean and standard deviation of F_0, F_0 energy and F_1 energy of FFR for each stimulus at brainstem level in each normal hearing sub-group. The F_0, F_0 energy and F_1 energy of FFR recorded for the transition portion of each stimulus were reduced with age. Further, the F_0, F_0 energy and F_1 energy to each stimulus between the sub-groups met the assumption of normal distribution on Kolmogorov-Smirnov normality test ($p > 0.05$) and homogeneity on Levene's test ($F < 2$). Hence, MANOVA was administered on the data of F_0, F_0 energy and F_1 energy for each stimulus to know if there were any significant differences in the means of the F_0, F_0 energy and F_1 energy between the sub-groups with normal hearing.



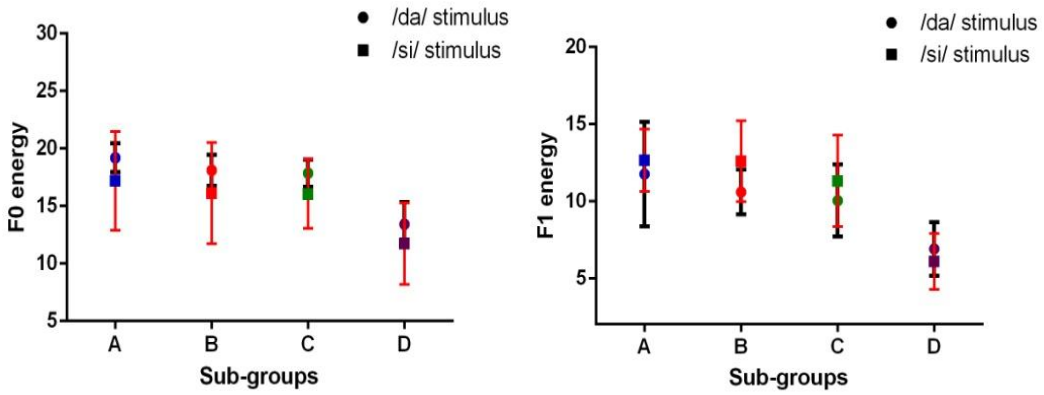


Figure 4.18. The mean and standard deviation of F_0, F_0 energy and F_1 energy of FFR for each stimulus in normal hearing sub-groups.

Table 4.17

The mean (M), standard deviation (SD) of brainstem responses to each stimulus in sub-groups with normal hearing

Brainstem Responses	*Sub-groups (N=10)*	*/da/ stimulus M± SD*	*/si/ stimulus M± SD*
F_0	*'A'*	135.76±3.55	139.31±2.36
	'B'	134.65±3.22	131.64±2.60
	'C'	134.59±5.34	139.81±2.95
	'D'	132.60±3.08	142.11±3.40
F_0 energy	*'A'*	19.19±1.26	17.19±4.29
	'B'	18.11±1.36	16.11±4.39
	'C'	17.86±1.20	16.06±3.01
	'D'	13.43±1.93	11.73±3.56
F_1 energy	*'A'*	11.76±3.40	12.66±2.02
	'B'	10.59±1.46	12.59±2.63
	'C'	10.03±2.34	11.03±2.98
	'D'	6.90±1.75	6.80±1.82

Table 4.18

The F (3, 36) ratio and p-values of MANOVA on brainstem responses to each stimulus in sub-groups with normal hearing

Brainstem Responses	*/ḍa/ stimulus*		*/si/ stimulus*	
	F –ratio	*p-value*	*F –ratio*	*p-value*
F_0	1.13	0.350	1.54	0.220
F_0 *energy*	29.93	0.000**	3.92	0.016*
F_1 *energy*	7.69	0.000**	13.00	0.000**

Note: * p<0.05; ** p<0.01; *** p<0.001;

The F-ratio and p-values of MANOVA for F_0, F_0 energy and F_1 energy of FFR for each stimulus are tabulated in Table 4.18. The results indicated that there was a significant reduction in F_0 energy [F (3, 36) = 29.93, p=0.000] and F_1 energy [F (3, 36) = 7.69, p=0.000] between age for /ḍa/ stimulus. Further, for /si/ stimulus, the F_0 energy [F (3, 36) = 3.92, p=0.016] and F_1 energy [F (3, 36) = 13.00, p=0.000] reduced significantly between sub-groups. In order to know the sub-groups that might have accounted for significant difference in F_0 energy and F_1 energy for each stimulus, Duncan post-hoc test was carried out separately for F_0 energy and F_1 energy. The results of Duncan test for F_0 energy to each stimulus revealed that only sub-group ‘D’ was significantly different (p<0.05) from the other sub-groups. Similar, results were obtained for F_1 energy.

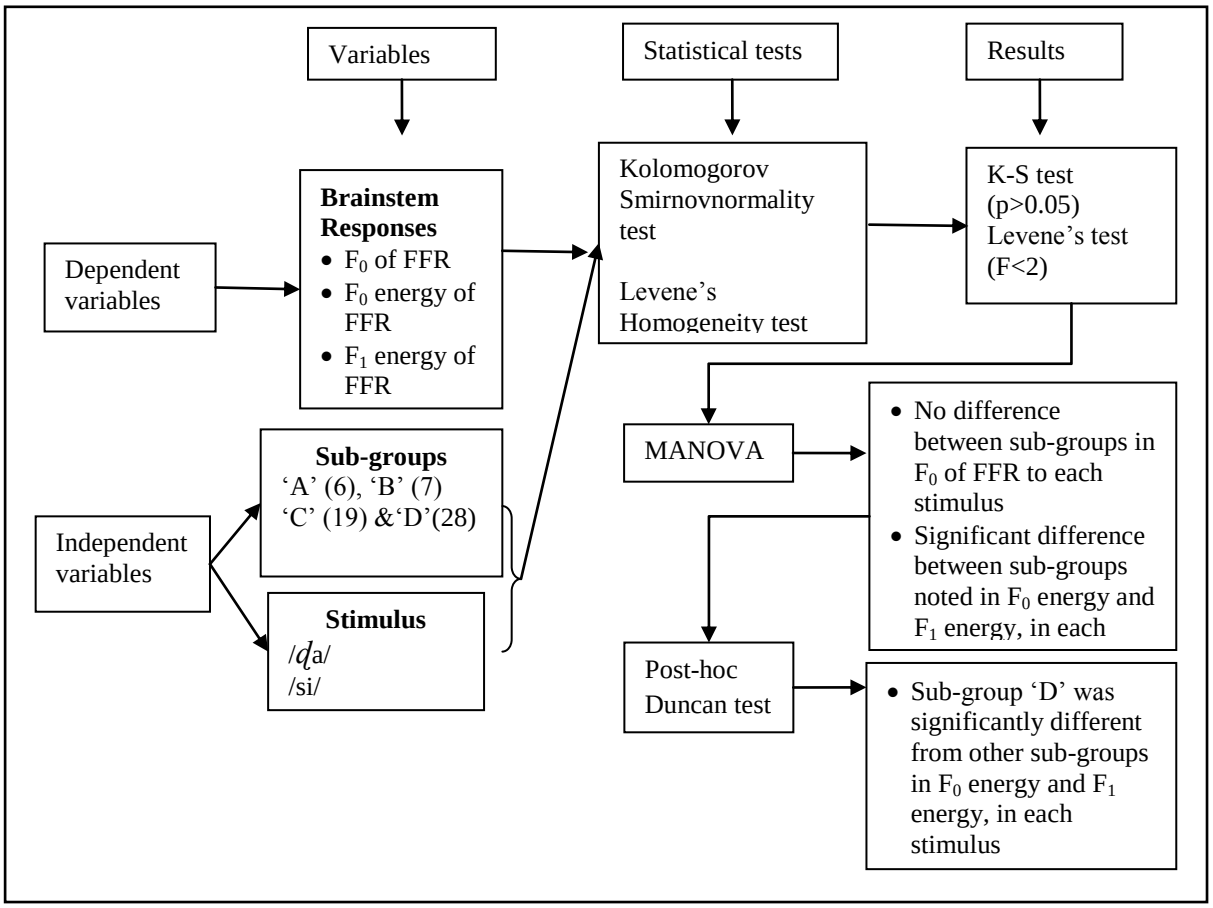


Figure 4.19. Illustrates statistical tests performed on data of F_0, F_0 energy and F_1 energy of FFR obtained from normal hearing sub-groups.

As noted earlier, the F_0 of FFR obtained in the clinical sub-groups of 'A', 'B' and 'C' was significantly different from sub-group 'D' to both the stimuli. Hence, the data from clinical sub-groups of 'A' 'B' and 'C' were combined in the clinical group. In the group with normal hearing, there was no significant difference between the sub-groups in the F_0 of FFR obtained to each stimulus. Though the sub-groups with normal hearing did not differ, only data on F_0 of 'A', 'B' and 'C' sub-groups were combined in order to compare this with the clinical group. Thus, the F_0 obtained from FFR was compared between clinical and normal hearing groups in the combined sub-group of 'ABC'. In addition, the F_0 of FFR was compared separately between sub-group 'D' of clinical and normal hearing groups.

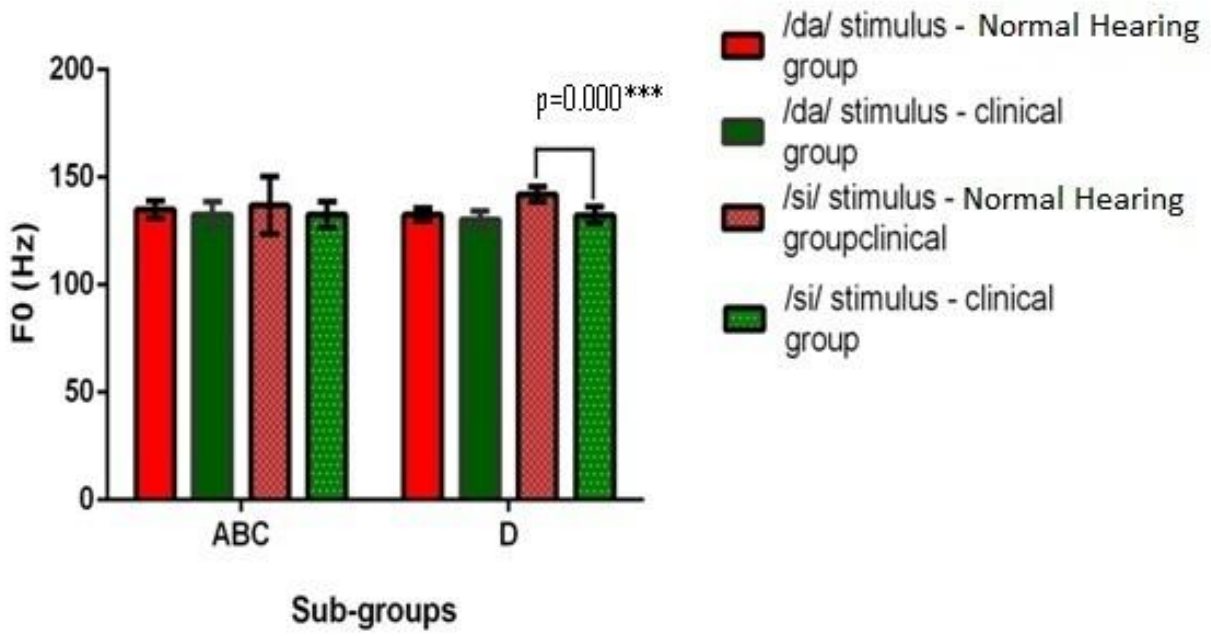


Figure 4.20. Mean and standard deviation of F_0 of FFR to /ɖa/ and /si/ stimuli in combined sub-group 'ABC' and sub-group 'D' of clinical group and normal hearing group.

The mean and standard deviation of F_0 of FFR to each stimulus obtained from clinical group and normal hearing group are tabulated in Figure 4.20. It can be noted that the F_0 of FFR did not differ much in clinical group and in the group with normal hearing in the combined sub-groups of 'ABC'; and in sub-group 'D'. This was true for each stimulus. *Figure 4.21* illustrate statistical tests performed on data of F_0 of FFR obtained from clinical and normal hearing sub-groups. To know the distribution of F_0 of FFR to each stimulus, from combined sub-group of 'ABC'; and sub-group of 'D' in clinical and normal hearing groups, Kolmogorov-Smirnov normality test and Levene's homogeneity test were performed. The result of Kolmogorov-Smirnov revealed that there was no significant difference ($p>0.05$) in the distribution of F_0 of FFR for each stimulus within combined sub-group of 'ABC' and sub-group 'D' in both clinical and normal hearing groups. In Levene's homogeneity test, the variance was less ($F<2$) indicating that the clinical and normal hearing sub-groups of 'ABC' were homogenous in F_0 of FFR for each stimulus. Similar result was noted in sub-group 'D' between clinical and normal hearing groups. The results of normality and homogeneity of

variance indicate that the F_0 of FFR was normally distributed within groups and homogenous between groups. Thus, parametric statistical test was administered. To know if there was any significant difference in the F_0 of FFR to each stimulus between clinical and normal hearing groups, independent samples t-test was performed, separately in combined sub-groups 'ABC'; and sub-group 'D'.

Table 4.19

The mean (M), standard deviation (SD), t-value and p-value of independent samples t-test on F_0 (in Hz) of FFR to /ḍa/ and /si/stimulus, in the normal and clinical sub- groups

Sub-groups	*Groups (No. of participants)*	*F_0 of FFR to /ḍa/ stimulus*			*F_0 of FFR to /si/ stimulus*		
		M±SD	*t-value*	*p-value*	*M±SD*	*t-value*	*p-value*
'ABC'	*Normal hearing(30)*	135.00±4.04	-1.74	0.086	136.92±13.32	-1.33	0.187
	Clinical(32)	132.68±6.14			132.68±6.14		
'D'	*Normal hearing(10)*	132.60±3.08	1.54	0.131	142.11±3.40	-6.80	0.000***
	Clinical(28)	130.40±3.96			132.48±3.97		

Note :*** =p<0.001

The mean, standard deviation, t-value, and p-value of independent samples t-test on F_0 of FFR to each stimulus in clinical and normal hearing groups, are tabulated in Table 4.19. The result revealed that F_0 of FFR did not differ significantly in the clinical and normal hearing combined sub-groups of 'ABC' for each stimulus. There was no significant difference in F_0 of FFR between the clinical and normal hearing groups in sub-group 'D' for /ḍa/ stimulus. However, for /si/ stimulus, there was a significant difference in the encoding of F_0 of FFR between the clinical and normal hearing sub-group of 'D' [t (36) = -6.80, p=000.0], with the F_0 of FFR for /i/ of /si/ being slightly higher in normal hearing than in clinical group.

From this it can be inferred that, the representation of F_0 of FFR for each stimulus does not differ in the combined sub-group ('ABC') of normal hearing and clinical groups; except for F_0 of FFR to /si/ which was higher in normal hearing than in clinical sub-group 'D'.

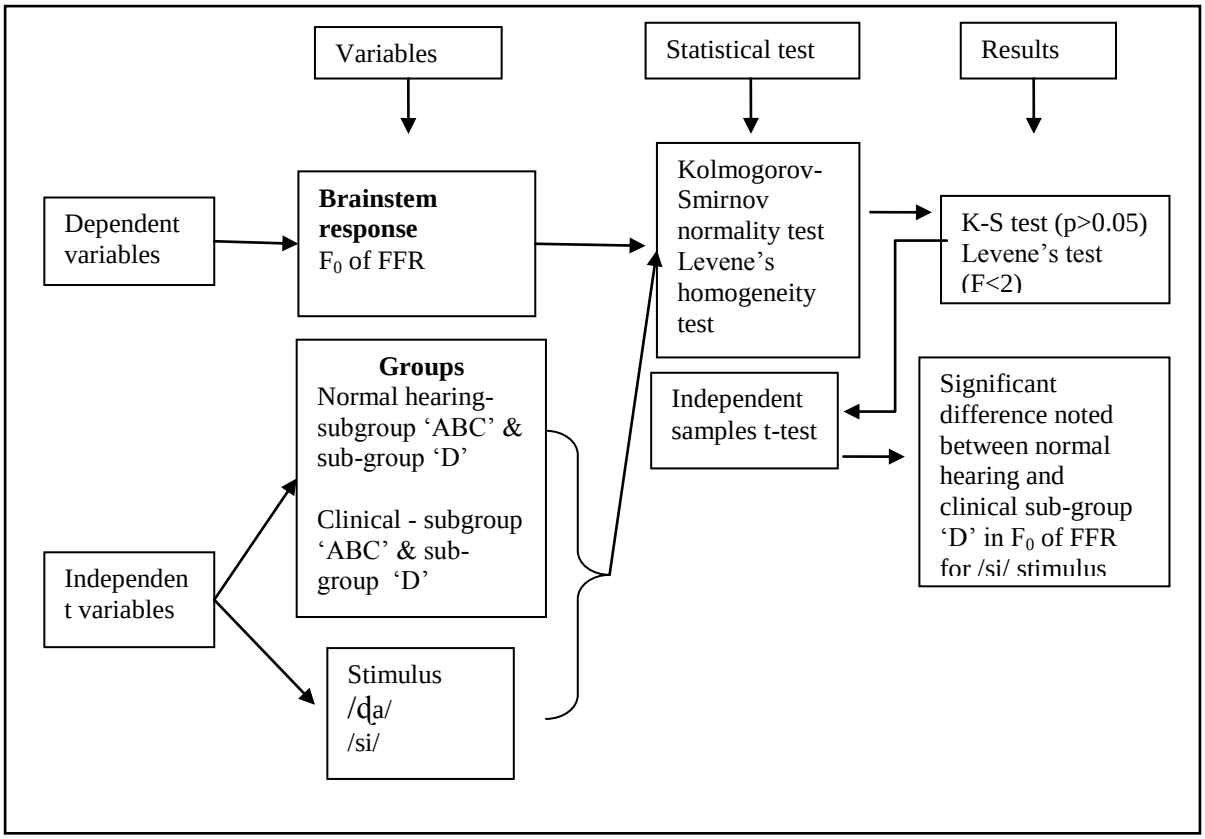


Figure 4.21. Illustrates statistical tests performed on data of F_0 of FFR obtained from clinical and normal hearing sub-groups.

The F_0 energy and F_1 energy obtained from FFR in the clinical sub-groups of 'A' and 'B' were significantly different from sub-groups of 'C' and 'D' for both the stimuli. Further, sub-group 'C' was significantly different from sub-group 'D' in both F_0 energy and F_1 energy of FFR to each stimulus. Hence, the data from clinical sub-groups of 'A' and 'B' were combined. The sub-groups of normal hearing 'A', 'B' and 'C' were significantly different from sub-group 'D' in F_0 energy and F_1 energy obtained from FFR to each stimulus. Thus,

the data on F_0 energy and F_1 energy of FFR to each stimulus from sub-groups of 'A' and 'B' were combined in clinical group. Similarly, this was done for normal hearing groups. Further, F_0 energy and F_1 energy were compared between different sub-groups (i.e., combined 'AB', sub-group 'C' and sub-group 'D') of clinical and normal hearing groups.

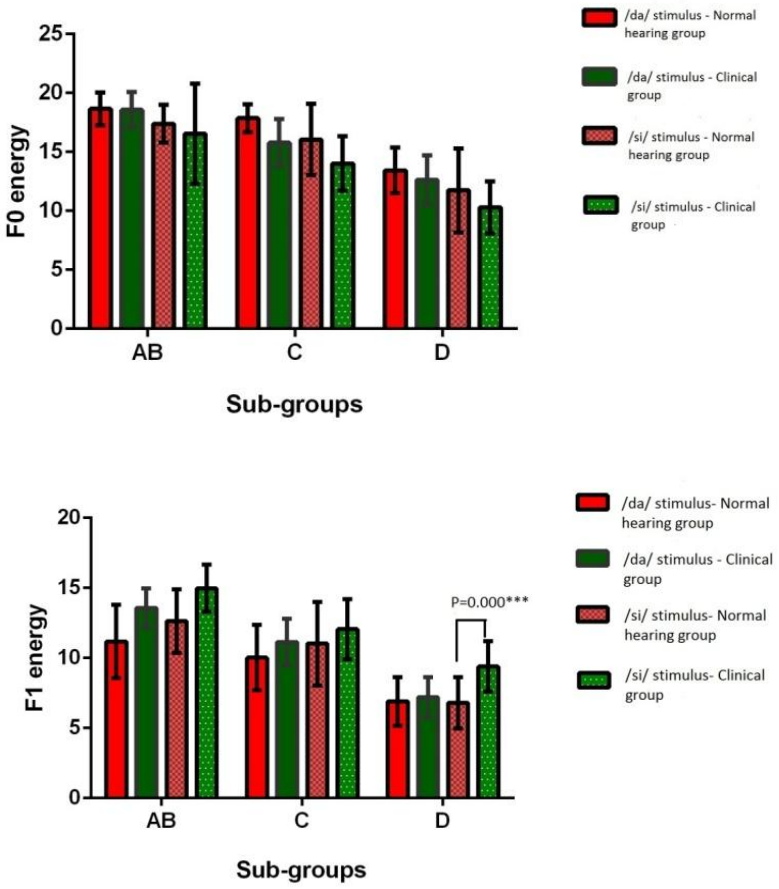


Figure 4.22. Mean and standard deviation F_0 energy and F_1 energy (in absolute unit) of FFR in sub-groups 'AB', 'C' and 'D' of clinical and normal hearing sub-groups for /ɖa/ and /si/ stimuli.

Table 4.20

Mean (M) and standard deviation (SD) of F_0 energy and F_1 energy of FFR to each stimulus in sub-groups 'AB','C' and 'D' of group with normal hearing and clinical sub-group

Sub-groups	*Groups (No. of Participants)*	*/ḍa/ stimulus*		*/si/ stimulus*	
		F_0 energy	*F_1 energy*	*F_0 energy*	*F_1 energy*
		M±SD	*M±SD*	*M±SD*	*M±SD*
'AB'	*Normal hearing (20)*	18.65±1.39	13.57±1.39	17.39±1.61	14.98±1.69
	Clinical (13)	18.58±1.51	11.18±2.62	16.55±4.26	12.63±2.28
'C'	*Normal hearing (10)*	17.86±1.20	11.14±1.67	16.06±3.01	12.05±2.14
	Clinical (19)	15.79±2.01	10.03±2.34	14.02±2.30	11.03±2.98
'D'	*Normal hearing (10)*	13.43±1.93	7.21±1.43	11.73±3.56	9.40±1.79
	Clinical (28)	12.61±2.10	6.30±1.75	10.30±2.20	6.80±1.82

The mean and standard deviation of F_0 energy and F_1 energy of FFR for /ḍa/ and /si/ stimulus in clinical and normal hearing groups are provided in Table 4.20.The F_0 energy and F_1 energy of FFR (Figure 4.22) obtained to each stimulus was higher in the group with normal hearing than in clinical group, in combined sub-group of 'AB'. This was true for sub-group 'C' and sub-group 'D'.

Table 4.21

The t-value and p-value of independent samples t-test on F_0 energy and F_1 energy of FFR to both stimuli between normal and clinical sub-groups

Sub-groups	*Groups (N)*	*/ḍa/ stimulus*				*/si/ stimulus*			
		F_0 energy		*F_1 energy*		*F_0 energy*		*F_1 energy*	
		t-value	*p-value*	*t-value*	*p-value*	*t-value*	*p-value*	*t-value*	*p-value*
'AB'	*Normal hearing (20)*	-1.44	0.886	0.62	0.557	0.59	0.557	-1.34	0.186
	Clinical (13)								
'C'	*Normal hearing (10)*	-0.06	0.945	1.47	0.153	-1.81	0.080	1.53	0.137
	Clinical (19)								
'D'	*Normal hearing(10)*	-1.08	0.287	0.55	0.584	-1.49	0.144	3.90	0.000***
	Clinical (28)								

Note: ***= p<0.001

Figure 4.23 illustrates the statistical tests performed on data of F_0 energy and F_1 energy of FFR for each stimulus obtained from clinical and normal hearing sub-groups. In order to know the distribution of F_0 energy and F_1 energy for each stimulus in different sub-groups of clinical and normal hearing groups for each stimulus, normality test of Kolmogorov-Smirnov and Levene's homogeneity tests were performed. The former test results ($p>0.05$) indicated that the F_0 energy and F_1 energy obtained from FFR for each stimulus were normally distributed, within different sub-groups of clinical and normal hearing groups. Additionally, the F_0 energy and F_1 energy were homogenous ($F<2$) between clinical and normal hearing groups, in both combined sub-group of 'AB' for each stimulus.

Similar result was noted in sub-group 'C' and sub-group 'D'. Thus, parametric statistical test was conducted. The means of F_0 energy and F_1 energy for each stimulus in combined sub-groups 'AB'; sub-group 'C' and sub-group 'D' between clinical and normal hearing groups were subjected to independent samples t-test. The t-value and p-value of independent samples t-test of F_0 energy and F_1 energy are tabulated in Table 4.21. The result revealed a significantly higher F_1 energy of FFR to /si/ stimulus 'D' [t (36) = 3.90, p=0000] in clinical sub-group 'D' than in normal hearing sub-group.

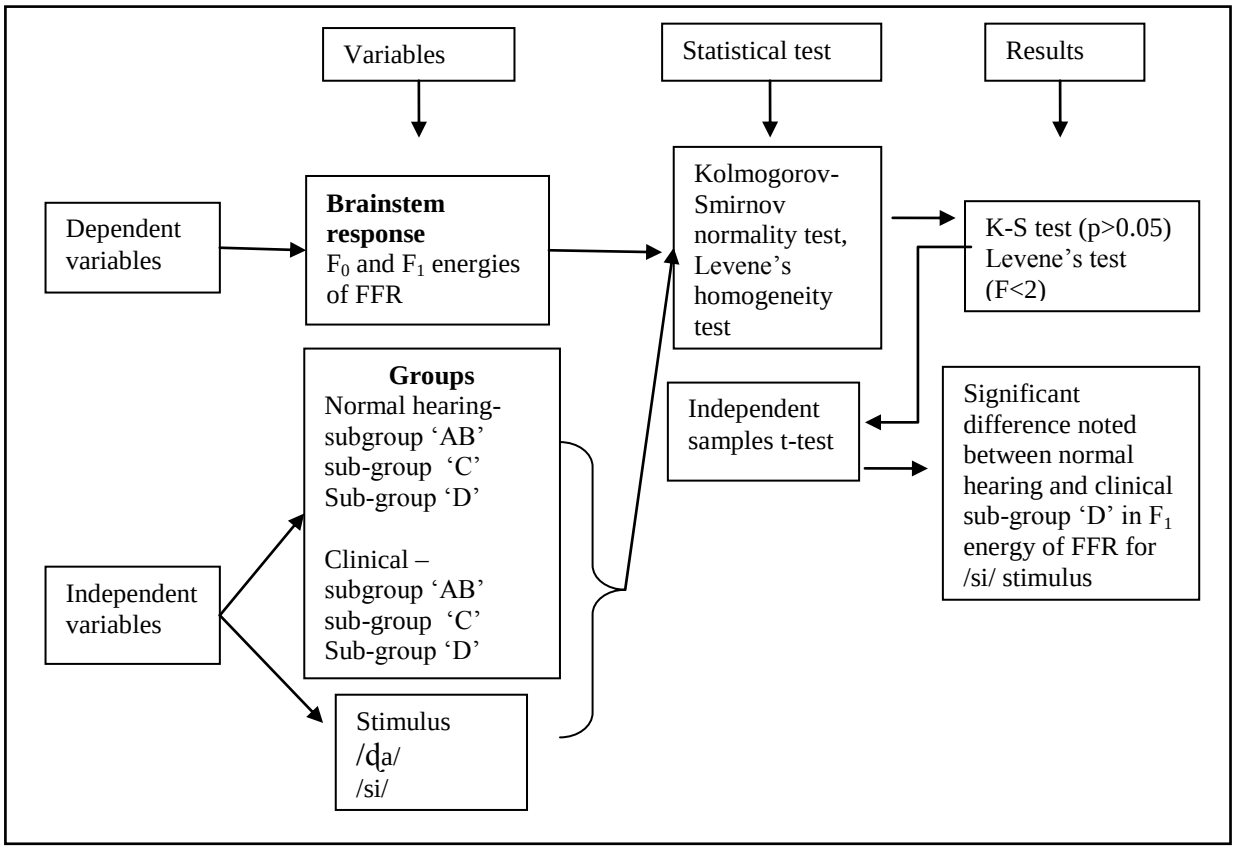


Figure 4.23. Illustrates statistical tests performed on data of F_0 energy and F_1 energy of FFR obtained from clinical and normal hearing sub-groups.

At cortical level, the representation of speech syllables in clinical group and normal hearing group

In clinical sub-group, the slope of N1-P2 of LLR to /ɖa/ stimulus; and latency and amplitude of different components of ACC to /si/ stimulus recorded at 65 dB SPL in unaided and aided conditions were analyzed. The responses were absent in the unaided condition as the signal audibility was not sufficient to elicit the response. Further in each participant of group with normal hearing, the slope of N1- P2 of LLR for /ɖa/ stimulus; and the latency and amplitude of ACC components for /si/ stimulus obtained in the unaided condition were tabulated.

The data from aided response obtained at cortical level of auditory pathway in clinical sub-groups were combined, if there was no significant difference. Likewise, the response (unaided) obtained in sub-groups with normal hearing was combined if there was no significant difference. Further, the aided response from the cortical level of the auditory pathway from clinical sub-groups was compared with unaided response from sub-groups with normal hearing. This was done to investigate if the aided responses approximated the response from a normal auditory pathway.

Comparison of aided slope of N1-P2 in clinical group with unaided slope of N1-P2 in group having normal hearing. The slope of N1-P2 of LLR in the aided condition from clinical sub-groups was analyzed with the following statistical analysis.

1. Descriptive analysis was done on the slope of N1-P2 of LLR.
2. Kruskal-Wallis test was performed separately to know if there were any significant differences between sub-groups in the mean of slope of N1-P2 (Figure 4.25).

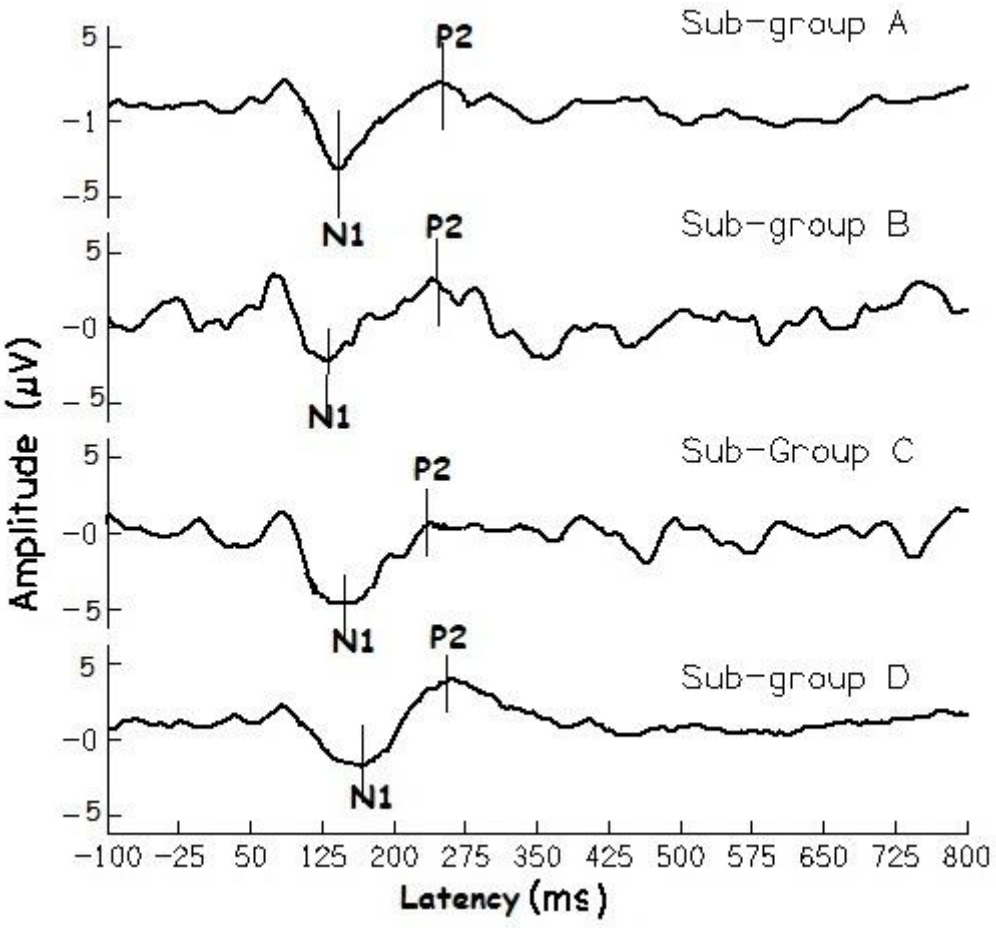


Figure 4.24. Grand average wavform of LLR obtained for /ɖa/ stimulus from four sub-groups of clinical group. N1 and P2 latencies were prolonged with respect to age.

The LLRs for /ɖa/ stimulus obtained from four clinical sub-groups in the aided condition are represented in Figure 4.24. The mean and standard deviation of the slope of N1-P2 of LLR among clinical sub-groups are tabulated in Table 4.22. It was found that the mean value of slope of N1-P2 was shallower with respect to age. Kruskal-Wallis test was performed to see if there was any difference in the mean slopes of N1-P2 between sub-groups. The result indicated that there was no significant difference in the slope of N1-P2 in the sub-groups.

Table 4.22

Mean (M), standard deviation (SD), χ^2 (3) and p-values of slope of N1-P2 (in µV/ms*) to /ḍa/ stimulus in clinical sub-groups*

	Slope of N1-P2		
Clinical Sub-groups (No. of participants)	*M±SD*	*χ^2-value*	*p value*
'A' (6)	0.65±0.025		
'B' (7)	0.58±0.007		
'C' (19)	0.49±0.019	7.620	0.055
'D' (28)	0.45±0.016		

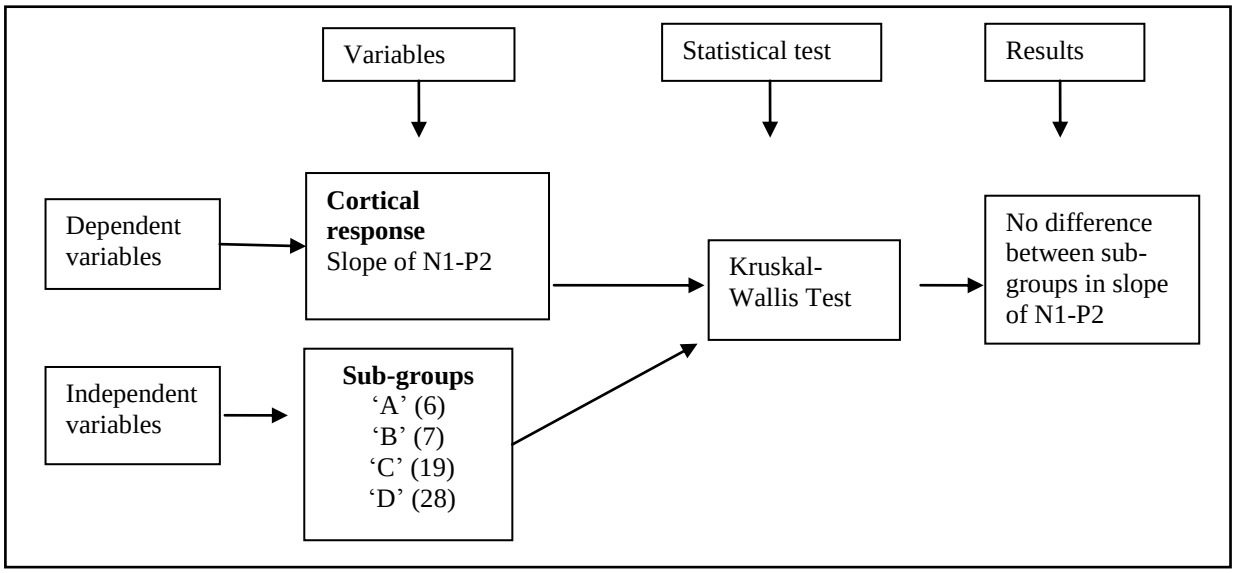


Figure 4.25. Illustrates statistical test performed on data of slope of N1-P2 obtained from clinical sub- groups.

The following statistical analyses were carried out on the data of slope N1- P2 of LLR for /da/ stimulus recorded from the normal hearing sub-groups in the unaided condition.

1. Descriptive analysis among sub-groups was performed for slope of N1-P2.
2. Kolmogorov-Smirnov normality test was performed to know the distribution of data within each sub-group and Levene's homogeneity test was conducted to know the homogeneity of data across sub-groups**.**

3. ANOVA was administered to evaluate the significant difference across the sub-groups in the slope of N1-P2.

The grand average waveform of LLR obtained from normal hearing sub-groups is represented in Figure 4.26. The mean value of slope of N1-P2 reduced with age indicating a shallower slope as factor of age.

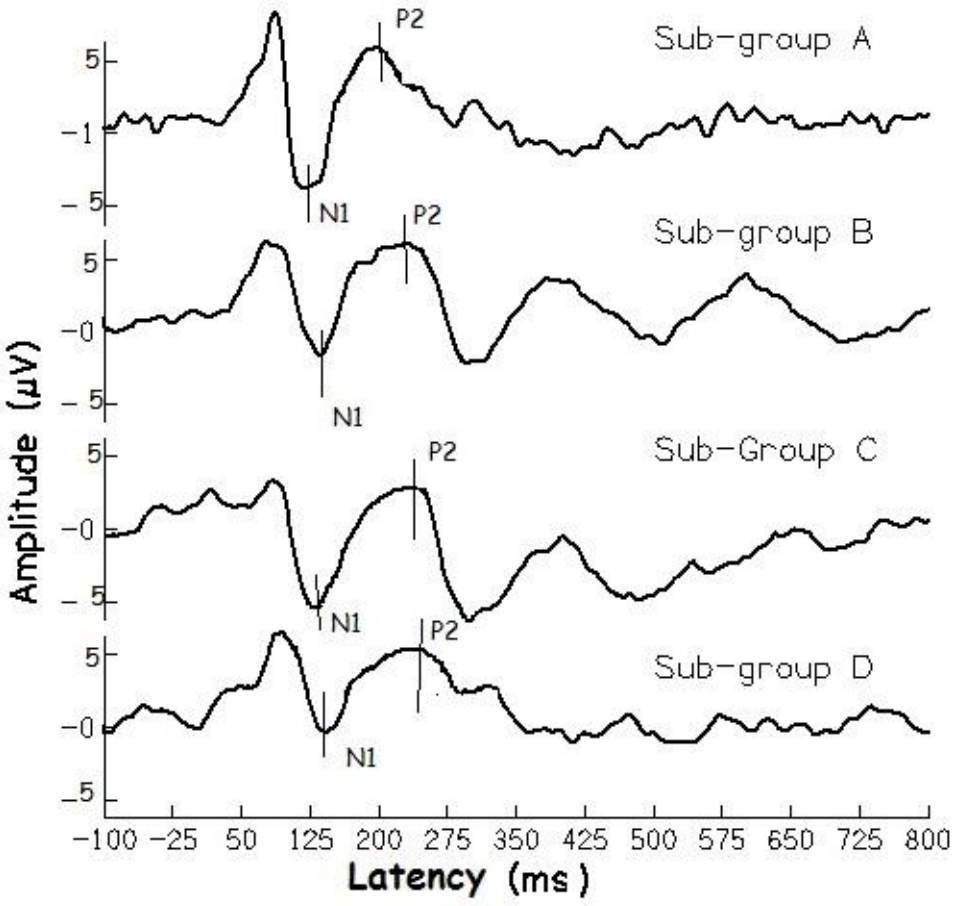


Figure 4.26. Grand average waveform of LLR obtained from normal hearing sub-groups. N1 and P2 registered at the auditory cortex for /ɖa/ stimulus.

Figure 4.27 illustrates statistical tests performed on data of slope of N1-P2 obtained from normal hearing sub-groups.The Kolmogorov-Smirnov normality test and Levene's homogeneity test were performed to know if the data on slope of N1-P2 were normally distributed and homogenous, within each group and between the normal hearing sub-groups. The result of normality test revealed that the data were normally distributed in each group.

The Levene's test indicated that the data were homogeneous between sub-groups (F ratio < 2). Thus, parametric ANOVA test was administered to see if there was any significant difference in the means of the slope of N1-P2 between the sub-groups. The results indicated that there was no significant difference between sub-groups on the slope of N1-P2 in the group with normal hearing.

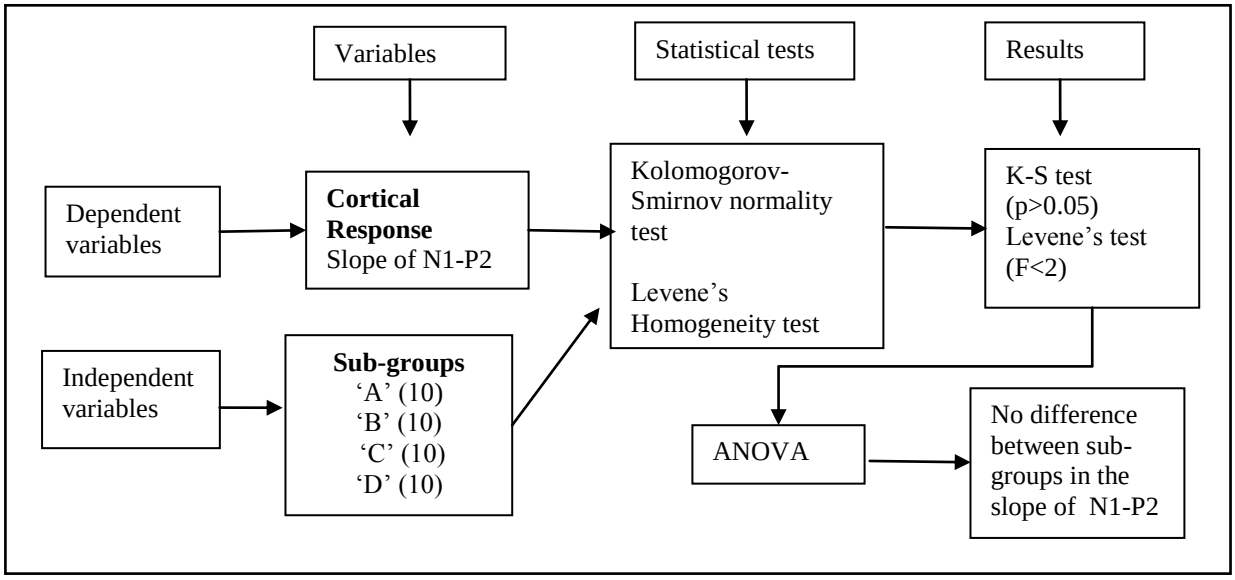


Figure 4.27. Illustrates statistical tests performed on data of slope of N1-P2 obtained from normal hearing sub-groups.

As noted earlier, the slope of N1-P2 revealed no significant difference between sub-groups in both clinical and normal groups. Thus, the slope of N1-P2 obtained from different sub-groups was combined separately for clinical group and group with normal hearing. Further, the slope of N1-P2 was compared between clinical and normal hearing groups. Figure 4.29. Illustrate statistical tests performed on data of slope of N1-P2 obtained from clinical and normal hearing sub-groups

Normality test of Kolmogorov-Smirnov and Levene's homogeneity test were performed to know the distribution of slope of N1-P2 in clinical and normal hearing groups. The result revealed that the data on the slope of N1-P2 were normally distributed (p>0.05) in

normal hearing group and clinical groups. The data on the slope of N-P2 were homogenous between clinical and normal hearing groups, as the variance was less (F<2). The results of normality and homogeneity tests indicated that parametric test was required to analyze the difference between the clinical and normal hearing groups. Thus, independent samples t-test was performed. The mean, standard deviation, t-value, and p-value of independent samples t-test on the slope of N1-P2 are tabulated in Table 4.23.

Table 4.23

Mean (M), standard deviation (SD,) t-value and p-value of independent samples t-test on slope of N1-P2 of LLR in normal hearing and clinical groups

Groups (No. of Participants)	*Slope of N1-P2 (in* µV/ms)		
	M ±*SD*	*t-value*	*p-value*
Normal hearing (40)	0.06±0.02	-3.11	0.002**
Clinical (60)	0.05±0.01		

Note: ** = p<0.01

The slope of N1-P2 of LLR (Figure 4.28) was steeper in the group with normal hearing than in clinical group. This difference in slope of N1-P2 was found significant on independent samples t-test [t (98) = -3.11, p=0.002].

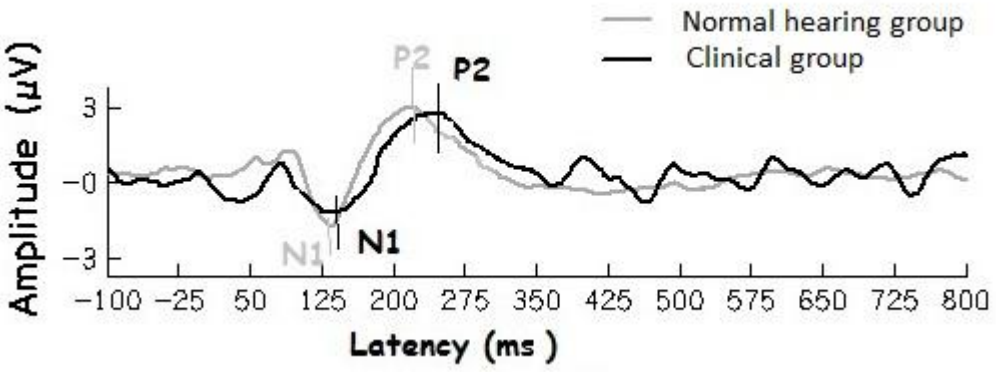


Figure 4.28. Grand average waveform of LLR obtained from clinical and normal hearing groups. Latency of N1 and P2 is prolonged in the clinical group compared to the normal haring group.

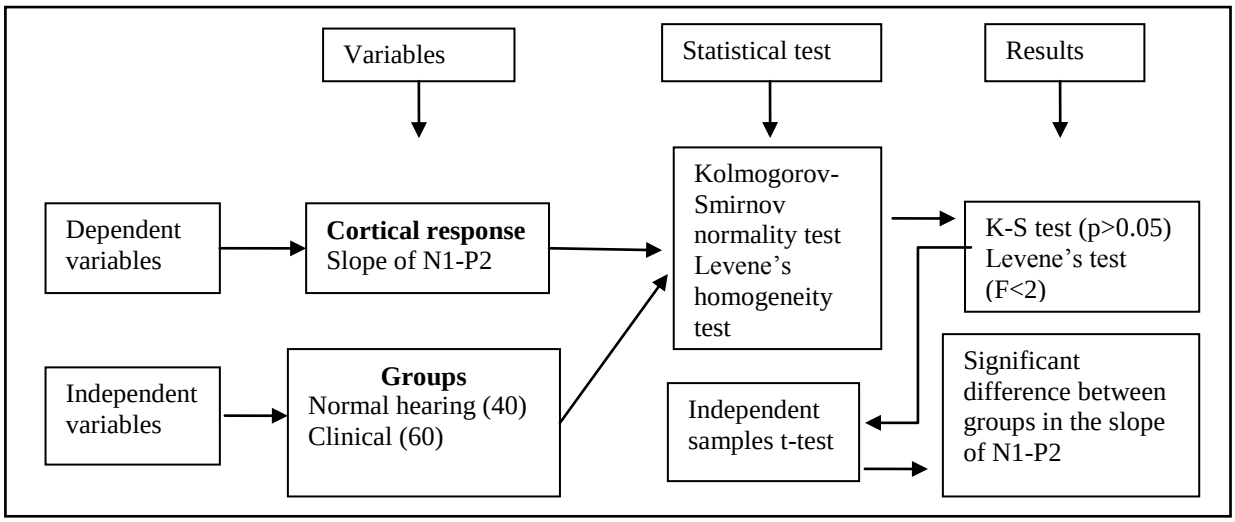


Figure 4.29. Illustrates statistical tests performed on data of slope of N1-P2 obtained from clinical and normal hearing sub-groups.

Comparison of latency of ACC components in aided condition from clinical group with unaided condition from group having normal hearing. The latency of components of ACC in the aided condition from the clinical sub-groups was analyzed with the following statistical analysis.

1. Descriptive analysis was done on the latencies of onset and transition components of ACC in each sub-group.
2. Kruskal-Wallis test was performed separately to know if there were any significant differences between sub-groups in the mean latency of ACC components.
3. Mann-Whitney U test was performed when indicated.

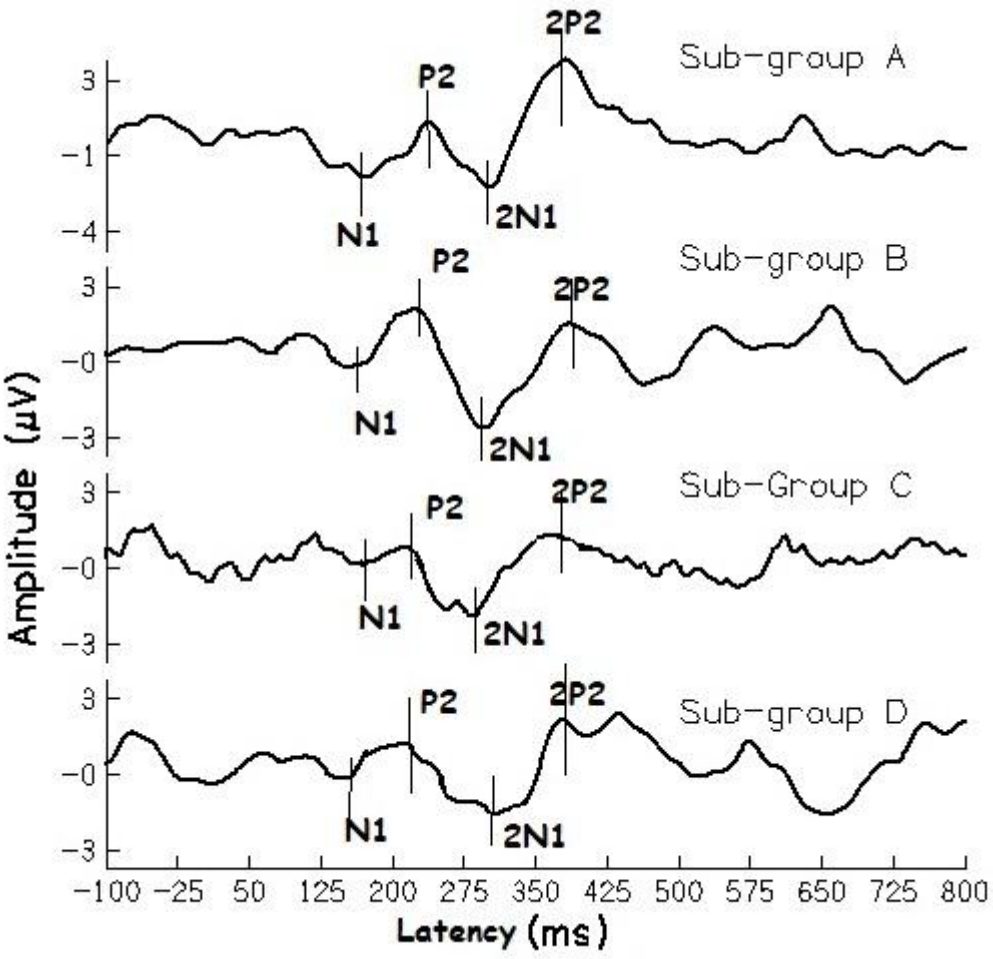


Figure 4.30. Grand average waveform of ACC obtained for /si/ stimulus from four clinical sub-groups. Latencies of N1 and P2 corresponds to onset of consonant prolonged with age. Similarly, latencies of 2N1 and 2P2 corresponds to onset of vowel prolonged with age.

The mean and standard deviation of latency of ACC components for /si/ stimulus are tabulated in Table 4.24. The mean onset latencies of N1 and P2 were prolonged with age (Figure 4.30). Similar findings were obtained in mean transition latencies of 2N1 and 2P2 (Figure 4.30).

Table 4.24

Mean (M) and standard deviation (SD) of latency of ACC components in each clinical sub-group

Clinical Sub-groups (No. of participants)	*Latency (ms)of ACC components*			
	N1	*P2*	*2N1*	*2P2*
	M±SD	*M±SD*	*M±SD*	*M±SD*
'A' (6)	136.16±2.13	200.16±12.27	280.16±5.70	365.50±12.04
'B' (7)	140.85±4.22	230.00±9.55	285.28±7.78	384.00±2.44
'C' (19)	145.31±12.65	239.16±10.12	287.78±8.63	384.63±7.88
'D' (28)	152.92±15.36	240.28±6.95	292.32±8.20	382.71±18.03

Figure 4.31 illustrate statistical test performed on data of latency of ACC obtained from clinical sub-groups. Non-parametric Kruskal-Wallis test was performed to know if there was any significant difference between sub-groups in latency of each component of ACC. The result revealed a significantly prolonged latencies in the onset components of ACC between the sub-groups, i.e., N1 [χ^2 (3) = 18.89, p=0.000] and P2 [χ^2 (3) = 14.56 p=0.002] of ACC. Further, the latencies of 2N1 [χ^2 (3) = 11.49, p=0.009] and 2P2 [χ^2 (3) = 7.83, p=0.049] found significantly prolonged between sub-groups, i.e., transition components of ACC. Further, in order to know the sub-groups that might have caused the difference in latencies of ACC components, Mann-Whitney *U* test was performed. The /U/ value and p-value for each component of ACC in sub-groups are tabulated in Table 4.25.

Table 4.25

/U/ and the p values of Mann-Whitney U test on different sub-groups in onset and transition response of ACC.

Sub-groups	*ACC-Onset response*				*ACC-Transition response*			
	N1		*P2*		*2N2*		*2 P2*	
	/U/	*p-value*	*/U/*	*p-value*	*/U/*	*p-value*	*/U/*	*p-value*
'A' vs. 'B'	-0.30	0.76	-0.33	0.700	-1.14	0.250	-0.37	0.410
'A' vs. 'C'	-1.42	0.11	-1.46	0.430	-1.56	0.110	-1.76	0.110
'A' vs. 'D'	-2.98	0.000***	-2.71	0.003**	-3.03	0.002**	-2.35	0.018*
'B' vs. 'C'	-0.78	0.432	-0.26	0.200	-0.69	0.486	-0.61	0.542
'B' vs. 'D'	-3.03	0.000***	-2.66	0.002**	-1.90	0.050*	-0.24	0.000***
'C' vs. 'D'	-2.30	0.017*	-3.02	0.003**	-2.75	0.002**	-2.87	0.000***

Note: * =p<0.05; ** = p<0.01; *** =p<0.001

The results of Mann-Whitney *U* test revealed that sub-groups 'A', 'B' and 'C' were significantly different from sub-group 'D' in terms of latencies of each component of ACC.

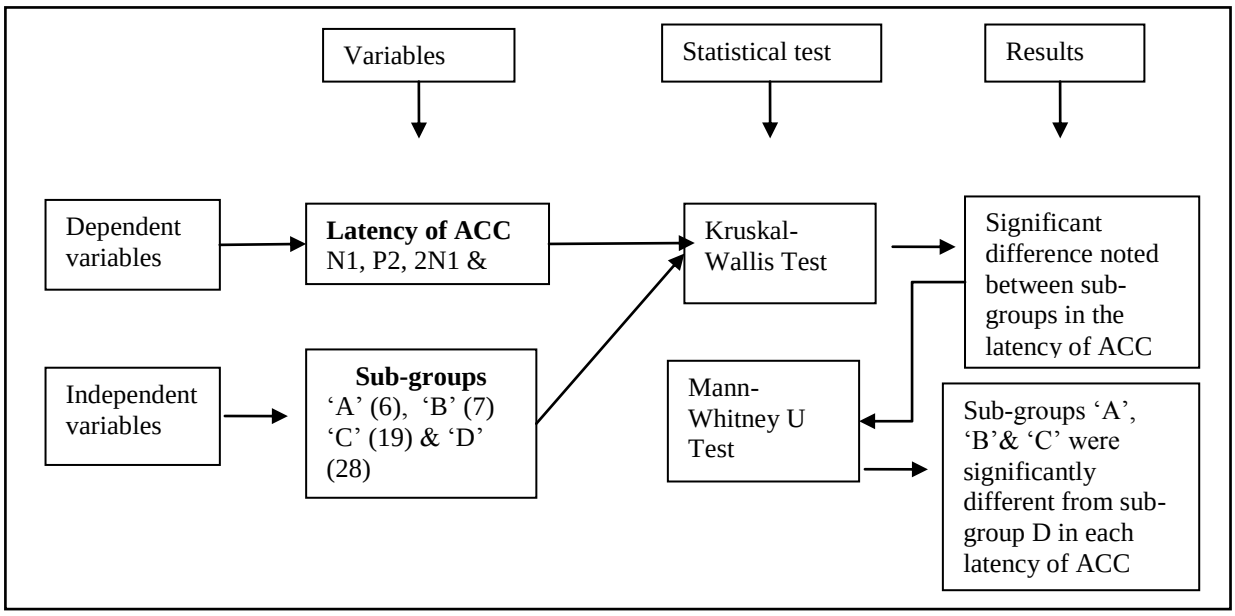


Figure 4.31. Illustrates statistical test performed on data of latency of ACC obtained from clinical sub-groups.

Further, the data on the latency of ACC components for /si/ stimulus from each participant of group with normal hearing were tabulated for the unaided condition. The following statistical analyses were carried out.

1. Descriptive analysis among sub-groups was performed for latency of ACC components.
2. Kolmogorov-Smirnov normality test was done to know the distribution of data within each sub-group and Levene's homogeneity test was carried out to know the distribution of data across sub-groups.
3. MANOVA was carried out to know if there were significant differences between the sub-groups in the mean values of latency of ACC components.

The mean and standard deviation of latencies of ACC are tabulated in Table 4.26. The mean latencies of N1 and P2 (corresponding to the onset of stimulus) were prolonged with age (Figure 4.32). Similar findings were obtained in the mean latencies of 2N1 and 2P2 corresponding to the transition portion of the stimulus (Figure 4.32).

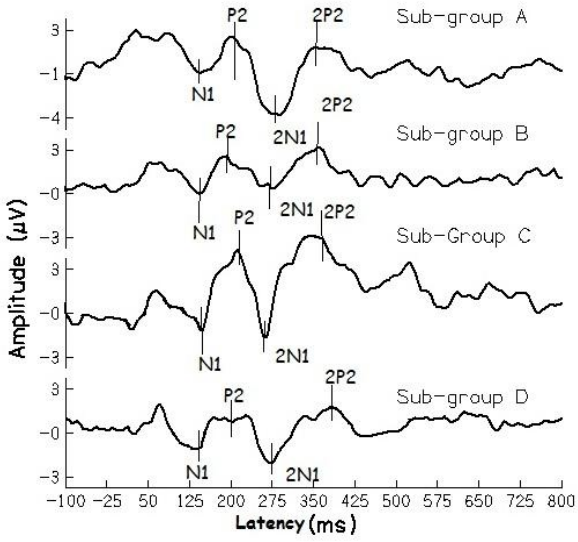


Figure 4.32. Grand average waveform of ACC obtained for /si/ stimulus. N1 and P2 correspond to onset of consonant and 2N1 and 2P2 corresponds to transition from consonant to vowel.

Table 4.26

Mean (M) and standard deviation (SD) of latency of ACC components in sub-groups with normal hearing

Sub-groups (No. of participants)	*Latency of ACC components (ms)*			
	N1	*P2*	*2N1*	*2P2*
	M±SD	*M±SD*	*M±SD*	*M±SD*
'A' (10)	141.00±22.18	202.00±10.40	271.20±11.54	350.80±10.40
'B' (10)	147.90±16.90	208.50±17.95	271.60±09.83	359.70±17.95
'C' (10)	154.60±15.55	209.70±20.13	276.10±03.44	365.30±20.13
'D' (10)	154.90±18.03	210.10±06.70	278.00±15.50	372.20±06.70

Figure 4.33 illustrates statistical tests performed on data of latency of ACC obtained from normal hearing sub-groups. Kolmogorov-Smirnov normality test and Levene's homogeneity test were performed to check the distribution and homogeneity of data. The result of normality test revealed no significant difference ($p>0.05$) in each sub-group and Levene's test revealed that there was no variance in latency of each of the component of ACC between the sub-groups ($F < 2$). As each component of ACC was normally distributed, parametric test of MANOVA was performed to see if there was a significant difference between the sub-groups in the latencies of ACC at the onset (N1-P2) and transition (2N1-2P2). It was found that there was no significant effect of age on the latencies at the onset and transition components of ACC for /si/ stimuli.

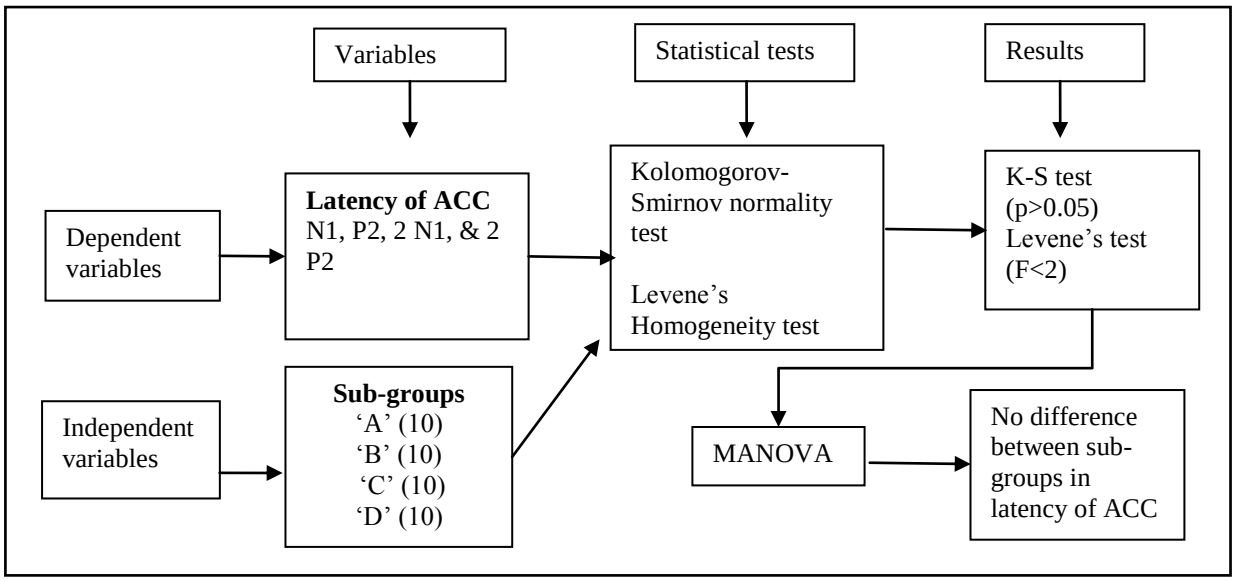


Figure 4.33. Illustrates statistical tests performed on data of latency of ACC obtained from normal hearing sub-groups.

As mentioned earlier, the sub-groups 'A', 'B' and 'C' in the clinical group differed significantly from the sub-group 'D' in the latencies of ACC components. However, there was no significant difference in the latency of each of the ACC components between sub-groups of those with normal hearing. Except for sub-group 'D', latency of each component of ACC was combined from sub-groups 'A', 'B' and 'C', in both clinical and normal hearing groups. Though the four sub-groups with normal hearing group did not differ, only data on latency of ACC components of 'A', 'B' and 'C' were combined in order to compare this with the clinical group. The latency of each component of ACC was compared between clinical group and the group having normal hearing.

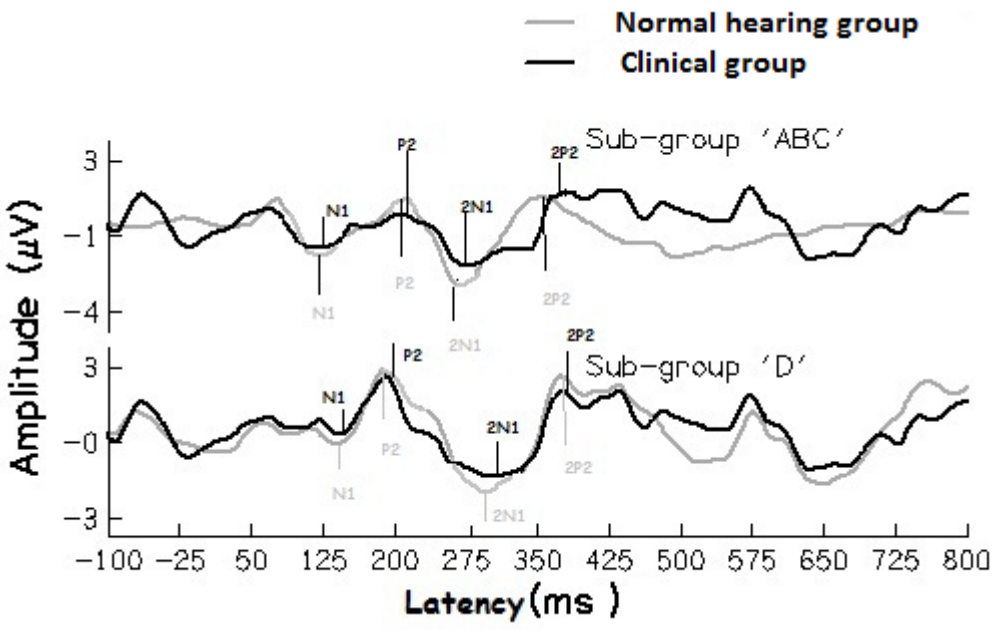


Figure 4.34. Representation of grand average waveform of ACC obtained from normal hearing group and clinical sub-groups. The latency of ACC is prolonged in clinical group compared to normal hearing group. This is true for sub-groups 'ABC' and 'D'.

The grand average waveform of ACC obtained from clinical and normal hearing sub-groups is represented in Figure 4.34. In combined sub-group of 'ABC' and sub-group 'D', the N1 latency was earlier in the group with normal hearing than in the clinical group. Similar pattern of result was noted in latencies of P2, 2N1 and 2P2. Figure 4.35 illustrates statistical tests performed on data of latency of ACC obtained from clinical and normal hearing sub-groups.

The normality test of Kolmogorov-Smirnov and Levene's homogeneity test were performed on latency of each component of ACC on different sub-groups (i.e., in 'ABC' and 'D' sub-groups) of clinical group and group with normal hearing. The results revealed that the data were normally distributed ($p>0.05$), within 'ABC' and 'D' sub-groups of clinical and normal hearing groups in each latency of ACC. The data on latency of each of the ACC component were homogenous on Levene's test, between clinical and normal hearing sub-group of 'ABC'; and sub-group 'D'. Thus, parametric independent samples t-test was conducted.

The mean, standard deviation, t-value and p-value of independent samples t-test on latency of ACC are tabulated in Tables 4.27 and 4.28. The latencies of all the ACC components were prolonged in clinical sub-groups than in normal hearing sub-groups. In order to know if these differences were significant, independent samples t-test was administered. There was a significant difference between clinical group and group with normal hearing in terms of latency of all the components of ACC, except for latency of N1 in the combined sub-groups of 'ABC' and sub-group 'D'; and also for the latency of 2P2 in sub-group 'D'.

Table 4.27

Mean (M), standard deviation (SD), t-values and p-values of independent samples t-test on latencies of N1 and P2 in normal hearing group and clinical sub-groups

Sub-groups	*Groups (No. of participants)*	*N1 latency (ms)*			*P2 latency (ms)*		
		M ±SD	*t-value*	*p-value*	*M±SD*	*t-value*	*p-value*
'ABC'	*Normal hearing (30)*	142.62±10.34	-1.36	0.177	206.73±16.46	5.02	0.000***
	Clinical (32)	147.83±18.66			229.85±19.51		
'D'	*Normal hearing (10)*	152.92±15.36	-0.33	0.741	220.10±6.70	7.94	0.000***
	Clinical (28)	154.90±18.03			240.28±6.95		

Note: ***= p<0.001

Table 4.28

Mean (M), standard deviation (SD), t-values and p-values of independent samples t-test on 2 N1 and 2 P2 latencies in normal and clinical sub-groups

		2 N1 latency (ms)			*2 P2 latency (ms)*		
Sub-groups	*Groups (No. of participants)*	*M ±SD*	*t-value*	*p-value*	*M±SD*	*t-value*	*p-value*
'ABC'	*Normal hearing (30)*	272.96±8.95	5.82	0.000***	358.60±26.61	4.37	0.000***
	Clinical(32)	295.81±8.30			380.90±10.82		
'D'	*Normal hearing (10)*	278.00±15.50	5.86	0.001**	372.20±20.04	1.58	0.133
	Clinical(28)	292.32±8.20			382.17±18.03		

Note: **p<0.01; ***p<0.001

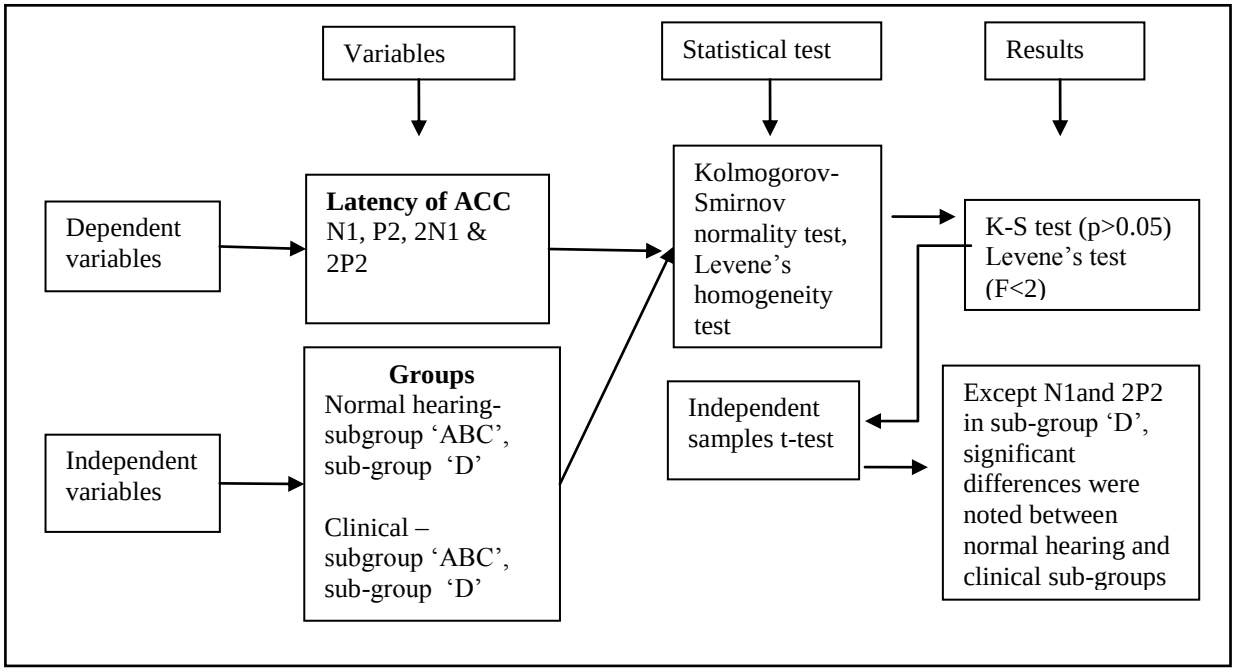


Figure 4.35. Illustrates statistical tests performed on data of latency of ACC obtained from clinical and normal hearing sub-groups.

Comparison of amplitude of ACC components in aided condition from clinical group with unaided condition from group having normal hearing. The amplitude of ACC in the aided condition from the clinical sub-groups was analyzed with the following statistical analysis.

1. Descriptive analyses were done on the amplitudes of onset and transition components of ACC in each sub-group.
3. Kruskal-Wallis test was performed separately to know if there were any significant differences between sub-groups in the mean amplitude of ACC components.
4. Mann-Whitney *U* test was performed when indicated.

The mean onset amplitudes of N1 and P2 of ACC reduced with age (Table 4.29). Similar findings were obtained in 2N1 and 2P2 transition amplitudes of ACC (Table 4.29).

Table 4.29

Mean (M) and standard deviation (SD) of amplitude of ACC in each clinical sub-groups.

Sub-groups (No. of participants)	*Amplitude of ACC (µV)*			
	N1	*P2*	*2N1*	*2P2*
	M±SD	*M±SD*	*M±SD*	*M±SD*
'A' (6)	-1.24±0.19	1.64±0.30	-1.91±0.44	1.69±0.72
'B' (7)	-1.16±0.59	1.51±0.21	-1.71±0.62	1.24±0.32
'C' (19)	-0.85±0.69	1.08±0.69	-1.39±0.43	1.40±0.71
'D' (28)	-0.66±0.30	0.74±0.49	-1.39±0.71	1.03±0.79

Figure 4.36 illustrates statistical tests performed on data of amplitude of ACC obtained from clinical sub-groups. The Kruskal-Wallis test was performed to see whether any significant differences were present between sub-groups in the onset and transition amplitudes of ACC. The result revealed that except 2 P2 amplitude, a significantly smaller amplitudes of N1 [χ^2 (3) = 11.85, p=0.008], P2 [χ^2 (3) = 20.94, p=0.000] and 2N1 [χ^2 (3) = 15.36, p=90.050] components of ACC were found between the clinical sub-groups. Further, in order to know the sub-groups that might have caused the difference in amplitudes of ACC components, Mann-Whitney *U* test was performed. The /U/ value and p-value of Mann-Whitney *U* test at the onset and transition components of ACC in different sub-groups are provided in Table 4.30.

Table 4.30

The /U/ value and p-value of Mann-Whitney U test on onset and transition amplitudes of ACC

Clinical Sub-groups	*Onset response*				*Transition response*	
	N1		*P2*		*2 N1*	
	/U/	*p-value*	*/U/*	*p-value*	*/U/*	*p-value*
'A' vs. 'B'	-0.28	0.774	-1.88	0.060	-1.15	0.250
'A' vs. 'C'	-1.36	0.171	-1.37	0.318	-1.91	0.056
'A' vs. 'D'	-3.48	0.000***	-3.21	0.001**	-1.98	0.047*
'B' vs. 'C'	-0.95	0.340	-1.56	0.230	-1.18	0.235
'B' vs. 'D'	-1.55	0.010*	-3.09	0.021*	-2.80	0.000***
'C' vs. 'D'	-2.65	0.002**	-1.98	0.047*	-3.45	0.000***

Note: * =p<0.05; ** = p<0.01; *** =p<0.001

The results of Mann-Whitney *U* test revealed that sub-groups 'A', 'B' and 'C' were significantly different in the amplitude of components of ACC from sub-group 'D'.

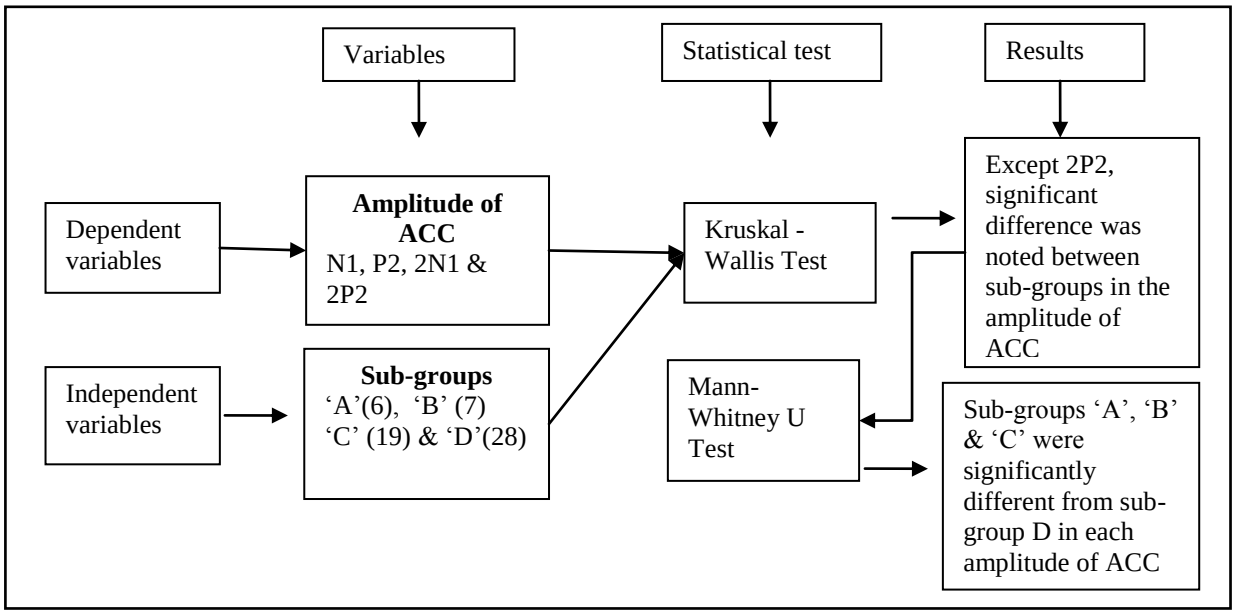


Figure 4.36. Illustrates statistical tests performed on data of amplitude of ACC obtained from clinical sub-groups.

In addition, the data on amplitude of ACC components for /si/ stimulus in the unaided condition for the group with normal hearing were tabulated. The following statistical analyses were carried out.

1. Descriptive analysis among sub-groups was performed for amplitude of ACC components.
2. Kolmogorov-Smirnov normality test was conducted to know the distribution of data within each sub-group and Levene's homogeneity test was carried out to know the distribution of data across sub-groups.
3. MANOVA was administered to know if there were significant differences between the sub-groups in the mean values of amplitude of ACC components.

The mean and standard deviation of the amplitude of ACC components at the onset and transition of /si/ are tabulated in Table 4.31. It was noted that the mean amplitudes of N1 and P2 reduced with age. Similar findings were obtained in amplitudes of 2N1 and 2P2.

Table 4.31

Mean (M) and standard deviation (SD) of amplitude of ACC components in sub-groups with normal hearing

Sub-groups (No. of participants)	*Amplitude (µV) of ACC components*			
	N1	*P2*	*2N1*	*2P2*
	M±SD	*M±SD*	*M±SD*	*M±SD*
'A' (10)	-1.33±0.90	1.70±0.99	-3.04±3.05	2.56±1.84
'B' (10)	-1.19±0.70	1.58±1.27	-2.98±1.42	2.26±2.21
'C' (10)	-0.95±0.73	1.33±1.05	-2.89±1.04	1.72±1.33
'D' (10)	-0.77±0.73	0.88±0.74	-2.00±1.74	1.57±1.11

Figure 4.37 illustrates statistical tests performed on data of amplitude of ACC obtained from normal hearing sub-groups. The Kolmogorov-Smirnov normality test and Levene's homogeneity test were carried out. The result of normality test revealed no significant difference ($p>0.05$) within each sub-group and Levene's test revealed homogeneity between sub-groups ($F < 2$) on the amplitude of each of the ACC components. Hence, MANOVA was performed to see if there was a significant difference across sub-groups in the amplitudes of ACC. It was found that there was no significant difference between sub-groups in the amplitude of each of the ACC components for /si/ stimulus.

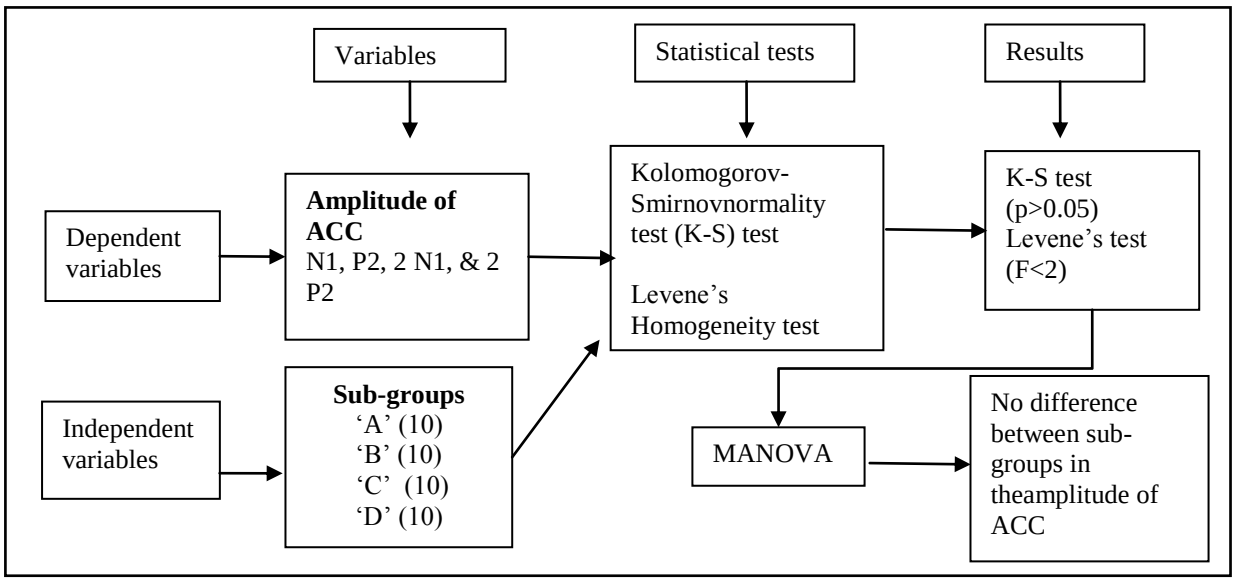


Figure 4.37. Illustrates statistical tests performed on data of amplitude of ACC obtained from normal hearing sub-groups.

Further, the amplitudes of different components of ACC were compared between different sub-groups of clinical group and group with normal hearing. The amplitudes of N1, P2 and 2N1 components of ACC in the clinical sub-groups of 'A', 'B' and 'C' were significantly different from sub-group 'D'. However, there was no significant difference between sub-groups in the amplitudes of N1, P2 and 2 N1 components of ACC in the group with normal hearing. Except for sub-group 'D', the amplitude data of N1, P2 and 2N1 components of ACC were combined separately in clinical group and group with normal hearing. Thus, the combined data on amplitude of each of the N1, P2 and 2 N1in clinical groups of 'A', 'B' and 'C' were compared with sub-groups of normal hearing. In the combined sub-group of 'ABC'; and sub-group 'D', the N1, P2 and 2 N1 amplitudes were higher in group with normal hearing than in clinical group. The mean and standard deviation of combined amplitudes of ACC are tabulated in Tables 4.32 and 4.33. It was noted that the amplitude of each of ACC components was higher in different sub-groups of normal hearing compared to clinical sub-groups.

Figure 4.38 illustrates statistical tests performed on data of amplitude of ACC obtained from clinical and normal hearing sub-groups. The normality test of Kolmogorov-Smirnov revealed that the data in the combined sub-group 'ABC' and 'D' were normally distributed, in terms of latencies of N1, P2 and 2N1 of ACC. The Levene's homogeneity test revealed that the group data on latencies of ACC components were homogenous ($F<2$) between clinical group and group with normal hearing in sub-groups of 'ABC' and 'D'. Thus, independent samples t-test was performed to see if there was any significant difference in the amplitudes of N1, P2 and 2N1 between normal hearing and clinical combined sub-groups 'ABC'; and sub-group 'D'. The mean, standard deviation, t-value and p-value of independent samples t-test of N1, P2 and 2N1 amplitudes of combined sub-group of 'ABC';

and sub-group 'D' of clinical group and group with normal hearing are tabulated in Tables 4.30 and 4.31. The results revealed that there was a significantly higher amplitude of 2 N1 [t (60) = 5.35, p = 0.000] alone in the group with normal hearing compared to that in the clinical group.

Table 4.32

Mean (M), standard deviation (SD), t-values and p-values of independent samples t-test on N1 and P2 amplitudes (in μV) in normal and clinical sub-groups

Sub-groups	*Groups (No. of participants)*	*N1 amplitude (μV)*			*P2 amplitude (μV)*		
		M ±SD	*t-value*	*p-value*	*M±SD*	*t-value*	*p-value*
'ABC'	*Normal hearing(30)*	-1.16±0.77	0.93	0.353	1.54±1.08	-1.17	0.245
	Clinical (32)	-0.99±0.62			1.28±0.60		
'D'	*Normal hearing (10)*	-0.77±0.73	0.66	0.512	0.88±0.74	-0.67	0.504
	Clinical (28)	-0.66±0.30			0.74±0.49		

Table 4.33

Mean (M), standard deviation (SD), t-values and p-values of independent samples t-test on 2N1 amplitude (in μV) between normal and clinical sub-groups

Sub-groups	*Groups (No. of participants)*	*2 N1 amplitude (μV)*		
		M ±SD	*t-value*	*p-value*
'ABC'	*Normal Hearing (30)*	-3.54±2.02	5.35	0.000***
	Clinical (32)	-1.56±0.51		
'D'	*Normal Hearing (10)*	-2.06±1.74	1.53	0.134
	Clinical (28)	-1.39±0.71		

Note: *** =p< 0.001

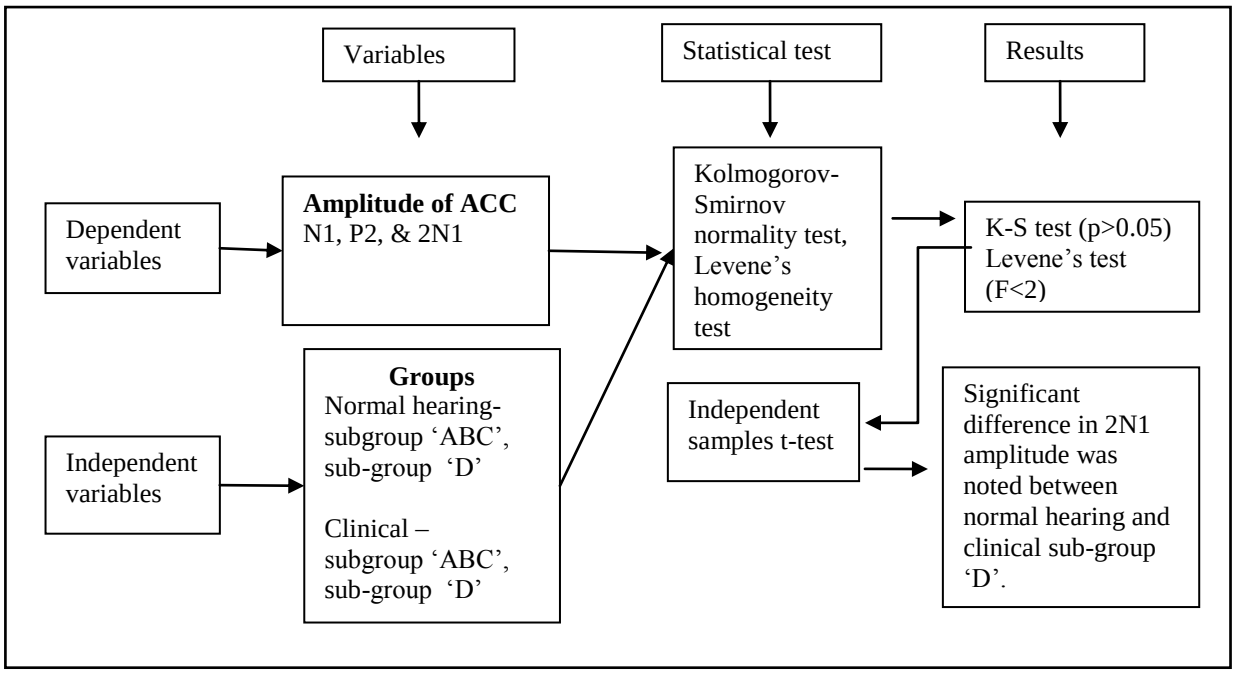


Figure 4.38. Illustrates statistical tests performed on data of amplitude of ACC obtained from clinical and normal hearing sub-groups.

Further, the data on amplitude of 2 P2 in the normal hearing group and clinical sub-groups were combined as there was no significant difference. Figure 4.39 illustrates statistical tests performed on data of 2 P2 amplitude of ACC obtained from clinical and normal hearing sub-groups. The normality test of Kolmogorov-Smirnov within the two groups on amplitude of 2 P2 revealed that the data were normally distributed ($p>0.05$). The Levene's homogeneity test revealed that there was no variance in amplitude of 2 P2 component of ACC ($F<2$) between clinical and normal hearing groups. Independent samples t-test was performed to see if there was any significant difference in the amplitude of 2 P2 between clinical and normal hearing groups. The mean, standard deviation, t-value, and p-value of independent samples t-test on 2 P2 amplitude in clinical group and group with normal hearing are given in Table 4.34. The result revealed that there was a significantly higher amplitude of 2 P2 was noted in the normal hearing group than in the clinical group [$t(98) = -3.21$, $p=0.002$].

Table 4.34

Mean (M), standard deviation (SD), t-value and p-value of independent samples t-test on 2P2 amplitude (in μV) in normal hearing and clinical groups

Groups (No. of participants)	*M± SD*	*t-value*	*p-value*
Normal Hearing (40)	2.02±1.66	-3.21	0.002**
Clinical (60)	1.24±0.74		

Note: ** = p<0.01

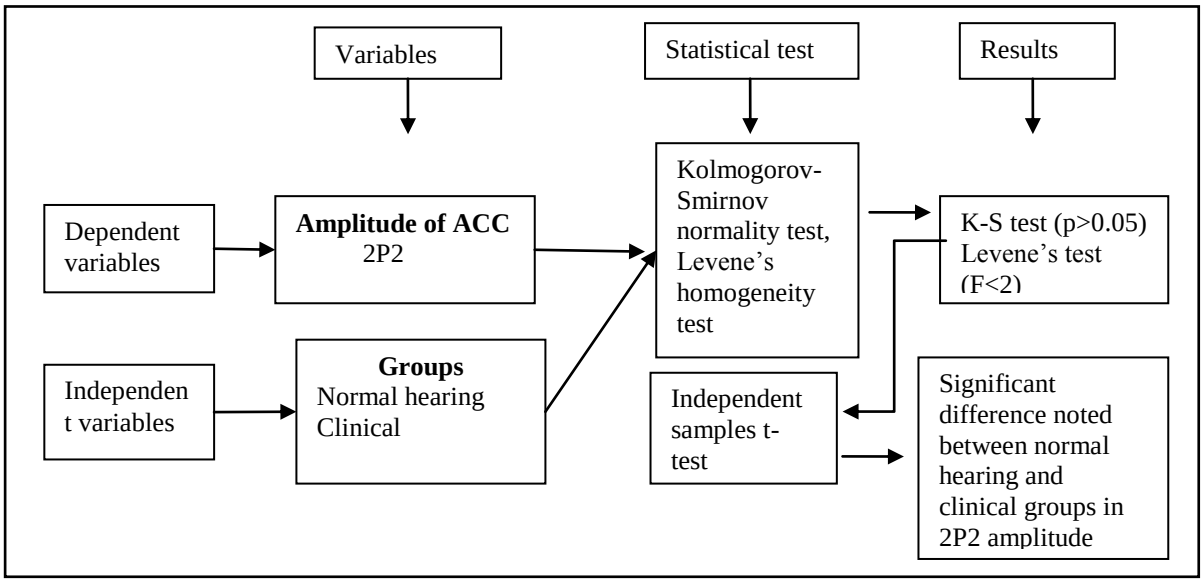


Figure 4.39. Illustrates statistical tests performed on data of 2P2 amplitude of ACC obtained from clinical and normal hearing sub-groups.

Comparison of brainstem responses and cortical responses in good and poor hearing aid performers

Each participant in clinical sub-groups was classified either as a good or a poor hearing aid performer based on their acceptable noise level (ANL) values. In the clinical group, if there was no statistically significant difference between sub-groups in data on each parameter obtained at different levels of auditory pathway, the data were combined, for good hearing aid performers (GHP). Similarly, the data on each parameter obtained at different levels of auditory pathway were combined if there was no significant difference between the

sub-groups, for poor hearing aid performers (PHP). Further, the response at each level of the auditory pathway, i.e., at brainstem and cortical levels, was compared between good and poor hearing aid performers.

1. Descriptive analysis was conducted on each parameter obtained at brainstem and cortical levels in the sub-groups of good and poor hearing aid performers.
2. Kruskal-Wallis test was performed to see if there were significant differences between the sub-groups in the mean values of the slope of V-A, F_0,F_0 energy and F_1 energy in FFR to each stimulus in GHP and PHP.
3. Mann-Whitney *U* test was performed to know if there was a significant difference between good and poor hearing aid performers on each parameter obtained at brainstem and cortical levels.

Comparison of brainstem responses in good and poor hearing aid performers.

Statistical analysis was done on the data collected from transient response and FFR at brain stem level. The data included the slope of V-A for /ɖa/ stimulus, and F_0, F_0 energy and F_1 energy for /ɖa/ and /si/ stimuli.

Comparison of slope of V-A in good hearing aid performers and poor hearing aid performers. Figure 4. 41 illustrates statistical tests performed on data of slope of V-A obtained from GHP and PHP groups. The mean and standard deviation of slope of V-A did not follow any pattern with respect to age. This was true in GHP and also in PHP. The mean and standard deviation of slope of V-A in each sub-group of GHP and PHP are shown in Tables 4.35 and 4.36 respectively. The data on slope of V-A from sub-groups of GHP was subjected to Kruskal-Wallis test. There was no significant difference in the slope of V-A

between sub-groups of GHP. Similar result was obtained for PHP. Hence, the data on slope of V-A from different sub-groups were combined separately in GHP and PHP. There was only one participant in the sub-group A of PHP, and hence was not considered for analysis.

Table 4.35

Mean and standard deviation of slope of V-A (in µV/ms*) in each sub-group of GHP*

Sub-groups (No. of participants)	*Slope of V-A Mean ±SD*
'A' (5)	-0.15±0.06
'B' (3)	-0.22±0.08
'C' (11)	-0.23±0.05
'D' (16)	-0.23±0.02

Table 4.36

Mean and standard deviation of slope of V-A (in µV/ms*) in each sub-group of PHP*

Sub-groups (No. of participants)	*Slope of V-A Mean ±SD*
'B' (4)	-0.14±0.07
'C' (8)	-0.11±0.07
'D' (12)	-0.22±0.04

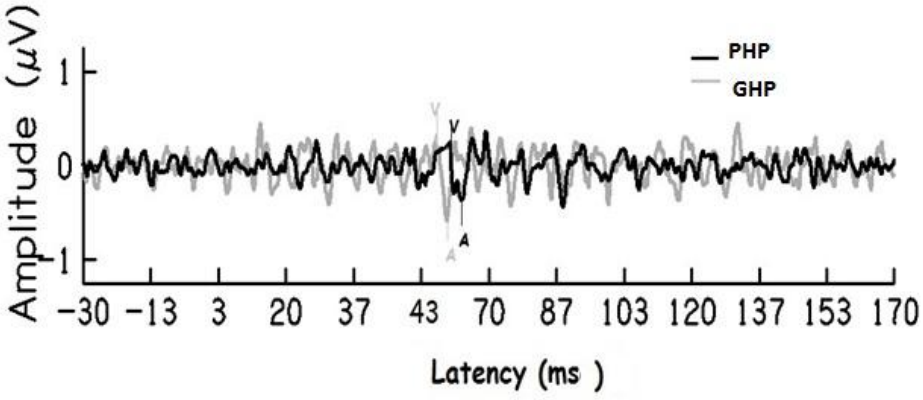


Figure 4.40. Grand average waveform of FFR obtained from GHP and PHP. The latencies of V and A are prolonged in PHP compared to GHP.

The mean, standard deviation, /U/ value and p-value of Mann-Whitney *U* test on slope of V-A, in good and poor hearing aid performers, are tabulated in Table 4.37.The slope of V-A was steeper in good hearing aid performers than in poor hearing aid performers

(Figure 4.40) . The results of Mann-Whitney *U* test revealed that this difference was not significant.

Table 4.37

Mean (M), standard deviation (SD), /U/ value and p-value of Mann-Whitney U test on slope of V-A in good (GHP) and poor (PHP) hearing aid performers

Slope of V-A (in µV/ms*)*			
Hearing aid performers (No. of participants)	*M ±SD*	*/U/ -value*	*p-value*
GHP (35)	-0.19±0.03	-0.69	0.490
PHP (25)	-0.21±0.04		

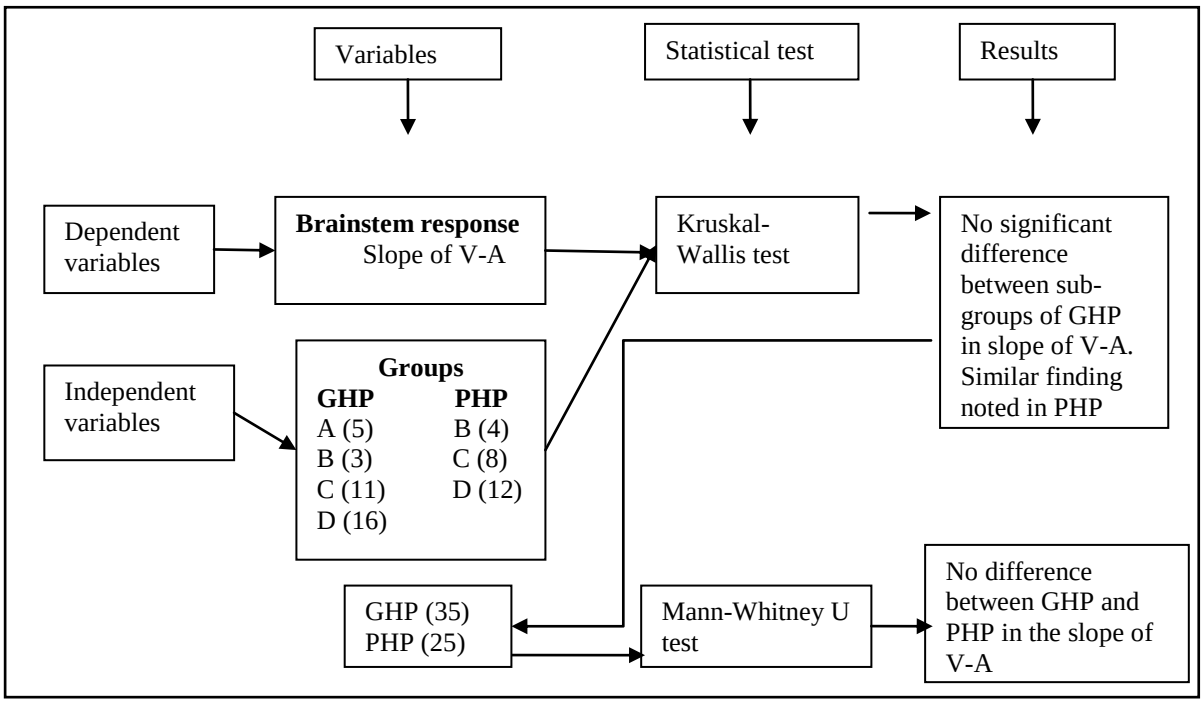


Figure 4.41. Illustrates statistical tests performed on data of slope of V-A obtained from GHP and PHP groups.

Comparison of FFR in terms of F0, F0 energy and F_1 energy in good hearing aid performers and poor hearing aid performers. Figures 4.43and 4.45 illustrates statistical tests performed on data of F_0, F_0 and F_1 energies of FFR to each stimulus obtained from GHP and PHP sub-groups. The mean and standard deviation of F_0, F_0energy and F_1 energy of FFR for each stimulus in good hearing aid performers are as shown in Table 4.38. It was found that mean encoding of F_0 of FFR did not change much between sub-groups for each stimulus. However, the F_0 energy and F_1 energy showed a decline with age. Kruskal-Wallis test was performed, to ascertain if the mean difference of the components of FFR (F_0, F_0 energy and F_1 energy) between sub-groups was significant. The result revealed that except for F_0 of FFR, the F_0 energy [χ^2 (3) =23.90, p = 0.000] and F_1 energy [χ^2(3) =26.21, p = 0.000] for /ɖa/ stimulus were significantly reduced as a function of age. Similarly, for /si/ stimulus, a significant reduction in the F_0 energy [χ^2(3) =24.99, p = 0.000] and F_1 energy [χ^2(3) =24.68, p = 0.000] were found between sub-groups, as a function of age.

Table 4.38

Mean and standard deviation (SD) of F_0, F_0 energy and F_1 energy of FFR for /ɖa/ and /si/ stimuli, in each sub-group of good hearing aid performers

Sub-groups (No.of participants)	*/ɖa/ stimulus*			*/si/ stimulus*		
	F_0	*F_0 energy*	*F_1 energy*	*F_0*	*F_0 energy*	*F_1 energy*
	Mean ±SD	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*
'A'(5)	136.37±2.03	19.25±1.65	14.17±1.65	140.60±2.79	18.22±1.63	15.89±1.65
'B'(3)	131.18±9.36	18.38±2.08	12.63±1.52	127.05±6.10	16.67±1.52	13.69±1.00
'C'(11)	133.80±7.01	16.59±1.70	10.083±1.63	136.02±3.22	15.01±2.32	13.15±1.92
'D' (16)	132.75±2.95	13.14±1.87	7.33±1.63	132.77±4.38	10.37±2.21	9.05±2.05

Mann-Whitney *U* test was performed to see which sub-group of GHP contributed for significant difference in the mean F_0 energy and F_1 energy of FFR for each stimulus. The /*U*/ value and p-value of Mann-Whitney *U* test on F_0 energy and F_1 energy of FFR for /ɖa/ and /si/ stimuli are tabulated in Table 4.39.

Table 4.39

|U|-value and p-value of Mann-Whitney U *test on F_0 energy and F_1 energy of FFR for /ḍa/ and /si/ stimuli, in good hearing aid performers*

Sub-groups	*F_0 energy*				*F_1 energy*			
	/ḍa/ stimulus		*/si/ stimulus*		*/ḍa/ stimulus*		*/si/ stimulus*	
	U	*p -value*	*U*	*p -value*	*U*	*p -value*	*U*	*p-value*
'A' vs.'B'	5.50	0.54	4.00	0.29	4.00	0.29	2.50	0.13
'A' vs.'C'	8.00	0.02*	9.00	0.03*	7.00	0.02*	7.00	0.02*
'A' vs.'D'	0.00	0.00***	0.00	0.00***	0.00	0.00***	0.00	0.00***
'B' vs.'C'	1.00	0.01*	0.50	0.01*	2.00	0.01**	4.00	0.04*
'B' vs.'D'	0.00	0.00***	0.00	0.00***	0.00	0.00***	0.00	0.00***
'C' vs.'D'	12.00	0.00***	2.00	0.00***	8.00	0.00***	8.00	0.00***

Note: * = p<0.05; ***= p<0.001

The results of Mann-Whitney *U* test revealed that there was no significant difference between sub-group 'A' and 'B' in F_0 energy of FFR for each stimulus. However, sub-groups of 'A' and 'B' were significantly different from sub-group 'C' and sub-group 'D' in F_0 energy of FFR for each stimulus. Further, sub-group 'C' was different from sub-group 'D' in F_0 energy of FFR for each stimulus. Similar results were found for F_1 energy obtained from FFR for each stimulus between sub-groups.

Further, the mean and standard deviation of F_0, F_0 energy and F_1 energy of FFR for each stimulus in poor hearing aid performers are as shown in Table 4.40. In sub-group A, there was only one participant. The F_0, F_0 energy and F_1 energy for this participant was 135 Hz, 17.05 and 12.97 for /ḍa/ stimulus and 136 Hz, 16.01 and 14.69 for /si/ stimulus. As there was only one participant in sub-group 'A', it was excluded from further analysis. It was found that the mean value of F_0 of FFR did not change much in other sub-groups. However, there was a decline in the mean values of F_0 energy and F_1 energy with increase in age.

Kruskal-Wallis test was performed to know if the mean values of components of FFR (F_0, F_0 energy and F_1 energy) between sub-groups were significantly different. The result revealed that except F_0 of FFR for each stimulus, a significant reduction of F_0 energy [χ^2 (2) =13.86, p = 0.001] and F_1 energy [χ^2 (2) =17.72, p = 0.000] of FFR were noted between sub-groups for /ɖa/ stimulus; and F_0 energy [χ^2 (2) =13.26, p = 0.001] and F_1 energy [χ^2 (3) =11.02, p = 0.004] for /si/ stimulus.

Table 4.40

Mean and standard deviation (SD) of F_0, F_0 energy and F_1 energy of FFR for /ɖa/ and /si/ stimuli, in each sub-group of poor hearing aid performers

Sub-groups	*/ɖa/ stimulus*			*/si/ stimulus*		
	F_0	*F_0 energy*	*F_1 energy*	*F_0*	*F_0 energy*	*F_1 energy*
	Mean ±SD	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*
'B'(4)	132.92±6.51	18.26±0.95	13.68±0.94	133.75±2.62	17.22±1.70	14.90±1.70
'C'(8)	128.96±4.71	14.69±1.96	10.61±1.57	127.48±2.81	13.14±2.02	11.61±2.22
'D'(12)	127.39±2.95	11.88±2.26	7.06±1.16	132.10±3.50	10.20±2.25	9.86±1.33

Mann-Whitney *U* test was performed to see the sub-group of PHP that could have contributed for a significant difference in the mean F_0 energy and F_1 energy of FFR for each stimulus. The /*U*/ value and p-value of Mann-Whitney *U* test on F_0 energy and F_1 energy of FFR for /ɖa/ and /si/ stimulus are tabulated in Table 4.41.

Table 4.41

|U|-value and p-value of Mann-Whitney U test on F_0 energy and F_1 energy of FFR for both /ḍa/ and /si/ stimuli in poor hearing aid performers

Sub-groups	*F_0 energy*				*F_1 energy*			
	/ḍa/ stimulus		*/si/ stimulus*		*/ḍa/ stimulus*		*/si/ stimulus*	
	U	*p -value*	*U*	*p –value*	*U*	*p-value*	*U*	*P –value*
'B' vs. 'C'	1.00	0.01*	1.50	0.01*	2.00	0.01*	4.00	0.04*
'B' vs. 'D'	0.00	0.00***	0.00	0.00***	0.00	0.00***	0.00	0.00***
'C' vs. 'D'	15.00	0.01*	3.00	0.00***	16.00	0.01*	22.50	0.04*

Note: * = p<0.05; ***= p<0.001

The results of Mann-Whitney *U* test revealed that sub-groups of PHP were significantly different from each other in F_0 energy of FFR for both stimuli. Similar results were found in F_1 energy of FFR between sub-groups of PHP. Hence the sub-group data on F_0energy and F_1 energy were not combined.

It was noted earlier that there was no significant effect of age in F_0 of FFR for both the stimuli, in GHP and PHP. Thus, the data on FFR for each stimulus from sub-groups were combined in GHP. Similarly, the data on F_0 of FFR for each stimulus from sub-groups of PHP were combined.

Table 4.42

Mean, standard deviation, U, and p-value of Mann-Whitney U test for F_0 of FFR, for /ɖa/ and /si/ stimuli, between good and poor hearing aid performers

Stimuli	*Hearing aid performers (No. of participants)*	*Mean ±SD*	*U*	*p-value*
/ɖa/ stimulus	*GHP (35)*	133.46±5.09	188.50	0.00***
	PHP(24)	128.84±4.52		
/si/ stimulus	*GHP (35)*	134.42±5.21	213.00	0.00***
	PHP(24)	130.84±3.92		

Note: *** = p<0.001

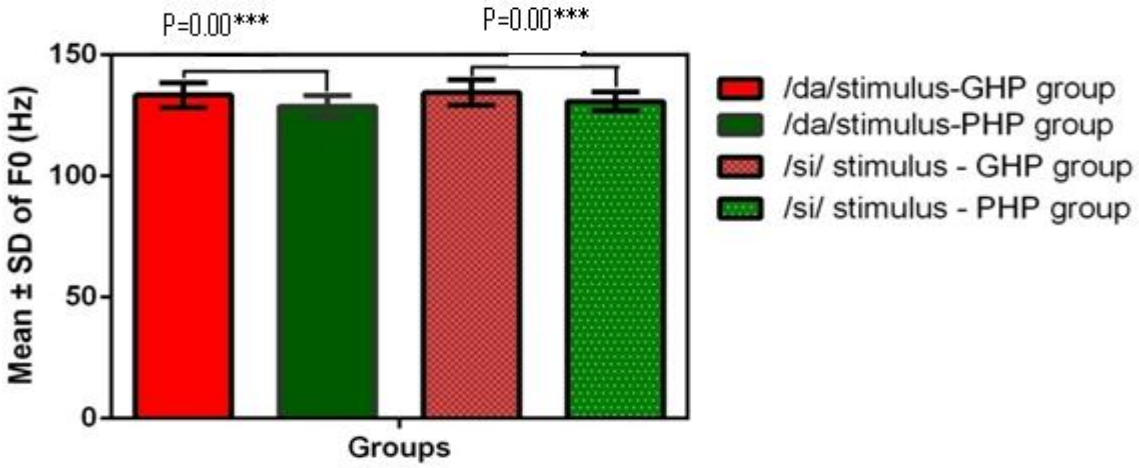


Figure 4.42. Mean, standard deviation and p-value of *Mann-Whitney U test on* F0 of FFR in GHP and PHP groups.

From the mean values it can be inferred that the F_0 of FFR was well represented in GHP than in PHP for each stimulus (Figure 4. 42). Further, the F_0 of FFR was compared between GHP and PHP using Mann-Whitney *U* test. The result showed there was a significant reduction of F_0 encoding in PHP compared to GHP (Table 4. 42) for /ɖa/ stimulus (/*U*/ = 188.50, p= 0.000) and /si/ stimulus (/*U*/ = 213.00, p= 0.001).

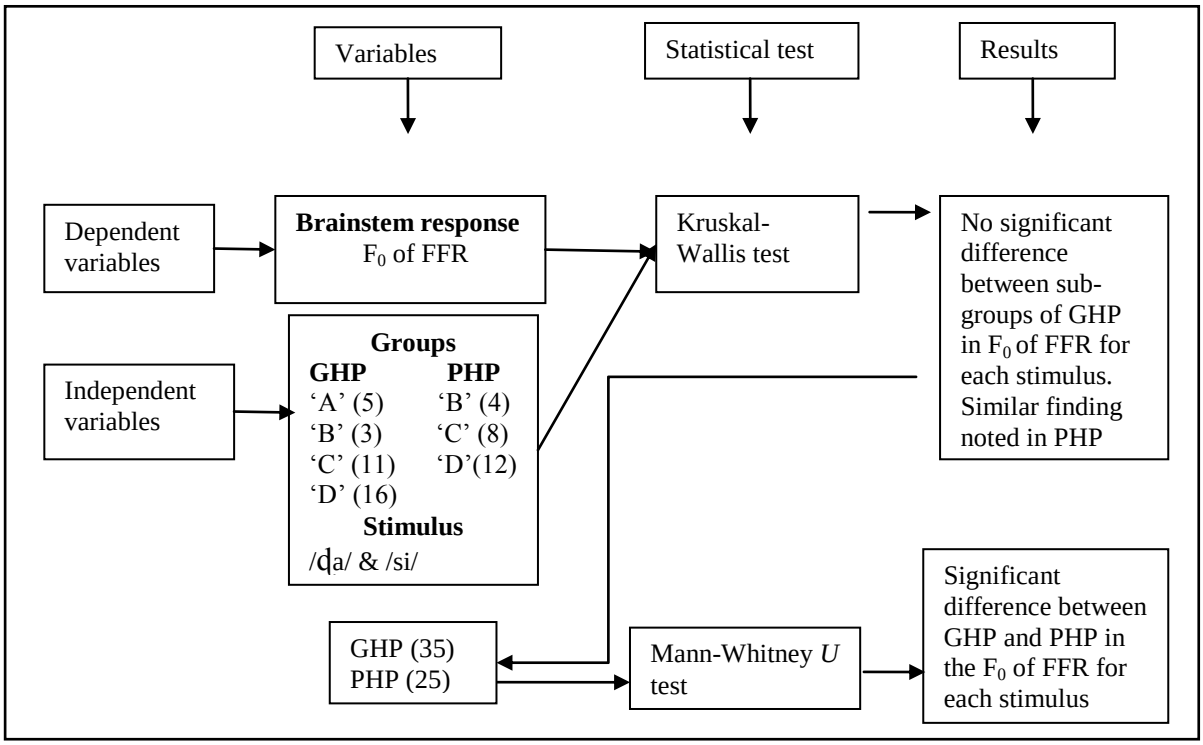


Figure 4.43. Illustrates statistical tests performed on data of F_0 of FFR obtained from GHP and PHP sub-groups.

In addition, the F_0 energy and F_1 energy of FFR to each stimulus from sub-groups of 'AB' of GHP was compared with sub-group 'B' of PHP. As noted earlier that in PHP, there was only one participant in sub-group 'A'. Thus, it was excluded from analysis.

Table 4.43

Mean and standard deviation of F_0 energy and F_1 energy of FFR for /ɖa/ and /si/ stimuli between good and poor hearing aid performers

Sub-groups	*Hearing aid performers (No. of participants)*	*/ɖa/ stimulus*		*/si/ stimulus*	
		F_0 energy	*F_1 energy*	*F_0 energy*	*F_1 energy*
		Mean ±SD	*Mean ±SD*	*Mean ±SD*	*Mean ±SD*
'AB'	*GHP(8)*	18.92±1.73	13.59±1.69	17.64±3.50	15.06±1.77
'B'	*PHP (4)*	18.26±0.95	13.68±0.94	17.22±1.70	14.90±1.70
'C'	*GHP(11)*	16.59±1.70	11.52±1.70	15.01±2.32	13.15±1.92
	PHP (8)	14.69±1.96	10.61±1.57	13.14±2.02	11.61±2.22
'D'	*GHP(16)*	13.16±1.87	7.33±1.63	10.37±2.23	9.05±2.05
	PHP (12)	11.88±2.26	7.06±1.16	10.20±2.25	9.86±1.33

From Table 4.43, it was noted that the mean and standard deviation of F_0 energy and F_1 energy of FFR to each stimulus were reduced in PHP than in GHP. Further, to know if there was any significant difference between GHP and PHP in the mean F_0 energy and F_1 energy of FFR for each stimulus, Mann-Whitney *U* test was performed. The result did not reveal any significant difference between GHP and PHP in F_0 energy and F_1 energy to each stimulus (Figure 4.44). Similar results were found in sub-group 'C' and sub-group 'D', in the F_0 energy and F_1 energy of FFR for each stimulus, between GHP and PHP

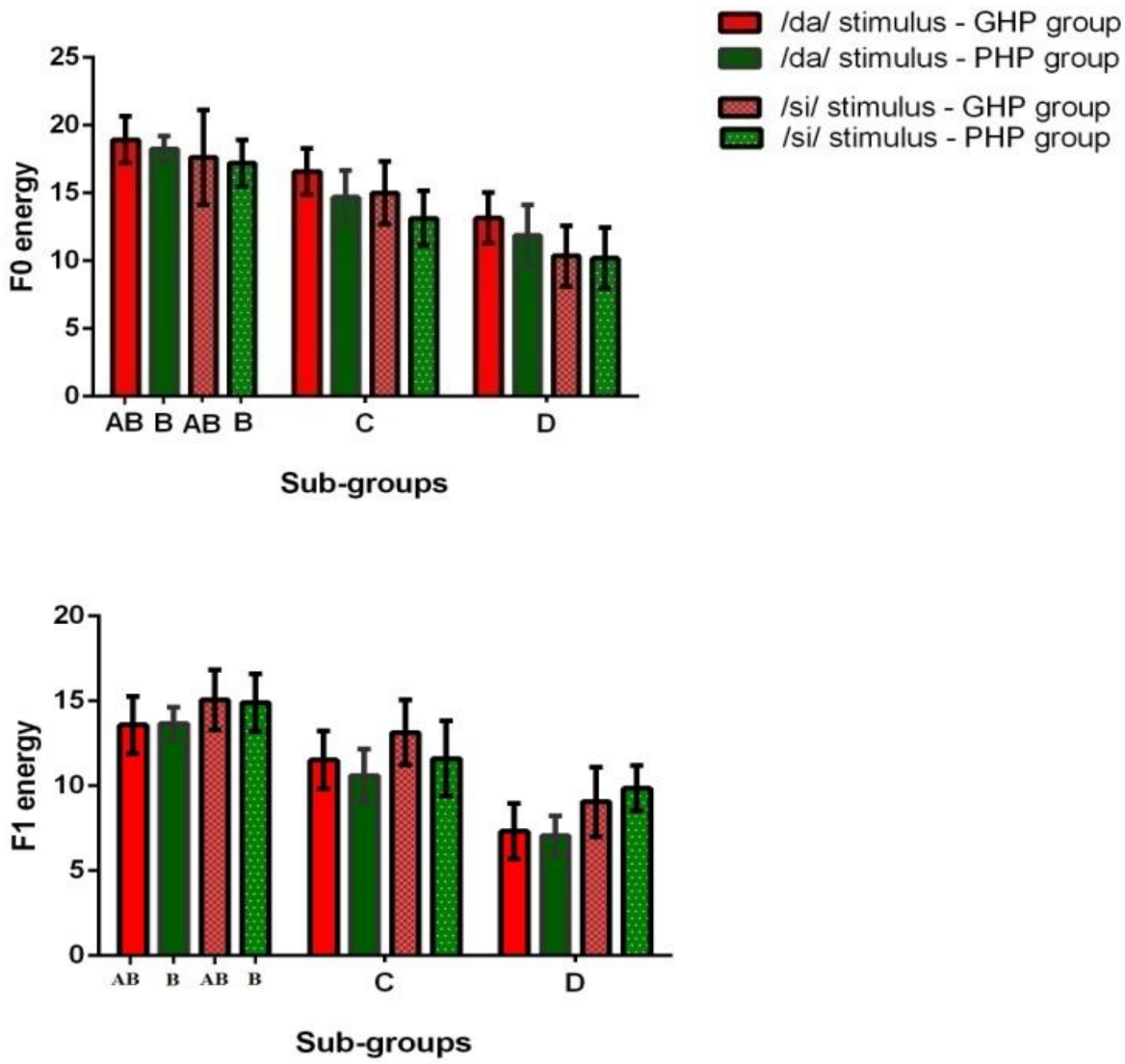


Figure 4.44. Mean and standard deviation of F_0 energy and F_1 energy in GHP and PHP sub-groups.

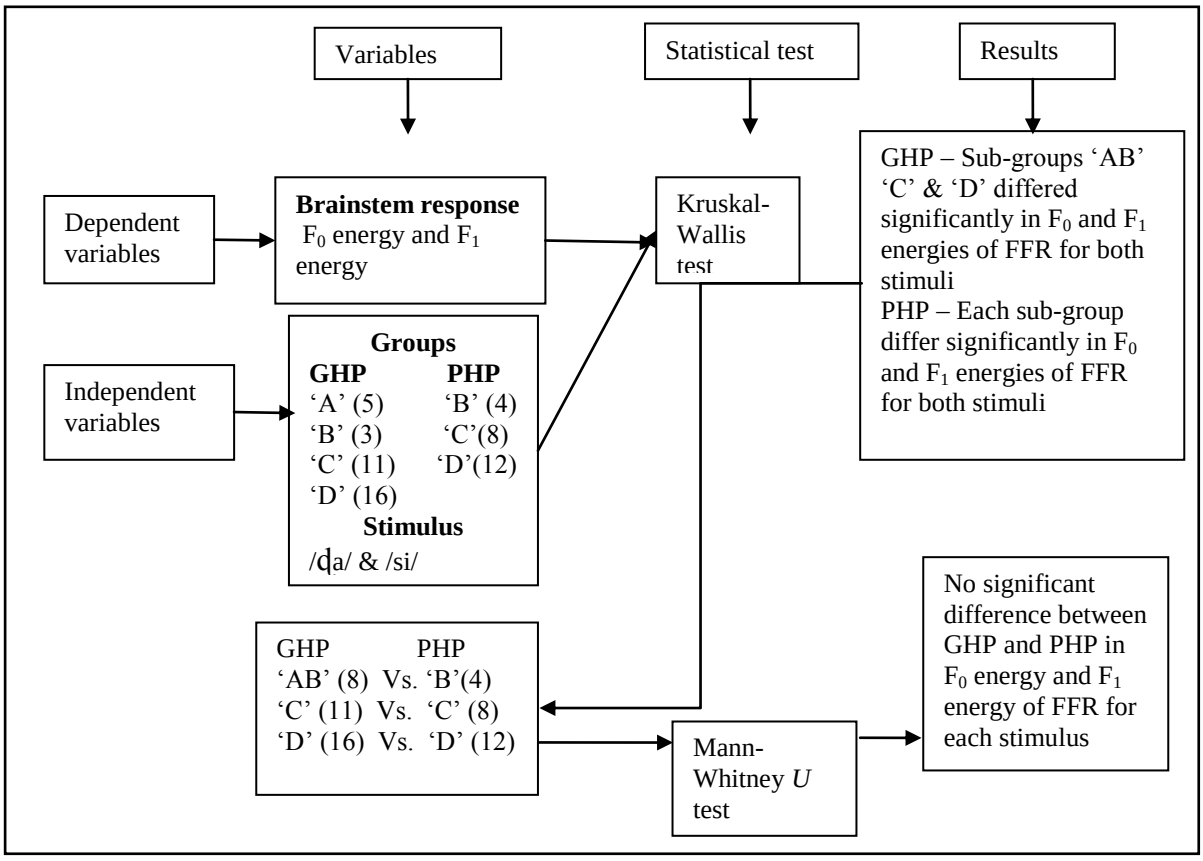


Figure 4.45. Illustrates statistical tests performed on data of F_0 energy and F_1 energy of FFR obtained from GHP and PHP sub-groups.

Comparison of the cortical responses in good and poor hearing aid performers.

Statistical analysis was performed on the ACC response data obtained from cortical level in terms of slope of N1-P2of LLR for /ɖa/ stimulus; and latency and amplitude of ACC for /si/ stimulus obtained from sub-groups of GHP and PHP.

***Comparison of slope of N1-P2 of LLR in good hearing aid performers and poor hearing aid performers*.** Figure 4.47 illustrates statistical tests performed on data of slope of N1-P2 obtained from GHP and PHP groups. The mean and standard deviation of slope of N1-P2 in GHP are as tabulated in Table 4.44. The mean slope of N1-P2 was shallower with respect to age in GHP. The mean and standard deviation of slope of N1-P2 in PHP are as tabulated in Table 4.45. In PHP, the sub-group 'A' was excluded from analysis, as there was

only one participant. The slope of N1-P2 of this participant in PHP group was 0.051. The mean slope of N1-P2 in PHP was also shallower with respect to age. The data on slope of N1-P2 from sub-groups of GHP and PHP were subjected to Kruskal-Wallis test. It was found that there was no significant difference in the slope of N1-P2 between sub-groups of GHP. Similar result was noted in the slope of N1-P2 in PHP. Thus, the data on slope of N1-P2 from sub-groups of GHP were combined. Similarly, the data on slope for sub-groups of PHP were also combined. In the combined data, it was noted that the slope of N1-P2 was steeper in GHP than in PHP (Figure 4.46). The result of Mann-Whitney *U* test revealed that this difference was not significant.

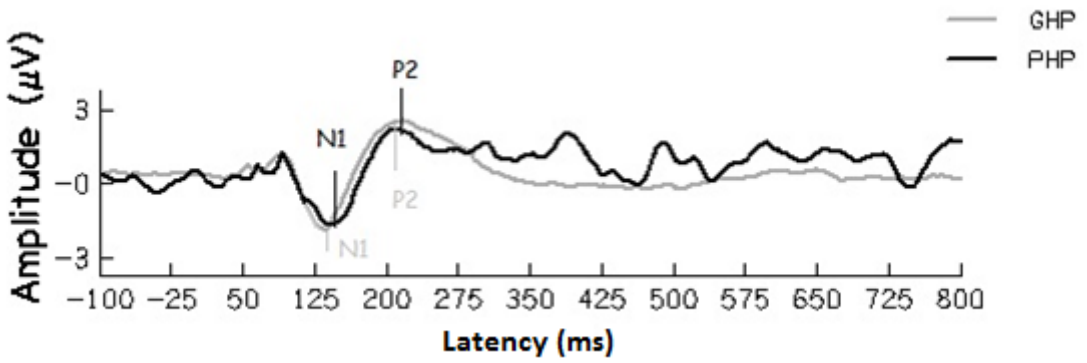


Figure 4.46. Grand average waveform of LLR obtained from GHP and PHP is represented. The latencies of N1 and P2 were slightly earlier in GHP than PHP.

Table 4.44

Mean and standard deviation (SD) of slope of N1-P2 (in μV/ms) *in sub-groups of GHP*

Sub-groups (No. of participants)	*Mean ±SD*
'A' (5)	0.086±0.02
'B' (3)	0.064±0.04
'C' (11)	0.041±0.01
'D'(16)	0.038±0.01

Table 4.45

Mean and standard deviation (SD) on slope of N1-P2 (in µV/ms) *in sub-groups of PHP*

Sub-groups (No. of participants)	*Mean ±SD*
'B' (4)	0.059±0.01
'C' (8)	0.042±0.03
'D'(12)	0.040±0.04

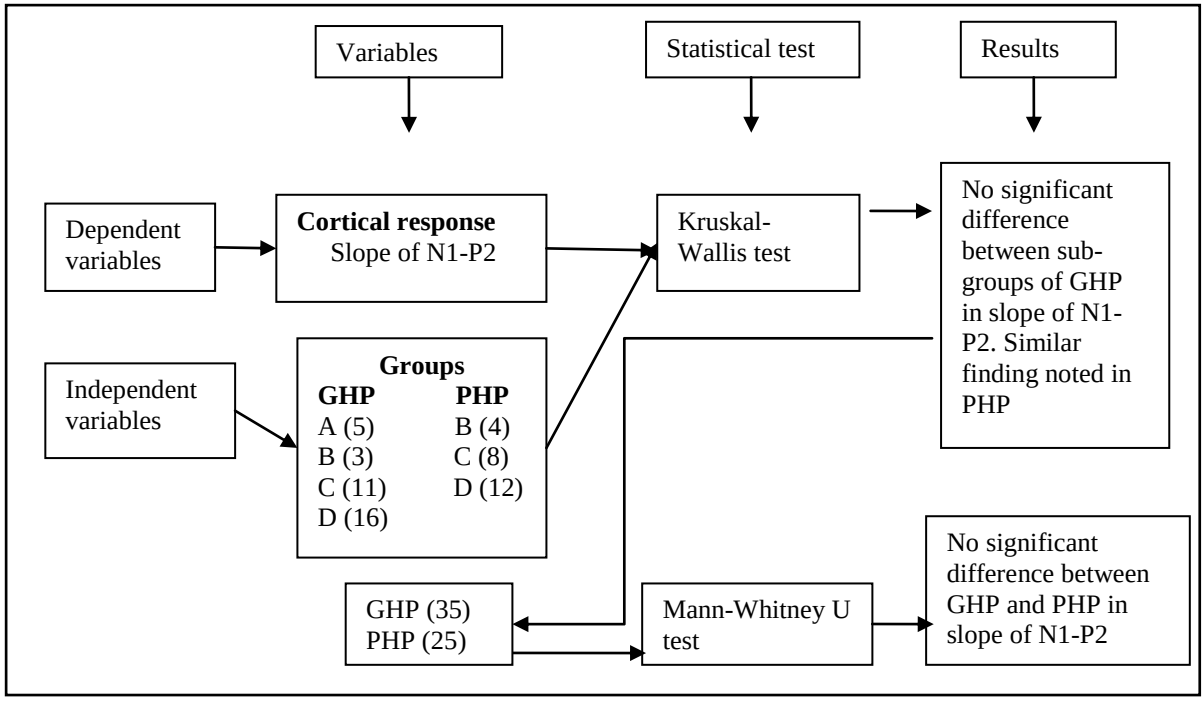


Figure 4.47. Illustrates statistical tests performed on data of slope of N1-P2 obtained from GHP and PHP groups.

Comparison of latency of ACC components in good hearing aid performers and poor hearing aid performers. Figure 4.49 illustrates statistical tests performed on data of latency of ACC obtained from GHP and PHP sub-groups. The mean and standard deviation of latency of ACC components for /si/ stimulus in good hearing aid performers are as shown in Table 4.46. It was found that mean latency of each component of ACC was prolonged with respect to age in GHP. In PHP, the sub-group A was excluded from analysis as there was

only one participant. The latency of ACC components of this participant of sub-group 'A' of PHP was 138.2 ms for N1, 245.32 ms for P2, 286.41 ms for 2 N1,and 390.55 ms for 2P2. The mean and standard deviation of latency of ACC for /si/ stimulus in poor hearing aid performers are as shown in Table 4.47. The mean latency of ACC was prolonged with respect to age in PHP.

Kruskal-Wallis test was performed to ascertain if the mean latency of ACC components between sub-groups of GHP were significant. Similarly, it was done in sub-groups of PHP. The result revealed that except in the latency of 2P2, there was a significant prolongation in the latencies of N1 [χ^2 (3) =11.30, p = 0.010], P2 [χ^2 (3) =18.96, p = 0.000] and 2N1 [χ^2 (3) =11.78, p = 0.008] components of ACC as a function of age in the sub-groups of GHP. In PHP, except for 2P2, a significant prolongation in the latencies of N1 [χ^2 (2) =7.52, p = 0.023], P2 [χ^2 (2) =3.22, p = 0.020] and 2N1 [χ^2 (2) =14.17, p = 0.003] components of ACC were noted as a function of age.

Table 4.46

Mean and standard deviation (SD) of latency of ACC in each sub-group of good hearing aid performers

Sub-groups in GHP (No. of participants)	*Latency (ms) of ACC components*			
	N1	*P2*	*2 N1*	*2 P2*
	Mean±SD	*Mean±SD*	*Mean±SD*	*Mean±SD*
'A' (5)	135.80±2.16	191.20±4.14	279.00±5.52	360.60±1.14
'B' (3)	137.00±4.14	223.00±5.14	282.33±5.77	383.33±2.88
'C' (11)	147.81±14.31	235.63±11.54	290.90±7.36	380.72±9.32
'D'(16)	154.18±18.47	247.10±5.78	292.80±6.51	393.25±14.19

Table 4.47

Mean and standard deviation (SD) of latency of ACC in each sub-group of poor hearing aid performers

Sub-groups in PHP (No. of participants)	*Latency (ms) of ACC components*			
	N1	*P2*	*2 N1*	*2 P2*
	Mean±SD	*Mean±SD*	*Mean±SD*	*Mean±SD*
'B' (4)	141.87±9.77	235.25±9.87	287.50±9.14	384.50±2.38
'C' (8)	143.75±3.09	244.02±5.16	294.50±5.04	385.87±5.69
'D' (12)	151.25±1041	254.41±6.37	309.25±10.01	395.33±14.81

Mann-Whitney *U* test was performed to see the sub-group of GHP that contributed for a significant difference in the mean latencies of N1, P2 and 2N1 components of ACC. The /*U*/ value and p-value of Mann-Whitney *U* test on latency of ACC are tabulated in Table 4.48. The results of Mann-Whitney *U* test revealed that there was no significant difference between sub-group 'A' 'B' and 'C' in latencies of N1, P2 and 2N1 components of ACC. However, sub-groups of 'A', 'B' and 'C' were significantly different from sub-group 'D' in latencies of N1, P2 and 2N1 components of ACC.

Table 4.48

|U|-value and p-value of Mann-Whitney U test on latencies of N1, P2 and 2N1 components of ACC in good hearing aid performers

Sub-groups in GHP	*Latency (ms) of ACC components*					
	N1		*P2*		*2 N1*	
	U	*p -value*	*U*	*p -value*	*U*	*p -value*
'A' vs. 'B'	4.50	0.24	9.00	0.09	6.00	0.65
'A' vs. 'C'	10.00	0.40	67.50	0.31	1.19	0.23
'A' vs. 'D'	10.00	0.01*	0.00	0.00***	8.50	0.00***
'B' vs. 'C'	6.00	0.09	21.00	0.73	16.00	0.93
'B' vs. 'D'	6.00	0.04*	0.00	0.00***	7.00	0.05*
'C' vs. 'D'	1.98	0.04*	0.00	0.02*	2.52	0.01**

Note: * = p<0.05; ***= p<0.001

Further, Mann-Whitney *U* test was performed to see which sub-group of PHP contributed for a significant difference in the mean latencies of N1, P2 and 2N1 components of ACC. The /*U*/ value and p-value of Mann-Whitney *U* test on latency of ACC are tabulated in Table 4.49.The results of Mann-Whitney *U* test revealed that there was no significant difference between sub-groups 'B' and 'C' in latencies of N1, P2 and 2N1 components of ACC. However, sub-groups of 'B' and 'C' were significantly different from sub-group 'D' in latencies of N1, P2 and 2N1 components of ACC.

Table 4.49

|U|-value and p-value of Mann-Whitney U test on latencies of N1, P2 and 2N1 components of ACC in poor hearing aid performers

Sub-groups	*N1*		*P2*		*2N1*	
in PHP	*U*	*P value*	*U*	*P value*	*U*	*P value*
'B' vs. 'C'	8.50	0.20	7.00	0.12	7.50	0.14
'B' vs. 'D'	5.00	0.02*	4.00	0.00***	1.98	0.04*
'C' vs. 'D'	20.00	0.03*	7.00	0.00***	18.00	0.05*

Note: * = p<0.05; ***= p<0.001

The latencies of N1, P2 and 2N1 components of ACC in the sub-groups of 'A', 'B' and 'C' were not significantly different in GHP, and hence were combined. Similarly, in PHP, the latencies of ACC components in sub-groups of 'B' and 'C' were combined.

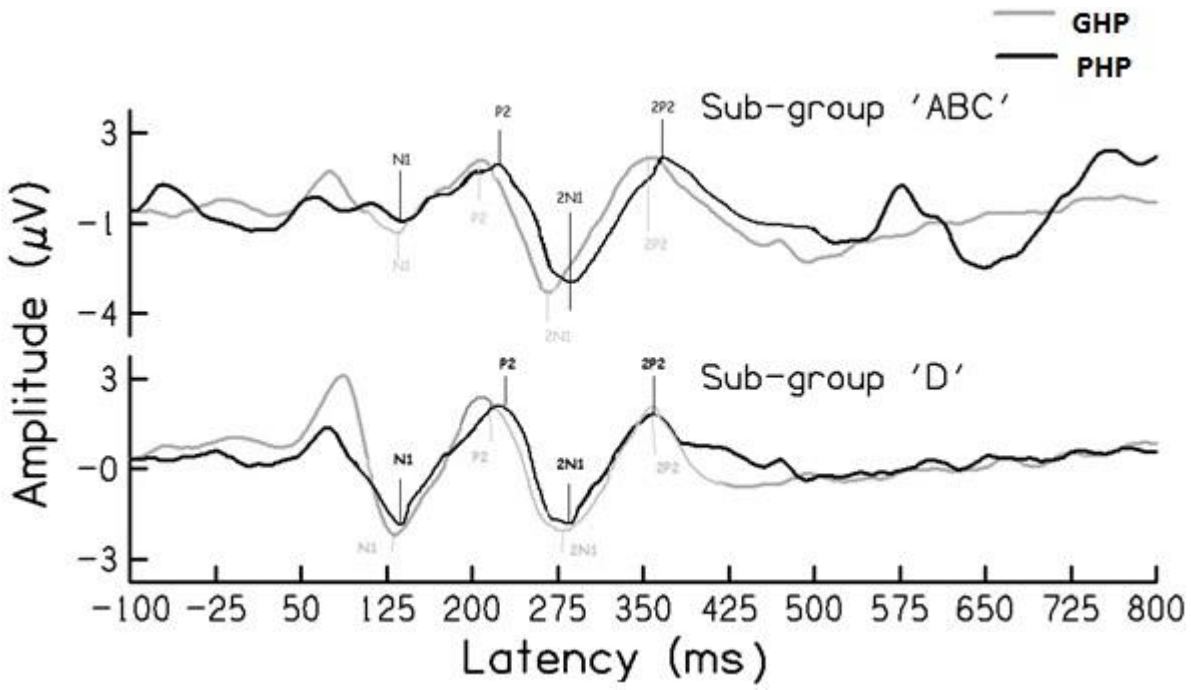


Figure 4.48. Grand average waveform of ACC obtained from GHP and PHP. The latency of ACC is earlier in GHP than PHP. This is true for both sub-groups.

The mean and standard deviation of latency of ACC of combined sub-groups of 'ABC' of GHP; and combined sub-group of 'BC' of PHP; and sub-group 'D' of both GHP and PHP are tabulated in Table 4.50. The latency of N1, P2, and 2N1 components of ACC was prolonged in PHP than in GHP, in both combined sub-group 'ABC' and sub-group 'D' (Figure 4.48). Further, the latency of ACC was compared between GHP and PHP in combined sub-group of 'ABC' and sub-group 'D' using Mann-Whitney *U* test. The result showed that, a significantly earlier latencies of P2 (/*U*/ = 42.50, p= 0.004) and 2N1 (/*U*/ = 31.00, p= 0.001) component of ACC were noted in the GHP (in the combined sub-group of ABC) compared to PHP (in the combined sub-group of BC). In the sub-group 'D', the latency of P2 (/*U*/ = 41.00, p= 0.010) was found significantly earlier in GHP than in PHP.

Table 4.50

Mean and standard deviation (SD) of latency of ACC in good and poor hearing aid performers

Sub-groups	*Hearing aid performers (No. of participants)*	*Latency (ms)of ACC components*		
		N1	*P2*	*2 N1*
		M±SD	*M±SD*	*M±SD*
'ABC'	*GHP(19)*	142.64±12.22	221.94±21.33	281.78±6.60
'BC'	*PHP(12)*	144.50±8.01	241.10±7.87	292.16±7.31
'D'	*GHP(16)*	154.18±18.47	237.18±5.78	290.87±6.51
'D'	*PHP(12)*	157.25±10.41	244.41±6.37	294.25±10.01

As mentioned earlier, the latency data of 2P2 component of ACC were combined in sub-groups of GHP, as there was no significant difference. Similarly, the data on latency of 2P2 component in PHP were combined, as there was no significant difference between sub-groups. The combined data of 2P2 latency of ACC were compared between good and poor hearing aid performers. The mean latency of 2P2 was earlier in GHP (375.60±13.32) than in PHP (390.37±11.89), and this difference was found to be significant on Mann-Whitney *U* test (U=122.50, p =0.000).

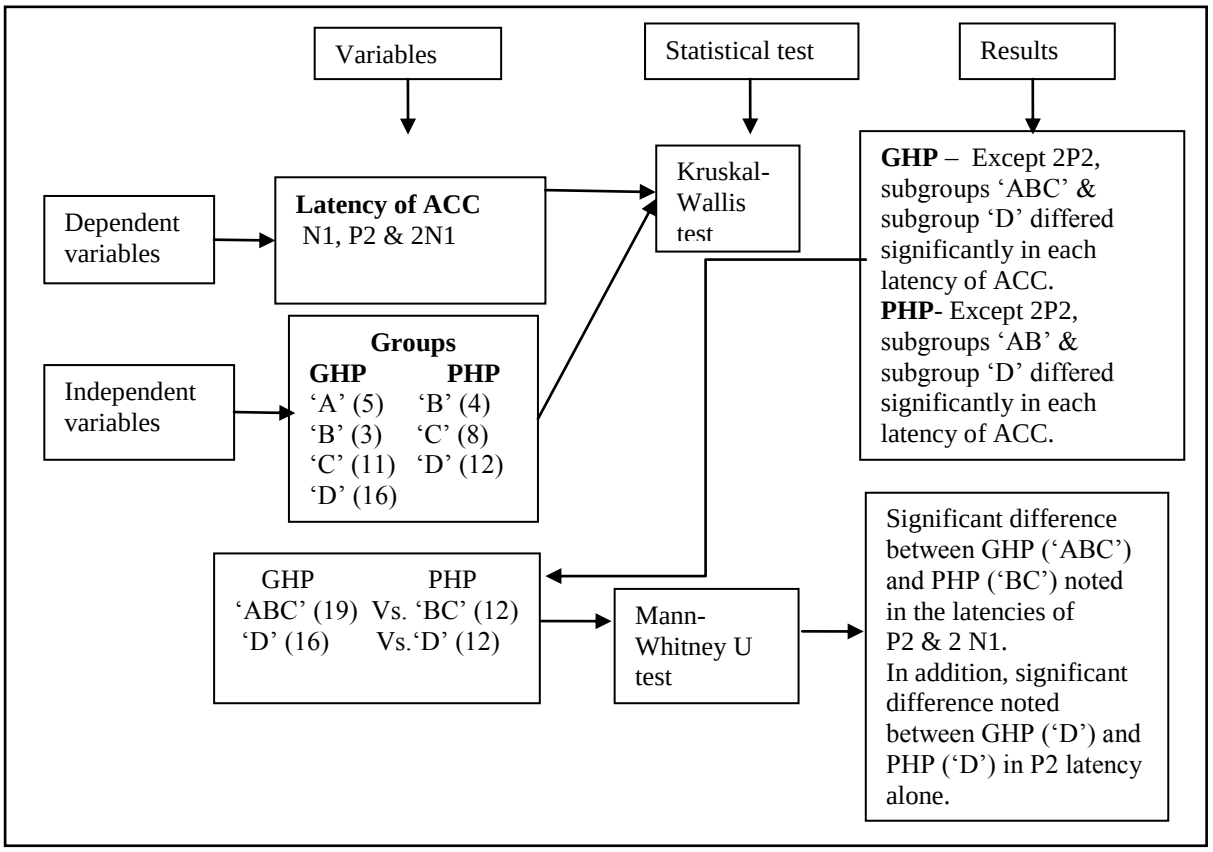


Figure 4.49. Illustrates statistical tests performed on data of latency of ACC obtained from GHP and PHP sub-groups.

Comparison of amplitude of ACC components in good hearing aid performers and poor hearing aid performers. Figure 4.50 illustrates statistical tests performed on data of amplitude of ACC obtained from GHP and PHP sub-groups. The mean and standard deviation of amplitude of ACC for /si/ stimulus in GHP and PHP were as shown in Tables 4.51 and 4.52. It was found that the mean amplitude of each of the components of ACC was reduced with respect to age in GHP and PHP. The sub-group 'A' of PHP had only one participant, whose amplitudes were N1 is -1.01µV, P2 is 1.04 µV, 2N1 is -1.06 µV and 2P2 is 1.01 µV. The data from this sub-group was not included for analysis. On the other sub-groups, Kruskal-Wallis test was performed to ascertain if there was any significant difference in the mean amplitude of ACC between sub-groups of GHP. Similarly, it was done in sub-groups of PHP. The result revealed that except for amplitude of 2P2, a significantly reduced amplitudes of N1 [$\chi^2(3) = 9.96$, $p = 0.019$], P2 [$\chi^2(3) = 11.92$, $p = 0.008$] and 2N1 [$\chi^2(3)$

=8.03, p = 0.045] components of ACC were found between sub-groups of GHP. In PHP too, except for 2P2, the N1 [χ^2 (2) =13.07, p = 0.001], P2 [χ^2 (2) =8.09, p = 0.017] and 2N1 [χ^2(2) =7.61, p = 0.022] components of ACC were significantly reduced between sub-groups.

Table 4.51

Mean and standard deviation (SD) of amplitude of ACC in sub-groups of good hearing aid performers

Sub-groups in GHP	*Amplitude (µV) of ACC components*			
	N1	*P2*	*2 N1*	*2 P2*
	Mean±SD	*Mean±SD*	*Mean±SD*	*Mean±SD*
'A' (5)	-1.29±0.17	1.76±0.9	-2.08±0.17	1.83±0.72
'B' (3)	-0.70±0.57	1.61±0.8	-2.23±0.57	1.56±0.54
'C' (11)	-0.49±0.67	1.20±0.55	-1.39±0.45	1.07±0.09
'D' (16)	-0.21±0.13	0.60±0.47	-0.64±0.48	1.34±0.68

Table 4.52

Mean and standard deviation (SD) of amplitude of ACC in each sub-group of poor hearing aid performers

Sub-groups in PHP	*Amplitude (µV) of ACC components*			
	N1	*P2*	*2 N1*	*2 P2*
	Mean±SD	*Mean±SD*	*Mean±SD*	*Mean±SD*
'B' (4)	-1.52±0.32	1.44±0.27	-1.39±0.31	1.37±0.39
'C' (8)	-1.33±0.35	0.92±0.85	-1.32±0.43	1.17±0.18
'D'(12)	-0.50±0.16	0.54±0.46	-0.53±0.35	0.62±0.77

Mann-Whitney *U* test was performed to see the sub-group of GHP that contributed for a significant difference in the mean amplitudes of N1, P2 and 2 N1 components of ACC. The /*U*/ value and p-value of Mann-Whitney *U* test on amplitude of ACC are tabulated in Table 4.53. The results of Mann-Whitney *U* test revealed that there was no significant difference between the sub-groups of 'A', 'B', and 'C' (in GHP) in amplitude of N1, P2 and 2N1 components of ACC. The sub-groups of 'A', 'B' and 'C' were significantly different from sub-group 'D' in the amplitude of N1, P2 and 2N1 component of ACC.

Table 4.53

|U|-value and p-value of Mann-Whitney U test on amplitudes of N1, P2 and 2N1 components of ACC in good hearing aid performers

Sub-groups	*Amplitude (µV) of ACC components*					
	N1		*P2*		*2 N1*	
	U	*p -value*	*U*	*p –value*	*U*	*p -value*
'A' vs. 'B'	4.00	0.29	51.50	0.07	7.00	0.87
'A' vs. 'C'	21.00	0.73	9.00	0.09	9.00	0.09
'A' vs. 'D'	0.00	0.00***	6.00	0.00***	17.00	0.05*
'B' vs. 'C'	16.00	0.93	6.00	0.09	6.00	0.10
'B' vs. 'D'	4.00	0.00***	0.00	0.02*	1.98	0.04*
'C' vs. 'D'	2.61	0.00***	1.98	0.04*	4.00	0.00***

Note: * = p<0.05; ***= p<0.001

Further, Mann-Whitney *U* test was carried out to see which sub-group of PHP contributed for a significant difference in the mean amplitude of N1, P2 and 2 N1 components of ACC. The /*U*/ value and p-value of Mann-Whitney *U* test on amplitude of ACC are tabulated in Table 4.54. The results of Mann-Whitney *U* test revealed that there was no significant difference between sub-group 'B' and 'C' in amplitudes of N1, P2 and 2N1

components of ACC. However, sub-groups of 'B' and 'C' differed significantly from sub-group 'D' in amplitudes of N1, P2 and 2N1 component of ACC.

Table 4.54

|U|-value and p-value of Mann-Whitney U test on amplitude of N1, P2 and 2N1 components of ACC in poor hearing aid performers

Sub-groups	*N1*		*P2*		*2N1*	
	U	*p value*	*U*	*p value*	*U*	*p value*
'B' vs. 'C'	12.00	0.49	6.00	0.08	15.00	0.86
'B' vs. 'D'	2.00	0.00***	3.00	0.01*	1.98	0.04*
'C' vs. 'D'	8.00	0.00***	6.00	0.04*	0.00	0.00***

Note: * = p<0.05; ***= p<0.00

In GHP, the data on amplitude of N1, P2 and 2N1 components of ACC from sub-groups of 'A', 'B' and 'C' were combined. Similarly in PHP, the data on amplitude of N1, P2 and 2N1 components of sub-groups of 'B' and 'C' were combined. The mean and standard deviation on amplitude of ACC of combined sub-group of 'ABC' of GHP; combined sub-group of 'BC' of PHP; and sub-group 'D' of both GHP and PHP are tabulated in Table 4.55. The amplitude of N1, P2, and 2N1 components of ACC was higher in GHP than in PHP in both combined sub-group 'ABC' and sub-group 'D'. In order to know if this difference was significant between GHP and PHP in combined sub-group of 'ABC' and sub-group 'D', Mann-Whitney *U* test was done. The result revealed a significantly higher amplitude of N1 (/*U*/ = 39.00, p= 0.002) was noted in GHP (in the combined sub-group of ABC) than in PHP (in the combined sub-group of BC). In sub-group 'D', a significantly higher amplitude of P2 (/*U*/ = 52.00, p= 0.041) was found in GHP than in PHP.

Table 4.55

Mean and SD of amplitude of ACC components in sub-groups of GHP and PHP

Sub-groups	*Hearing aid Performers (No. of participants)*	*Amplitude (µV) of ACC components*		
		N1 M±SD	*P2 M±SD*	*2 N1 M±SD*
'ABC'	*GHP(19)*	-1.73±0.64	1.41±0.49	-1.70±0.55
'BC'	*PHP(12)*	-1.39±0.34	1.09±0.74	-1.37±0.38
'D'	*GHP(16)*	-0.72±0.33	0.90±0.47	-1.44±0.68
'D'	*PHP(12)*	-0.62±0.26	0.54±0.46	-1.33±0.78

The data on 2P2 component of ACC were combined between sub-groups in GHP, as there was no significant difference. Similarly, the data on amplitude of 2P2 component in PHP was combined, as there was no significant difference between sub-groups. The combined data of 2P2 amplitude of ACC were compared between good and poor hearing aid performers. The mean amplitude of 2P2 was higher in GHP (1.46±0.74) than in PHP (0.93±0.64), and this difference was found to be significant on Mann-Whitney *U* test (U=229.50, p =0.03).

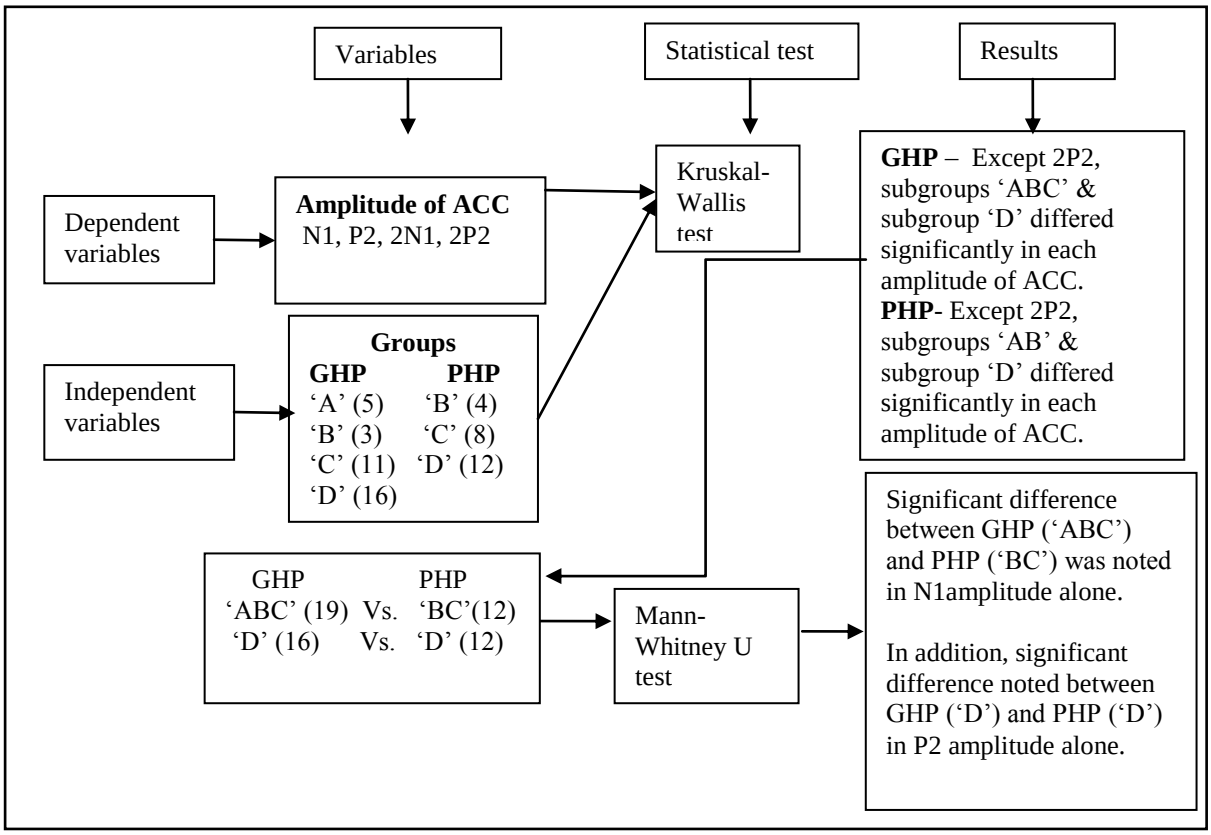


Figure 4.50. Illustrates statistical tests performed on data of amplitude of ACC obtained from GHP and PHP sub-groups.

To summaries, at the ear canal, there was no significant difference between unaided and aided conditions in the each spectral parameter of speech syllables. In addition, the temporal parameter of each speech sound is minimally altered as indicated by EDI measurement. The acoustic cues available at the brainstem level are less precisely represented in clinical group than compared to the normal hearing group. The shallower slope of V-A, reduced encoding of FFR for each stimulus was noted in the clinical group compared to their counterpart. The encoding of F_0 energy and F_1 energy of FFR for each stimulus were significantly reduced in clinical sub-group 'D' than in normal hearing group.

In the cortical level, a shallower slope of N1-P2 was noted in clinical group compared to their counterpart, this difference found significant. Further, prolonged latencies and reduced amplitudes of ACC components were found in clinical group than in normal

hearing group. In addition, except the latencies of N1 and 2P2 component of ACC in sub-group ‘D’, significantly earlier latencies were noted in normal hearing group than in clinical group. In addition, significantly higher amplitude of 2N1 component of ACC was noted in normal hearing group than in the clinical group.

Further, the clinical group was classified into good and poor hearing aid performers using ANL measure. This was done to investigate to know how the amplified speech syllables are represented at brainstem and cortical levels of auditory pathway. The findings of these investigations were as follows. At the brainstem level, the steeper slope of V-A, higher F_0 energy and F_1 energy were found in GHP than in PHP and these dfferences did not reach significant. However, the encoding of F_0 of FFR for each stimulus found significantly reduced in PHP than in GHP.

At the cortical level, the steeper slope of N1-P2 was noted in GHP than in PHP. Further, earlier latencies and higher amplitudes of ACC components were found in GHP than in PHP. The latencies of P2 and 2N1 components of ACC found significantly earlier in the GHP (‘ABC’) than in PHP (‘BC’). In addition, a significant earlier latency of P2 component of ACC was noted in the sub-group ‘D’ of GHP than in the PHP. However, the amplitude of N1 component of ACC found significantly higher in GHP (‘ABC’) than in the PHP (‘BC’). In sub-group ‘D’, the amplitude of P2 found significantly higher in GHP than in PHP.

CHAPTER - 5 DISCUSSION

The aim of the study was to investigate the representation of amplified speech at different levels of the auditory pathway in individuals with sensorineural hearing loss. In order to address this, the objectives were to a) measure the output of hearing aid at the ear canal; b) to compare the representation of speech syllables at the auditory brainstem level between clinical group and the group with normal hearing c) to compare the representation of speech syllables at the auditory cortical level between clinical group and normal hearing group, and d) to study the brainstem responses; cortical responses in good and poor hearing aid performers. The findings of the present study have been discussed under each objective.

To evaluate the objectives, the study was conducted in three phases. In Phase 1, the participants were assigned to either clinical or normal hearing groups on the basis of the audiological evaluations. The participants were assigned into four sub-groups on the basis of age. In addition, the clinical sub-groups were categorized as either good or poor hearing aid performers based on the scores of acceptable noise levels (ANL). In Phase 2, the change in the signal or speech syllables (/ḍa/ and /si/) at the output of the hearing aid was measured in the ear canal, of the participants in clinical group, using probe tube microphone measurement. In Phase 3, the representation of speech syllables (/ḍa/ and /si/) was studied at the brain stem level using FFR and at the cortical level using LLR and ACC, in both clinical and normal hearing groups. The FFR, LLR and ACC were recorded in the unaided condition only in the normal hearing group, whereas, these were measured in both unaided and aided conditions in the clinical group. A discussion of the results of Phases 2 and 3 are being provided in the following sections.

At the ear canal level, spectral and temporal parameters of speech syllables in unaided and aided conditions

The output recorded at the ear canal in the unaided and aided conditions for each stimulus was analyzed spectrally, only in the clinical group. The spectral parameters such as F_0, and formant frequencies (F_1 and F_2) at the onset and offset portions of transition in each stimulus were compared between clinical sub-groups. In addition, spectral energy at octave frequencies from 0.25 kHz to 8 kHz of each stimulus was compared between clinical sub-groups. Except spectra, the F_0 and format frequencies were compared between unaided and aided conditions. Further, the temporal parameter, i.e., EDI, computed for each stimulus by adopting the method given by Fortune, Woodruff, and Preves (1994), was also analyzed.

Effect of hearing aid processing on spectral parameters of speech stimuli. In the unaided condition, the spectral parameters (i.e., F_0, and formant frequencies F_1 and F_2 at the onset and offset of transition) were similar in the sub-groups for both the stimuli. This could be due to complete development of the external ear by the age of 15 years (Keefe, Bulen, Arehart, & Burns, 1993). Though narrowing of ear canal with aging has been reported (Schow, Randolph, &Nerbonne, 1980), in the present study it was noted that its effect on alteration of spectral parameters was negligible in the age group included in the study.

In the aided condition for each stimulus, the spectral parameters (F_0, F_1 and F_2) did not change significantly between the sub-groups. This could be because of the same hearing aid that was used for all the participants, and similar anatomical

structure of the external ear in all the participants from the age range of 15 to 65 years.

Further, the spectral parameters of the speech syllables in the unaided and aided conditions were compared to know the extent of spectral alteration brought about by the hearing aid. The aided spectral parameters in terms of frequency were similar to that in the unaided condition. Thus, it can be inferred that for the speech syllables tested, the hearing aid preserves the speaker identity (as reflected by F_0), manner (as reflected by F_1) and place (as reflected by F_2) features. This could be due to factors that determine the frequency response of the hearing aid.

In the spectral energy, each frequency showed minimal change with similar magnitude of variation as reflected from standard deviation in sub-groups, from unaided and aided conditions for both stimuli. This could be because of the age range of the participants i.e., 15 years to 65 years. In this age range, the cytoskeleton structure of ear canal is reported to have been developed completely (Keefe, Bulen, Arehart, & Burns, 1993). The smaller magnitude of variation reflected by the standard deviation could be because of insertion depth of probe tube microphone of the real ear measurement system used of measurement. Though precautionary measures were taken in placing and maintaining the length of the probe tube in the ear canal during the course of recording, the inter-subject variation in the representation of energy across frequencies is more likely. Caldwell, Souza, and Tremblay (2006) have reported 2-6 dB variation in each frequency when different probe tube insertion depths were used.

In the unaided condition for /ɗa/ and /si/ stimuli, the energy measured in octave frequencies was relatively larger at 2 kHz than at other frequencies. Further, there was a decline in energy after 4 kHz i.e., an approximately 10 dB per octave for /ɗa/ stimulus and 12 dB per octave for /si/ stimulus (Figures 3.4 & 3.3). This pattern of energy representation as a function of frequency, for both the stimuli, could be because the frequency response of microphone used in recording the target test stimuli.

In the aided condition for /ɗa/ stimulus and /si/ stimulus, the energy measured was relatively lesser at the extreme frequencies, i.e., at low frequency (0.25 kHz) and high frequencies (above 4 kHz). Thus, at extreme frequencies, the mean energy difference between unaided and aided conditions is less. At low frequency (0.25 kHz), lesser energy could be because of lesser gain in the low frequency given by the prescriptive formula (Dillon, Katsch, Byrne, Ching, Keidser, & Brewer, 1998). Additionally, lower energy noted in the low frequency region of /ɗa/ and /si/ stimulus could also be because of frequency response of the hearing aid. The low frequency cut-off of the frequency response of the test hearing aid was 0.21 kHz. At high frequencies i.e., above 4 kHz, the frequency response of /ɗa/ and /si/ stimulus had energy till 4 kHz (as noted from unaided condition), and thereafter the energy reduced approximately at the rate of 10 dB per octave for /ɗa/ and 14 dB per octave for /si/ stimulus. Therefore, more energy was being measured in the frequency range from 0.5 kHz to 4 kHz. Thus, remarkable energy differences in unaided and aided conditions were noted in the frequency range from 0.5 kHz to 4 kHz. Thus it can be inferred that there is amplification in the mid-frequency region of the hearing aid.

Effect of hearing aid processing on temporal parameter (EDI) of speech stimuli. The EDI that reflected the difference between the unaided and aided versions of each of the stimulus was analyzed. Though, the mean EDI was larger in sub-group 'D', followed by sub-groups 'C', 'B' and 'A' for each stimulus, there was no significant difference in the EDI between the sub-groups. Thus, the mean and standard deviation of EDI for the combined group were 0.355±0.06 for /ɗa/ and 0.352±0.05 for /si/ stimuli.

Fortune, Woodruff, and Preves (1994) have opined that an EDI variation of up to 0.03 reflect excellent agreement between any two envelopes; in this context, the unaided and the aided envelopes. In the present study, the mean EDI values for the combined group for /ɗa/ and /si/ reveal that there is a minimal difference in the envelope between the unaided and aided conditions. This could be due to the decreased modulation depth in the aided condition due to compression in different channels of the hearing aid. It would be interesting to investigate the EDI in compression and linear modes of hearing aid.

From the findings of the present study, it can be inferred that the hearing aid preserves the spectral information (F_0, F_1, F_2), at the level of ear canal. However, the envelope of speech stimuli is altered to a minimal extent in the aided condition, as reflected by the temporal measure, the EDI.

At the brainstem level, the representation of speech syllables in clinical group and group with normal hearing

At the brainstem level, the slope of V-A was registered from the transient portion of /ɗa/ stimulus. The F_0, F_0 energy and F_1 energy were analyzed from FFR elicited from transition portion of each stimulus. These responses obtained in aided condition from clinical sub-groups and in unaided condition from sub-groups with normal hearing were compared in order to know the effect of amplification. The reasons for neural encoding of acoustic cues are discussed as a function of age in both groups and then the extent to which the aided responses in the clinical group approximated the unaided responses in the group with normal hearing.

Representation of speech syllables at the brainstem level of auditory pathway in clinical sub-groups. In the clinical group, the brainstem response was absent in the unaided condition. This was due to the presentation level (i.e., 65 dBSPL) of the stimuli (/ɗa/ and /si/), which was sufficient for reaching audibility to elicit the brainstem response. Some of the important findings and discussion are reported in the following sections. The slope of V-A at brainstem level in clinical group was shallower with respect to age. Slight variation in F_0 of FFR, reduced F_0 energy and F_1 energy of FFR with respect to age were noted. The encoding of F_0 was relatively less precise in sub-group D than other sub-groups, which was found significant. In the F_0 energy and F_1 energy, sub-groups 'A' and 'B' were significantly higher from sub-group 'C' and sub-group 'D'.

In the aided condition, the latencies of transient response V-A was shallower for /ɗa/ stimulus, with increase in age. This finding was similar to that observed in the

group with normal hearing. Rowe (1978), and Jerger and Hall (1980) have opined that though the hearing loss was alleviated by the amplification device, hearing loss with aging alters the physiological mechanism of volume conduction and / or neurochemical regulatory process. This could be either due to aging or the impact of hearing loss on physiological mechanism even after having fitted with a hearing aid or a complex interaction between the two. Other variables such as the extent of auditory deprivation could also have an effect, but was not investigated in the present study, as that was not in the objectives of the present study.

The derivation of F_0 from FFR to each stimulus (/ɗa/ and /si/) was well represented. The F_0 value was very close to that of the aided filtered raw stimulus. This implies that the spectral content of the signal was relayed / preserved quite well from the hearing aid up to the level of the brainstem. However, as the sub-group 'D' was significantly different from other groups in terms of F_0, it could be inferred that the $F_0$was represented as lowered in frequency with age, at the level of brainstem. In literature, the explanation given is manifold, i.e., in terms of neurotransmitters, synchronicity and physiological processes. This could be because of loss of myelination, loss of neurons and altered inhibitory neurotransmitters such as GABAergic and glycinergic due to complex interaction of aging and hearing loss (Zettel, Zhu, O'Neill, & Frisina, 2007). This could also be due to temporal asynchronicity among neurons and reduced mechanism in ionic channel from altered physiological processes that disrupt the phase locking capacity of auditory brainstem neurons (Jewett & Williston, 1971).

Further, the F_0 energy and F_1 energy of FFR to both the stimuli reduced with age. For these parameters of FFR, the sub-groups 'A' and 'B' are significantly different from sub-groups 'C' and 'D'. The result suggests that the cues responsible for the manner of articulation reduced with age, as this was found to be significantly different in older sub-groups C and D. This could in turn lead to the speech recognition difficulties often faced by older adults even when fitted with a hearing aid, which has often been reported in literature (Humes, 2002).

Representation of speech syllables at the brainstem level of the auditory pathway in the sub-groups of normal hearing. The important findings are the slope of V-A was shallower with respect to age. Neurally slight difference in the representation of F_0 of the stimulus in FFR was noted with respect to age. The F_0 energy and F_1 energy of FFR reduced as a factor of age. The sub-group D was significantly lesser from other sub-groups in F_0 energy and F_1 energy.

The slope of V-A measures the synchronization of neuron, subsequent transmission and summation of underlying neural activity (Wible, Nicole, & Kraus, 2005). The slope of transient response V-A reflects the lateral lemniscal input to the inferior colliculus (wave V), and subsequent dendritic processing in the inferior colliculus (wave A) (Møller & Jannetta, 1985). In the present study, the slope of V-A was shollower with increase in age. The shallower slope of brainstem encoding suggests less precise timing of generation and/or transmission of responses in the lateral lemniscus and/or inferior colliculus.

The possible reason for shallower slope of V-A in older adults could be attributed to reduced inhibitory neurotransmitter. The altered physiological processes due to aging at the peripheral system block the inhibitory neurotransmitters, GABAergic and glycinergic, at the inferior colliculus. The resulted impact reflected in the imbalance of excitation and inhibition of neurons and finally disrupts the temporal processing. This has been reported in rats (Walton, Frisina, Ison, & O' Neill, 1998).

Further, animal models have suggested several physiologic mechanisms which may be related to decline in temporal inhibition (Clinard, Tremblay, & Krishnan, 2010). These mechanisms include decreased neural inhibition (Caspary, Schatteman, & Hughes, 2005), temporal jitter or greater variance affecting neural firing or synchrony (Pichora-Fuller & Schneider, 1992), and longer neural recovery time (Walton, Frisina, & O'Neill, 1998). Caspary, Schatteman, and Hughes (2005) reported that though the cochlear nucleus receives input from inner hair cells and sets up parallel processing pathways in the brainstem for sound analysis and perception, there is a decline in the glycinergic inhibitory neurotransmitter with age. Further, they opined that this decline in neurotransmitter reduces tuning of input to the vertical cells of dorsal cochlear nucleus (DCN). This in turn passes the afferent impulses to fusiform cells of DCN. These physiological alterations leads to impaired perception as the complex neural circuits fail to effectively transform the action potentials into the heard sound (Chisolm, Willott, & Lister, 2003). In addition, there is a significant structural change at the brainstem level in terms of neuron number, density and size (Willott, Jackson, & Hunter, 1987), and altered synaptic ending (Keithley & Croskrey, 1990). All or some of these are speculated to be the cause of disruption in temporal processing with aging.

Though no significant difference was noted between the sub-groups, the F_0 encoded at brainstem level from each sub-group differed from the F_0 of the filtered waveform of the test stimulus (/ɗa/ and /si/). This difference (F_0 encoded vs. F_0 of test stimulus) increased with advancing age. The findings suggest that neurons at auditory brainstem fail to precisely phase lock, to the order of fractions of milliseconds (Kraus & Nicol, 2005). However, this slight difference between encoding of F_0 and the F_0 in test stimulus with aging does not mean that the participants find it difficult to recognize the speaker identity.

Energy of the spectral component corresponding to the stimulus fundamental frequency conveys speaker identity. Energy of the spectral component corresponding to frequency of first formant of the stimulus gives information on phonetic content in the syllables, i.e., the manner cues of the syllable recognition (Russo, Nicol, Musacchia, & Kraus, 2004). The F_0 energy and F_1 energy of FFR reduced in each stimulus (/ɗa/ and /si/) as a factor of age. The sub-group 'D' alone was significantly different from other sub-groups. The result of the present study is in accordance with the findings reported by Sinha and Barman (2014). The possible reasons for reduction in F_0 energy and F_1 energy for each stimulus could be due to the biochemical, physiological and anatomical alterations. This has been inferred as Frisina and Walton (2006) have reported changes in biochemical, physiological and anatomical processes. The biochemical investigation suggested increased firing rates in cochlear nucleus due to decline in glycinergic inhibitory neurotransmitter, physiological alteration of synaptic processing and anatomical reduction of neurons in the cochlear nucleus and their output pathways.

Comparison of aided slope of V-A from clinical group with unaided slope of V-A from group having normal hearing. In the aided condition, the V and A components of FFR were present in clinical group. This is because the hearing aid fitted to clinical group has addressed the issue of audibility. It can be inferred that the hearing aid provides sufficient amplification to the burst of the stop consonant /ɗ/ in order to elicit the V and A peaks. This consonant has a rapid onset, low amplitude, high frequency and transient feature (Skoe & Kraus, 2010). This results in audibility of the rapid onset portion of /ɗ/, which is reported to be an important feature for the excitation (V) and inhibition (A) of auditory brainstem response (Skoe & Kraus, 2010).

The mean slope of V-A was slightly shallower in clinical group compared to the group with normal hearing. However, this difference between the two groups was not statistically significant. The reason for shallower slope in clinical group than in the normal group could be physiological alteration in the peripheral mechanism and its consequence at the brainstem level and also the complex interaction of hearing aid output relayed from ear canal till brainstem level, though the audibility was being compensated from the hearing aid to an extent.

Comparison of the aided F_0, F_0 energy and F_1 energy from clinical group with unaided F_0, F_0 energy and F_1 energy from group having normal hearing. In combined sub-groups 'ABC' of normal and clinical participants, the F_0 encoding for /ɗa/ or /si/ was well represented at the brainstem level to each stimulus. In addition, in the normal sub-group D, the F_0 encoding (132.60 Hz for /ɗa/ stimulus and 142.11 Hz for /si/ stimulus) was almost close to F_0 of the filtered raw stimulus (i.e., 135.7 Hz for

/ɗa/ stimulus and 145.7 Hz for /si/ stimulus). Further, in the clinical sub-group D, the F_0 encoding at the brainstem level (130.46 Hz) for /da/ stimulus is almost similar to aided stimulus (134.95 Hz). However, the representation of F_0 in clinical sub-group D at the brainstem level for /si/ stimulus is 130.48 Hz, which differed from F_0 of aided stimulus (144.74 Hz).

Thus, the results of F_0 of FFR in the present study revealed that for both the stimuli, the F_0 of FFR at the brainstem level showed no significant difference between clinical group and the group with normal hearing in the combined sub-groups of 'ABC'. This was also true for sub-group D for /ɗa/ stimulus. It can be inferred that the neurons of the auditory brainstem phase locked to the time varying acoustic features of a stimulus eliciting the FFR (Krishnan & Durrant, 1992). However, for /si/ stimulus, a significant difference for F_0 of FFR was noted between the sub-group D of clinical group and group with normal hearing, such that the mean F_0 of FFR was relatively well represented in the group with normal hearing (i.e., 142.11 Hz) compared to the clinical group (i.e.,130.48 Hz). In clinical sub-group D, a difference of 14 Hz was noted between encoding of F_0 of FFR (/si/ stimulus) and aided stimulus. This difference may not cause any change in perception of speaker identity. This is because according to Iles (1972) there is no difference in perception of speaker identity as frequency (F_0) changed up to ± 25 Hz.

To summarize, it was noted earlier that the hearing aid preserved the spectral parameters of speech at the ear canal. At the brainstem level, the FFR recorded in aided condition was elicited by the stimulus that was presented at 65 dB SPL. Thus, the aided stimulus reached the audibility level that is required for elicitation of FFR.

The low spontaneous nerve fibres of the auditory nerve carry information from cochlea through the process of rate-place mechanism. This information in turn reaches the neurons of inferior colliculus to elicit the FFR at the cochlear nucleus of the brainstem through the complex auditory pathway (Krishnan, 2002). The neurons at the brainstem precisely fire in synchrony with F_0 (1/period) of stimulus frequency. The F_0 energy was relatively higher than F_1 energy of FFR and this was as expected. This pattern was found in both normal and clinical groups. The F_0 (1/period) of the stimulus frequency represents the first harmonic. The harmonic number corresponding to frequency of formant and was determined from dividing the frequency of formant by the fundamental frequency. For example, if the frequency of first formant of a stimulus is 519 Hz, and the fundamental frequency is 135 Hz, the fourth harmonic (i.e., 519/135) corresponds to the frequency of first formant. The harmonics corresponding to F_0 will have higher energy than the other harmonics corresponding to formant frequencies. This is because, acoustically, it is documented that the energy reduces with increased harmonics (Fant, 1960). Physiologically, the neurons fire more synchronously or maximally to the harmonics corresponding to the fundamental and formant frequencies. The neurons inhibit the other harmonics thereby maintaining frequency selectivity (Krishnan, 2002). The result of the present study is in agreement with that reported by Krishnan (2002). In his study too, it was reported that the highest energy was noted for F_0 and relatively lesser energy for other harmonics corresponding to the formants for the three synthetic vowels.

It was noted that the F_0 energy and F_1 energy of FFR to both the stimuli was higher in normal hearing group than in clinical group. Though the hearing in the participants of clinical group was aided from amplification device of appropriate gain,

it failed to completely address the issue of impaired neural mechanism (Anderson, Parbery-Clark, White-Schwoch, Drehobl, & Kraus, 2012). The reasons for the reduction of F_0 energy and F_1 energy in clinical group compared to the normal hearing group could be two possible sources of distortions, i.e., distortion from hearing aid and / or physiological distortion from cochlea. It was noted earlier that hearing aid had a negligible amount of distortion as confirmed through electro-acoustic measurement. Further, the hearing aid precisely processes the spectral parameters, which was noted from probe tube microphone measurement in the ear canal. The other source of distortion is still unresolved, as to how the cochlear distortion in sensorineural hearing loss is relayed to the auditory brainstem level. Thus, further research is warranted to resolve this issue.

At the cortical level, the representation of speech syllables in clinical group and group with normal hearing

At the cortical level, the slope of N1-P2 was registered from the transient portion of /ɗa/ stimulus. The latency and amplitude of ACC were tabulated for /si/ stimulus. It must be noted that these responses were obtained in aided condition from clinical sub-groups and in unaided condition from sub-groups with normal hearing.

Representation of speech syllables at the cortical level of auditory pathway in the clinical sub-groups. The cortical responses were absent in the unaided condition as the stimuli were not audible. In the aided condition, slope of N1-P2 complex of LLR to /ɗa/ stimulus and ACC to /si/ stimulus were present in all the participants when presented at 65 dB SPL. The results of the present study

demonstrate that use of hearing aid has an impact on the timing (as reflected by latency) and strength (as reflected by amplitude) of the cortical processes involved in representation of time varying acoustic cues, which are inherent in speech (Korczak, Kurtzberg, & Stapells, 2005). These findings suggest that hearing aid activated a large pool of cortical neurons which contributed to the response seen in the aided condition at 65 dB SPL (Whiting, Martin, & Stapells 1998). The improved audibility of both the stimuli (/da/ and /si/) is the prime factor for elicitation of the response.

With advancement in age, the ACC response in terms of onset of consonant (i.e., N1 and P2) and transition to the vowel (i.e., 2N1 and 2P2) were prolonged, the amplitudes were reduced, and the slope of N1-P2 was shallower. In addition, except the amplitude of 2P2, the latency and amplitude of ACC are significantly different in sub-group-D from the other sub-groups. Prolonged timing and reduced strength in aided cortical representation with respect to age could be due to factors such as loss of myelination, neuron loss, and altered inhibitory neurotransmitter (Zettel, Zhu, O'Nell, & Frisina, 2007). Yet another reason could be the protein and lipids from malfunctioning of the neurons due to aging and / or hearing loss combine with free radicals produced from healthy neuron cell and thus reduce the efficiency of the neurons (Walton, Frisina, & O'Neil, 1998).

Representation of speech syllables at the cortical level of auditory pathway in the sub-groups with normal hearing. The results of the present study reveal that the components of LLR and ACC change in a systematic manner with age. The slope of N1-P2 complex of LLR was shallower with age. The latencies of N1 and P2 of ACC to onset of consonant; 2N1 and 2P2 of ACC to the onset of vowel

(Ostroff, Martin, & Boothroyd, 1998) were prolonged with age. In addition, amplitude of ACC reduced with age. These results are in accordance with the reports by Callowey and Hallidey (1973), and Goodin, Squires, Henderson, and Starr (1978). The possible explanation for the above results could be due many factors. They include a) decrease in rate of transmission due to the altered function within the neural population (Caspary, Schattenan, & Huges, 2005), b) decrease in the amount of both excitatory and inhibitory transmitters and / or their associated enzymes with age (Goodin, Squires, Henderson, & Starr, 1978) c) decrease in conduction velocity due to age related alteration in myelination (Pfefferbaum, Ford, Roth, & Kopell, 1980), and d) alteration in structural and / or neurochemical mechanism regulating the excitatory and inhibitory processes (Goodin, Squires, Henderson, & Starr, 1978).

Comparison of the cortical responses (slope of N1-P2, latency and amplitude of ACC) in clinical group and group with normal hearing. The slope of N1-P2 of LLR was shallower in the clinical group than in the normal hearing group. Further, the mean latencies of N1 and P2 (i.e., onset of consonant) components of ACC were prolonged and their mean amplitudes were reduced in combined clinical sub-group of 'ABC' compared to the combined normal hearing sub-groups of 'ABC'. Similar result was noted in sub-group 'D'. A significant difference was noted only in the latency of P2 between normal hearing and clinical groups. This is could be due to the reduced signal to noise ratio after being processed by the hearing aid. The reduced signal to noise ratio is due to the added internal noise of the hearing aid. A similar finding was noted by Korczak, Kurtzberg, and Stapells (2005). Another factor could be the hearing loss induced changes at the cortical level leading to altered metabolic activity (Willott, 1991). Such peripheral hearing loss has a direct effect on the quality

of the neural information that is transmitted to the auditory cortex through the auditory pathway. Hellstrom and Schmiedt (1991) are of the opinion that cochlear hearing loss results in loss of synchronous neural firing in spiral ganglion. This indirectly influences the central neural processing.

Similar results were noted in the latencies and amplitudes of 2 N1and 2 P2 (onset of vowel). A significant difference was noted in latencies and amplitudes of 2 N1 and 2 P2 between the normal hearing and clinical combined sub-group ABC. Except for latency and amplitude of 2 P2, a significant difference was noted in latencies and amplitudes of 2 N1 between the normal hearing and clinical sub-group 'D'. It indicates that the participants of clinical group require more neural processing time to detect a time varying portion of the vocalic segment of the syllable /si/. Hence, the processing is relatively prolonged and strength of cortical response is reduced, even when the reduced audibility due to hearing loss was compensated by a hearing aid. The reason could be energies of both the consonant (/s/) and vowel (/i/) was well above the compression threshold of 55 dB SPL, such that compression was relatively more for vowel than for the consonant. This in turn resulted in reducing the contrast of CV boundary, which is an important factor for eliciting the transition response. The results of the present study are in accordance with the research report on neural representation of amplified speech at cortical level in individuals with normal hearing (Tremblay, Billings, Friesen, & Souza, 2006).

From the findings of the present study, it can be inferred that the processing delay of hearing aid and altered physiological mechanism due to sensory loss and its concomitant changes relayed at the cortical level could partly have led to prolonged latency and reduced amplitude of the ACC. In addition, the temporal features of the

speech sound are altered after amplification, which was reflected by the envelope difference index (EDI). Thus, it can be deduced that there is an interaction between the amplified signal and their neural representation. The finding of this study supports the conclusion that though hearing loss is alleviated by amplification device, prolonged processing and reduced strength of central auditory processing in clinical group could be due to temporal alteration from the hearing aid and interaction of hearing aid output with the unknown mechanism of cochlear distortion in sensorineural hearing loss and its concomitant change in the higher auditory pathway.

Comparison of brainstem responses and cortical responses in good and poor hearing aid performers

At the brain stem and cortical levels of auditory pathway, the response obtained from good hearing aid performers (GHP) and poor hearing aid performers (PHP) at each auditory level is being discussed in the light of neuro-physiological studies.

Comparison of slope of V-A in good hearing aid performers and poor hearing aid performers. The slope of V-A was steeper in good hearing aid performers than in poor hearing aid performers. This suggests that the auditory processing mechanisms of listeners who are willing to accept higher levels of background noise, as indicated by ANLs (i.e., in GHP), are distinct from those who are not (i.e., in PHP). Steeper V-A slope in the current study appears to suggest that good hearing aid performers exhibit faster neural conduction time at the level of upper brainstem than poor hearing aid performers. The faster conduction time results from greater depolarization i.e., decreased trans-membrane threshold and increased neural

firing or inhibition (Ahveninen, Jaakelainen, Pekkonen, Hallberg, Hietanen, Naatanen, & Sillanaukee, 1999). The trans-membrane current flow helps to generate action potential travelling along the axon of a neuron. The action potentials generated from peripheral structures of auditory pathway propagates to central pathway (Oxenham & Bacon, 2003).

Comparison of F_0 of FFR, F_0 energy and F_1 energy in good hearing aid performers and poor hearing aid performers. For /ɗa/ stimulus, the mean F_0 of FFR was higher in GHP (133.46 Hz) compared to PHP (128.84), such that the difference was found to be significantly different. This was true for F_0 of FFR for /si/ stimulus between GHP (134.42 Hz) and PHP (130.84 Hz). Further, the F_0 of the aided stimulus of /ɗa/ was 134.95 Hz and that for /si/ was 144.74 Hz. The difference in F_0 (in Hz), between encoding of F_0 at brainstem level and F_0 of aided test stimulus was 1 Hz in GHP and 6 Hz in PHP for /ɗa/ stimulus. Similarly, the difference noted was 10 Hz in GHP and 14 Hz in PHP for /si/ stimulus.

The mean difference between the GHP and PHP in the encoding of F_0 was 5 Hz for /ɗa/ and 4 Hz for /si/ stimulus. Though this difference was significant in the encoding of F_0 between GHP and PHP for both stimuli, this may not bring a change in speaker identity. This is because, according to Iles (1972), a change of up to ±25 Hz in the F_0 will not bring about a change in speaker identity. The finding of the study is in accordance with the research report by Horii (1979) who reported that a difference of greater than 25 Hz in the F_0 between the two same stimuli does not cause difference in speaker identity. Additionally, the intra-subject variability of F_0 in a

normal vocal effort ranged between ± 9.6 Hz (Abberton, 1976).Thus, it can be inferred that the mean $F_0$of FFR to /ɖa/ and /si/ stimuli was neurally well represented in both good and poor hearing aid performers.

It was clear that the F_0 energy and the F_1 energy of FFR to each stimulus in GHP was higher than in PHP, but this difference was not significant in each sub-group. The higher energy of F_0 and F_1 in GHP might be due to stronger efferent fibres that inhibit the other harmonics that do not correspond to the fundamental frequency and formant frequencies. This is in accordance with the research reports by Ashmore (1991) and Knight (1997). To be more specific, the central afferent mechanism is stronger in the group of GHP such that neurons at inferior colliculus fire precisely to the harmonics corresponding to F_0 and F_1. In addition, the efferent mechanism might be stronger such that the efferent fibres inhibit the other harmonics which do not correspond to the fundamental frequency and formant frequencies, thereby fine tuning the auditory input. The excitatory and inhibitory mechanisms of neurons of the underlying neural generator of the inferior colliculus in GHP fire more or less precisely to the corresponding F_0 and F_1 components of the stimulus. The inference of the present study is supported by the findings reported by Krishnan (2002). He demonstrated that efferent auditory pathway suppresses the energy adjacent to the harmonics corresponding to F_0 and F_1 energy of FFR. Along with an active afferent pathway, the afferent auditory nerve generates the electrical activity more precisely corresponding to the F_0 and F_1 of the stimulus. This involves the release of neurotransmitter, thereby reducing the trans-membrane threshold and increase in neural firing.

In poor hearing aid performers, though the above explained physiological activity was present, probably a lack of precision in neural activity due to less sensitive afferent and weak efferent auditory pathway, might have failed to provide higher energy at harmonics corresponding to F_0 and F_1 of the each stimulus. It can be inferred from the present study that subtle physiological variations might be present in the inferior colliculus of the auditory pathway in the poor hearing aid performers with reference to that in good hearing aid performers.

Comparison of the cortical responses in good and poor hearing aid performers. The neurons in the auditory system exhibit hyperpolarization (inhibition) and depolarization (excitation) to the sensory input from synaptic junction (from cochlea to auditory nerve) till the brain. At the time of excitation, the neurotransmitter reduces the transmembrane threshold which in turn increases the neural firing. During inhibition, the neurotransmitter increases the transmembrane threshold, this in turn reduces the neural firing (Ashmore, 1991). Further, the descending pathway is much stronger in projection. Activation of medial superior olivary (MSO) complex bundle from stimulation of the contralateral outer hair cell shows inhibitory effect and this subsequently fine tune the primary afferent neurons (Davis, 1983). The inhibitory and excitatory mechanisms occur within a fraction of a millisecond in both afferent and efferent auditory pathway (Chao & Knight, 1997). However, in case of sensorineural hearing impairment, the excitatory mechanism is more prominent due to loss of outer hair cells and broadened auditory filters (Sommers & Humes, 1993). Conversely, the strength of the inhibitory mechanism decreases due to the loss of outer hair cells which in turn leads to lesser activation of MSO bundle (Guinan, 1996).

In the present study, the difference in cortical response between the GHP and PHP were present on the slope of N1-P2 complex of LLR, latency and amplitude of ACC components. Specifically, the slope of N1-P2 was steeper in GHP than in PHP, though this difference was not significant. Reduction in latency and increase in amplitude of ACC were found in GHP compared to PHP. This could be due to greater depolarization that leads to faster neural conduction at the cortical level. In addition, stronger efferent feedback might have played an instrumental role for earlier processing and increased strength in GHP. It suggests that in GHP, the central afferent and efferent mechanisms are more active. This speculation needs to be confirmed through further research.

The findings of the present study are in contrast to the reports by Tampas and Harkrider (2006), and Harkrider and Tampas (2006). They documented that in individuals with high ANLs, the latency of P1, N1 and P2 was prolonged; and amplitude of P1-N1, N1-P2 components of LLR was higher. They attributed the findings to sensitive auditory afferent and stronger efferent mechanisms. The difference in findings could be due to the variability in the selection of participants. In their study, only participants having normal hearing with a mean age of 24 years were used. However, in the present study both normal hearing and individuals with hearing impairment were participated. Thus, the difference in the participants might have brought distinction in the findings. The earlier latency and higher in amplitude of ACC found in GHP might be due to relatively effective efferent system than in PHP, which might have fine tuned the sensory input. Yet another reason could be greater depolarization in individuals who are suffering from hearing loss. This was supported by research report by Kotak, Fujisawa, Lee, Karthikeyan, Aokie, and Sanes (2005)

who opined that though the auditory filters are widened a greater depolarization noted in individuals having stronger efferent pathway.

The difference in GHP and PHP were seen in some components of AEP measurements and not in others. In some components, where the difference was noted, the difference was in the amplitudes and not in latencies (Mc-Fadden & Champlin, 2000). Yet another reason for this difference could be that the stimuli used were different in obtaining ANL and AEPs. In the procedure for obtaining ANL, to identify BNL, running speech in the presence of multi-talker babble stimulus was used. To elicit AEPs, tone burst of 500 and 3000 Hz was used in their study. However, in the present study, a speech passage was utilized to determine ANL and speech noise was used to establish BNL; and consonant-vowel syllables were used to elicit the response at the cortical level.

To summarize the present study, at the ear canal, though minimal temporal alteration was present after amplification, spectral information of speech was well preserved. The minimal alteration in the temporal envelope after processed through hearing aid reject the null hypothesis. Further, the available acoustic cues relayed at the auditory brainstem level. Each component of the brainstem response (i.e., F_0 of FFR, F_0 energy and F_1 energy) were represented close to the stimulus frequency in clinical group of younger adults (combined sub-group 'ABC'). This is true for both the stimuli. At auditory cortical level, steeper slope of N1-P2 was noted in younger adults. Further, the processing and strength of the ACC were represented precisely in the combined sub-group of 'ABC'.

However, in older adults (sub-group 'D'), the F_0 of FFR for /si/ stimulus was less precisely encoded with respect to stimulus frequency. A difference of 15 Hz between F_0 encoded at auditory brainstem and stimulus frequency does not bring any change in speaker identity. Encoding of F_0 energy and F_1 energy were reduced in older adults (sub-group 'D') than other groups (sub-group 'ABC'). In auditory cortical level, shallower slope of N1-P2 was noted. In the ACC, prolonged processing and reduced strength were found though the hearing loss was alleviated by amplification device. It infers that temporal asynchrony and reduced frequency resolution present in older adults even after fitted with hearing aid.

To investigate if the aided responses approximated the response from normal auditory pathway, the responses obtained at each level were compared between normal hearing and clinical groups. The response of slope of V-A elicited at auditory brainstem level was found to be steeper in the normal hearing group than in clinical group. The F_0 of FFR encoded was well represented in both normal and clinical groups with respect to the stimulus frequency, such that the difference in mean F_0 of FFR for each stimulus between normal hearing and clinical groups was minimal. Though the F_0 energy and F_1 energy reduced in clinical group than the normal hearing group, a significant difference was not noted. At the cortical level, in younger adults of clinical group, the slope of N1-P2, processing and strength of the ACC were almost the same as that of the normal group (combined sub-group of 'ABC'). However, in older adults of clinical group (sub-group 'D'), the slope of N1-P2 was shallower than in normal hearing group. Further, the response of ACC in older adults in clinical group revealed prolonged latency and reduced amplitude. These responses in clinical group reflect the asynchronous firing, interaction of hearing aid output and the

transduction process in peripheral ear pathology and also the blurring of CV boundaries after amplification. These results from older adults at cortical level of auditory pathway reject the null hypothesis.

Further, each response obtained at different levels of auditory pathway was compared between GHP and PHP. At the brainstem level, the slope of V-A was shallower, F_0 of FFR was represented less precisely with respect to the stimulus frequency and the F_0 and F_1 energies were reduced in PHP compared to that in GHP. At cortical level, the slope of N1-P2 was found shallower; and relatively prolonged processing and reduced strength in the ACC components were noted in PHP than in GHP. The reason for this finding could be relatively lesser generation of action potentials (i.e., less sensitive afferent auditory pathway) and weaker efferent auditory mechanism in PHP than in GHP. Thus, the results of the study imply that the physiological responses obtained from individuals with GHP at different levels of auditory pathway differed from individuals with PHP, thus rejecting the null hypothesis.

To conclude, though the hearing aid altered the temporal parameter minimally, the spectral paramerters of speech syllables were preserved. The available acoustic cues at the auditory brainstem and cortical levels differed in older adults of clinical group than in normal hearing group. It deduce that spectral and temporal alteration from hearing loss, the altered temporal asynchrony in older adults and minimal temporal alteration from hearing aid unble the auditory braintem and cortical neurons to encode the speech syllables precisely. Further, the responses at each level of auditory pathway were relatively better in GHP than in PHP. These findings speculate

that the stronger efferent auditory pathway which fine tune the acoustic input processed by afferent neurons in the clinical group individuals who accept more noise (GHP) than compared to those who do not accept more noise (PHP).

CHAPTER - 6 SUMMARY AND CONCLUSIONS

The purpose of the study was to investigate the representation of spectral and temporal parameters of consonant-vowel syllables at the ear canal in unaided and aided conditions, using probe tube microphone measurement. In addition, the purpose was also to know the subsequent representation of the aided speech syllables at the brainstem and cortical levels of the auditory pathway. Further, the encoding of signals at different levels of auditory pathway in individuals with hearing impairment was compared with the individuals having normal hearing. Comparison was also made between the encoding of speech syllables at each level of the auditory pathway in good and poor hearing aid performers.

The study was conducted in three phases. In Phase 1, audiological evaluation was carried out for selection and categorization of participants into clinical and normal hearing groups. Each clinical and normal hearing group consisted of four sub-groups based on age. Further, each participant in the clinical group was classified as either good or poor hearing aid performers based on the acceptable noise level (ANL) measure.

In Phase 2, the test hearing aid was fitted and optimized to each test ear of the participant in the clinical group. The output of the hearing aid for the CV syllables was recorded at the ear canal using probe microphone measurements, in the unaided and aided conditions. This recorded output of speech syllables were analyzed, using Adobe Audition 3.0, to obtain the spectral parameters such as F_0, and the frequencies of first two

formants F_1 and F_2. The first two formant frequencies F_1 and F_2 were analyzed at the onset and offset of the transition to each stimulus in unaided and aided conditions to determine spectral processing by the hearing aid. Further, the EDI was computed for each stimulus in order to know the extent of temporal alteration in the speech sound brought about by the hearing aid.

In Phase 3, the transient response followed by FFR at the brainstem level; and LLR and ACC at the cortical level were recorded from clinical and normal hearing groups in unaided condition. In addition, these potentials were recorded in aided condition from clinical group alone. At the brainstem level, the slope of V-A was calculated from the transient response. The 'Autocorrelation tool' of MATLAB was utilized on FFR to obtain F_0 for each stimulus; and Fast Fourier Transform technique was utilized on the waveform of FFR for each stimulus, to obtain energies at F_0 and F_1. At the cortical level, the slope of N1-P2 of LLR, and latency and amplitude of ACC components were analyzed.

The findings of the present study have been discussed in the light of findings reported in literature on neurophysiological research in hearing. The findings of the study are with reference to the stimuli used for the purpose. In each clinical sub-group, the output of hearing aid at the ear canal was recorded and analyzed for spectral and temporal parameters. The hearing aid preserved the spectral parameters quite well. However, there is minimal alteration in temporal parameter. This could be due to the decreased

modulation depth due to compression in different channels of the hearing aid and varied gain across the frequency.

Further, to know the extent of changes provided from amplification device in clinical group, the responses were compared with normal hearing group at brainstem and cortical levels of the auditory pathway. The results at brainstem level revealed that the slope of V-A was steeper in normal hearing group than in clinical group. The F0 of FFR encoded for each stimulus was well represented in both groups. In addition, the F_0 energy and F_1 energy reduced in clinical group than the normal hearing group, a significant difference was not noted. It inferred that after amplification the available acoustic cues are encoded precisely at the brainstem level.

At the cortical level, in the combined clinical sub-groups of 'ABC' the amplified responses of slope of N1-P2, latency and amplitude of ACC were almost same as that of normal hearing sub-group of 'ABC'. However, the amplified response of ACC in clinical sub-group 'D' revealed prolonged latency and reduced amplitude than unaided response obtained from normal hearing sub-group 'D'. These responses in clinical group reflect the asynchronous firing, interaction of hearing aid output and the transduction process in peripheral ear pathology and also the blurring of CV boundaries after amplification. It infer that in older adults, though the hearing aid was prescribed to overcome the problem in audibility, temporal distortion of input signal from hearing aid was present due to different compression parameters in two channels. Another source of distortion could be temporal asynchronous firing introduced by peripheral pathology in the older auditory

system. The altered envelope by amplification system and temporal asynchrony in peripheral pathology has complex interplay on perception. These effects modify the signals and are relayed as concomitant changes in the central auditory system, which in turn might have perceptual consequences.

Further, the responses at each level of the auditory system (brainstem and cortical) were compared between good and poor hearing aid performers. The responses at each level were relatively well represented in good hearing aid performers than in poor hearing aid performers. It can be inferred from these findings that there is relatively stronger afferent and efferent auditory pathways in good hearing aid performers i.e., in GHP, the central afferent mechanism are stronger such that neurons fire more robustly than in PHP. Additionally, the efferent mechanism is stronger, thus fine tuning the auditory input through inhibiting the fibres, which do not correspond to either F_0 or formant frequencies of the stimulus.

The present study provides an approach to measure the representation of amplified speech at different levels of the auditory pathway. Collecting data from this approach throws light on representation of speech at different levels of the auditory pathway. Further, it gives a direction for the audiologists and technologists to improve on the parameters in a hearing aid that might improve the performance. The obtained information will be utilized in counselling the clients regarding the encoding of speech at different levels and the limitation / ability of the available technology to solve the issue.

IMPLICATIONS OF THE STUDY

The implications of the present study include:

1. The study presents an evidence to use objective approaches to validate the hearing aid output at different levels of the auditory pathway.
2. To know the extent of modification induced by hearing aid, the acoustic content of the incoming signal serves as the template for output of the hearing aid. Utilization of the real ear measurement for analyzing the hearing aid output in the ear canal will help in knowing the representation of spectral and temporal properties of speech in the ear canal of the participant.
3. Studying the encoding of amplified speech in the impaired auditory pathway assists in guiding the hearing aid manufacturers to come up with the advanced technology. It also solves some of the practical problems faced by the audiologists regarding amplification parameters in providing the maximum usable information.
4. Findings of the present study help the audiologist in counselling a hearing aid user regarding the benefit derived even with the best hearing aid prescribed.

FUTURE DIRECTIONS

From the findings in the present study it is imperative to conduct further research. These include -

1. Acceptable noise level (ANL) is one of the behavioural measures that can be used to predict the hearing aid usage. However, ANL considers the acceptance of noise as one of the prime factor for deciding hearing aid usage. Though the ANL is a good behavioural tool for the acceptance of hearing aid, there are other factors which include psychological attitude towards willingness to wear hearing aid and cognitive status of participants, which might also influence its acceptance. Thus, these factors also need to be given consideration along with ANL to predict performance with the hearing aid. It helps the clinician to know the confronting variables that might contribute in rejection of the hearing aid and consequently taking appropriate measures or steps to utilize the available technology as well as counselling to solve the problem faced by individuals with the hearing impairment.
2. The current study gives information only on detection response at various levels of auditory pathway. This is because the conventional method was adopted in recording the FFR, LLR and ACC, as the stimulus was presented repeatedly. With the use of the stimulus in odd-ball paradigm, in addition to the detection process, the clinician can study the discrimination (mismatch negativity) and identification (P 300) processes of audition in individuals with hearing impairment.

3. The processing of hearing aid by the impaired auditory system was measured using electrophysiological approach in quiet conditions. However, in noisy situation, the individuals with hearing impairment experience difficulty in understanding speech. Thus, there is a need to know how the individuals with sensorineural hearing impairment challenges the noise as it obscure the temporal and spectral content of message though the hearing aid alleviates the problem of audibility but introduce minimal temporal alteration.
4. Immediately after a listener is fitted with hearing aid, objective tests help to study the representation of amplified speech along the auditory pathway. However, the effect of experience in hearing aid usage (part time & full time hearing aid users) on the representation of amplified speech at different levels of auditory pathway needs to be investigated on a long-term basis over a period of time.

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